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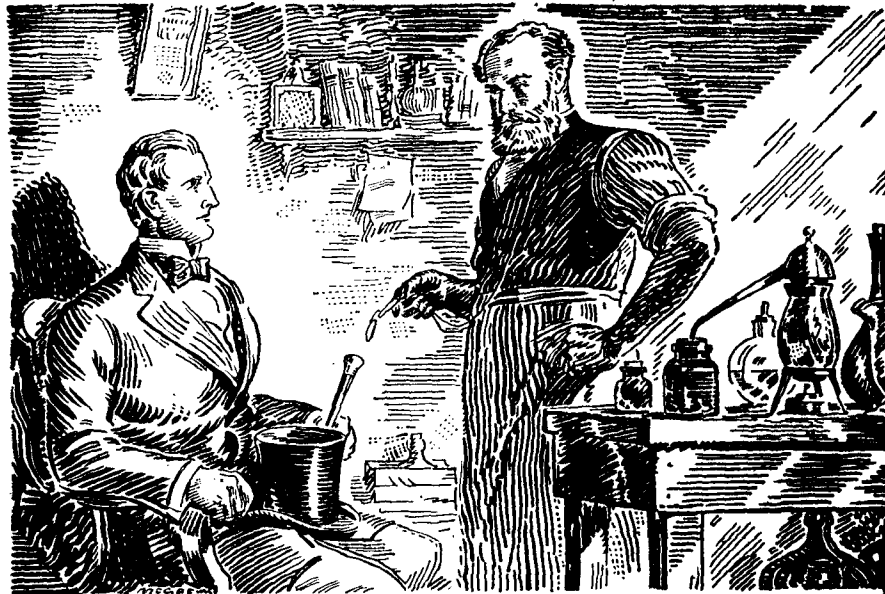
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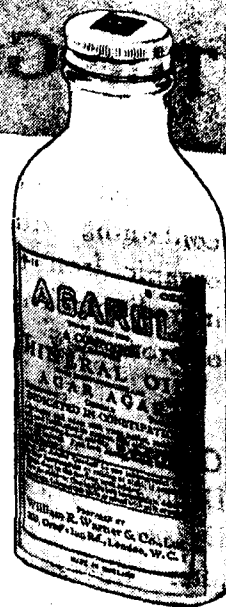
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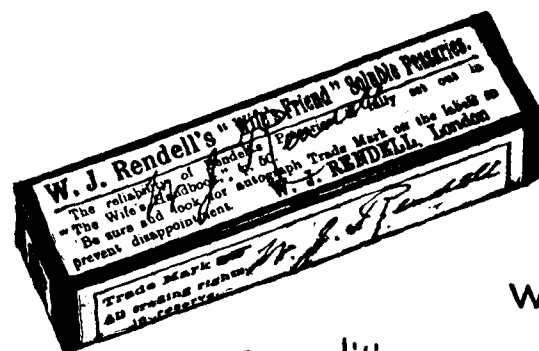
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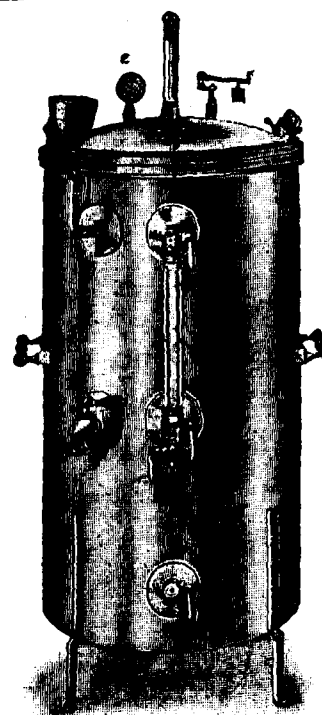
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H.K. SEN, ESQR., M.A., D.Ic., D.Sc., (Lond.)
Sir Rashbihari Ghose Professor of
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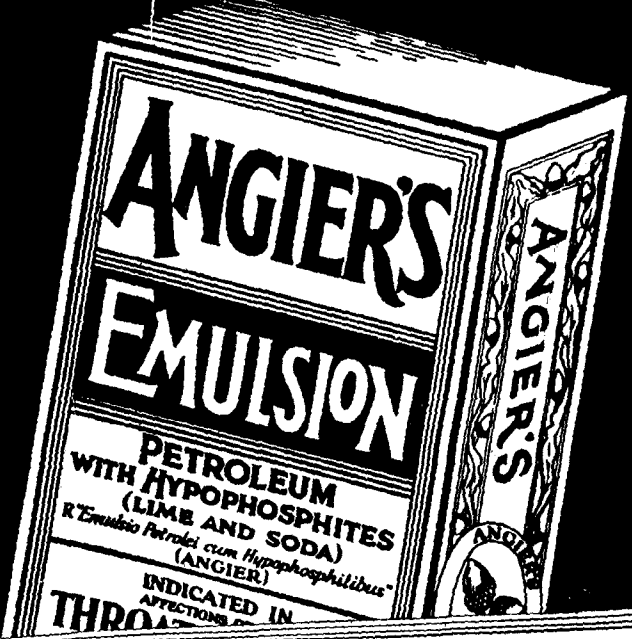


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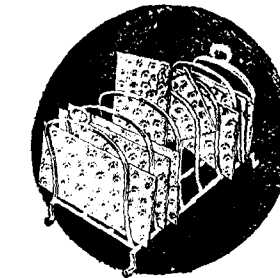
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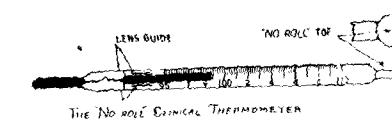


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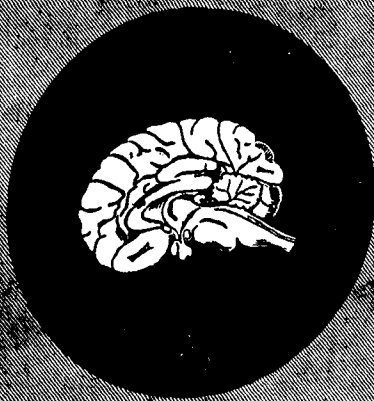
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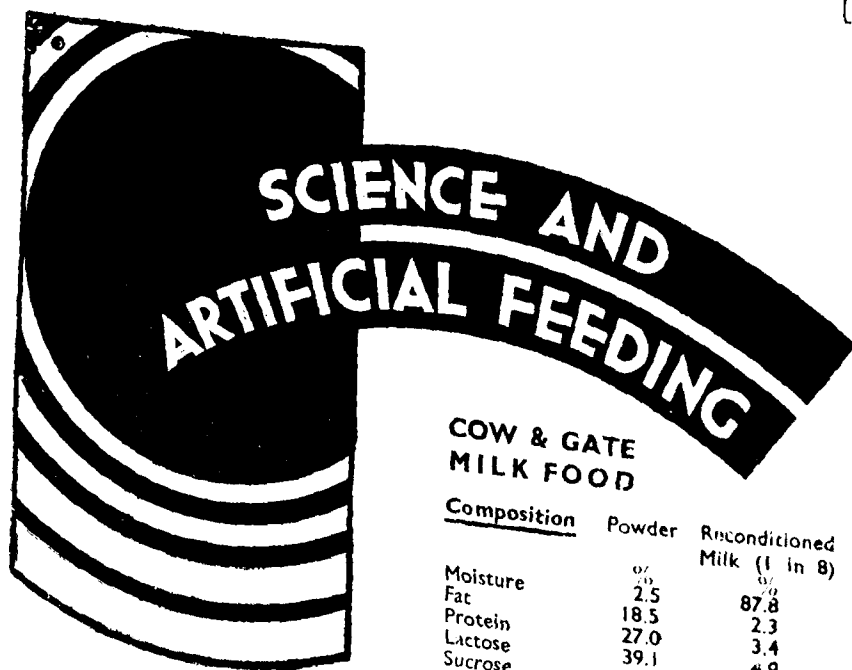
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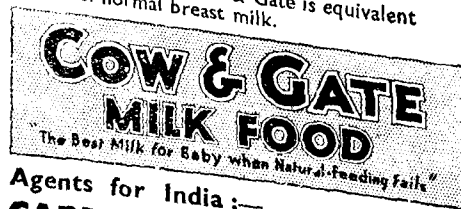
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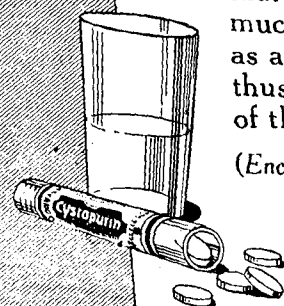
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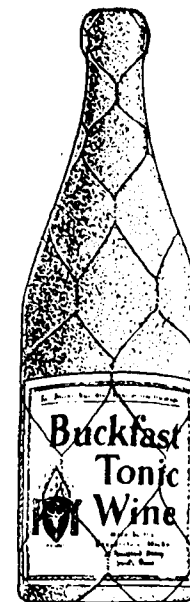
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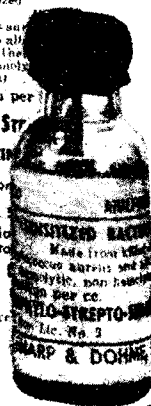
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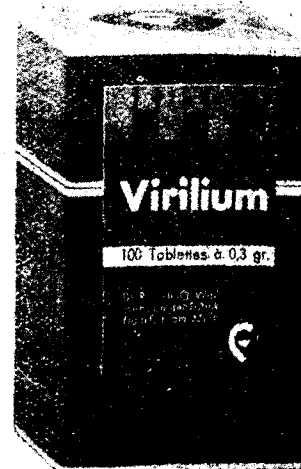
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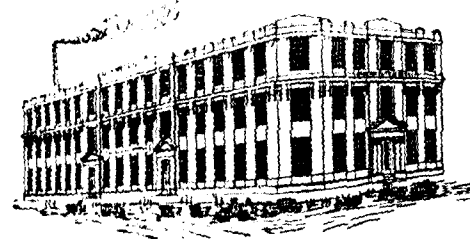
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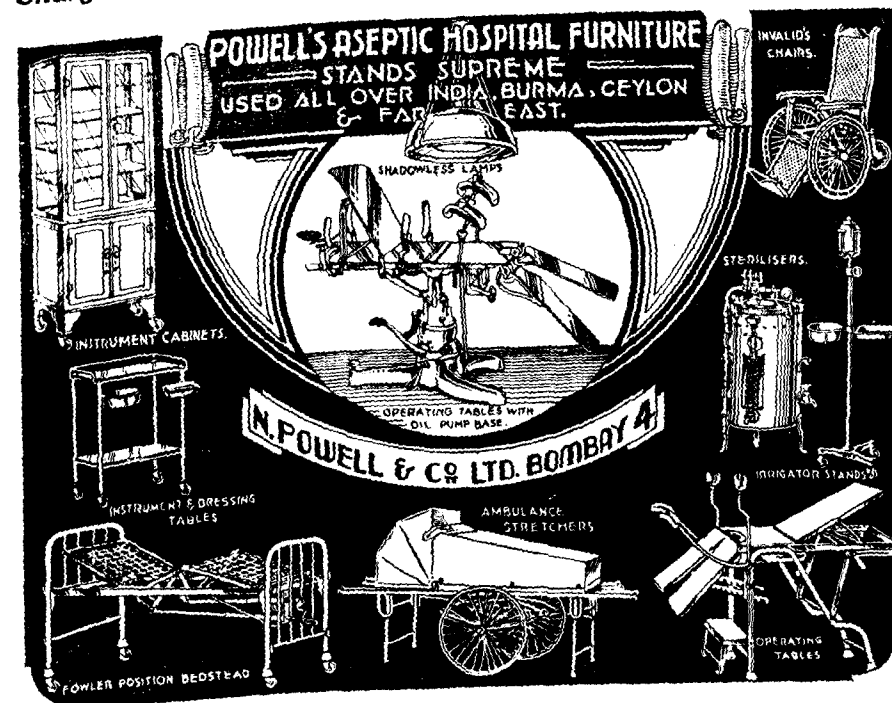
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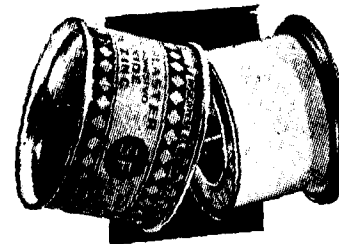
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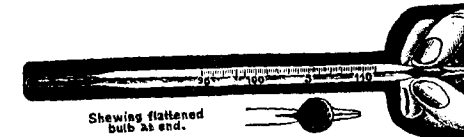
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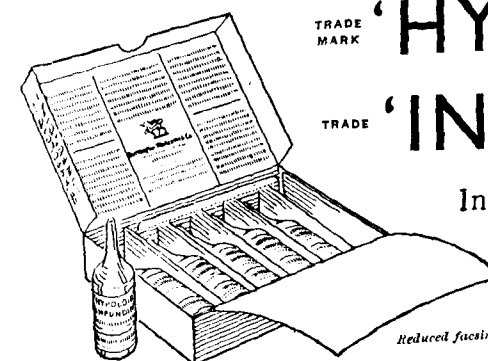
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SECTION VI. LEPROSY

Leprosy—its Diagnosis and Treatment

BY

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Definition.—Leprosy is a chronic, infectious and mildly contagious disease peculiar to man and caused by *Mycobacterium leprae* of Hansen with a prolonged period of incubation and characterised by lesions of the skin, mucous membrane and nerves.

History.—Leprosy is as old as the world. In India, the word *kushta*, meaning leprosy is found used in the Rig Veda of date 1,500 B. C. Manu, (880 B. C.), the ancient Hindu Law-giver, laid down certain rules for the guidance of lepers, evidently with a view to prevent its further spread. In the ancient Hindu Medicine, (Susrutha, 500 B. C.), leprosy is treated in great detail, dividing it into 18 sub-types.

Geographical distribution.—In India, leprosy is endemic and so, it is met with all over. There are no reliable data about its incidence. It is estimated that there are about two to three million lepers in India. According to 1931 census, there are 33,381 lepers in the Madras Presidency, of whom 24,951 are males and 8,330 females. The most affected district is South Arcot with

4,129 lepers or 150 per 100,000 and the least affected is the Nilgiris with 11 lepers or less than 10 per 100,000. The Public Health Department also conducted a rough leprosy survey and detected 48,000 lepers in this Presidency. In Vizagapatam, there are only 71 lepers according to the census but 546 cases have so far come voluntarily for treatment to the clinic. So, the census figures have to be multiplied ten times or more to be nearer the correct figure.

Aetiology.—The causative organism of leprosy has been definitely acknowledged to be *M. leprae* discovered by G. Armour Hansen in 1873. No doubt it has not satisfied two of the four Koch's postulates namely, cultivation of the bacilli *in vitro* and reproduction of the disease from such culture in experimental animals. Besides this specific causative organism, there are many factors or predisposing causes which help the development of leprosy. Some of these are:

(1) *Civilization.*—Though leprosy is found among both the civilized and uncivilized, it is practically unknown amongst the aborigines and wild tribes. The incidence is proportionate to the degree of civilization and it has been found that the greater incidence is in the "contact zones". In other words, leprosy is more prevalent amongst those who are just changing from the primitive to the early stage of civilization.

(2) *Over-crowded and unhygienic dwellings* with insanitary surroundings and lack of Sun.

(3) *Peculiar social conditions* such as sleeping in the same bed, eating from the same plate, smoking the common pipe and cheroot etc.

(4) *Certain injurious habits* such as lack of personal hygiene, irregular and sedantary habits, sexual excesses, laziness, over-work, walking bare-footed etc.

(5) *Defective diet.*—Decomposed foods such as stale rice, salted fish or decomposed meat, over-cooked or under-cooked food, food rich in animal proteins, fats and mineral salts, predispose to leprosy.

(6) *Heredity.*—Though leprosy is not inherited, predisposition to leprosy may be transmitted from the parent to the offspring.

(7) *Individual susceptibility.*

(8) *Certain physiological states* as puberty, pregnancy and lactation.

(9) *Starvation.*

(10) *Recent attacks or co-existence of other diseases* such as, acute infectious diseases e.g., small-pox, influenza, enteric fever, cholera, malaria, kala-azar, dysentery, helminthiasis etc.

(11) *Focal sepsis* particularly oral sepsis.

(12) *Climate.* Hot moist and wet climates have a debilitating effect.

(13) *Fear of leprosy*, and its absence with the consequent neglect.

(14) *Intimate continuous and prolonged contact* with an infectious leper, particularly in childhood.

Mycobacterium leprae.—They are rod shaped organisms resembling tubercle bacilli morphologically and tinctorially. They are acid-fast and gram-positive. They stain readily with Ziehl-Neelson's method. In stained specimens they appear in groups, bunches or bundles known as "cigar bundles". They are seen packed within large cells known as "lepra cells" and also outside them. The organisms appear in a variety of forms such as regular uniformly stained rods, rods with a beaded appearance, rods showing polar staining, broken rods, budding and branching rods etc. In treated cases, they stain differently, taking different shades of colour as blue, purple etc., instead of red. This characteristic pleomorphism may be the result of degeneration or represent different stages in the evolution of the organism or different phases of the disease. *M. leprae* can be differentiated from tubercle bacilli by (1) their arrangement as "cigar bundles", (2) their large number in every field, (3) their being straighter and thinner, (4) their presence in certain regions. Tubercle bacilli are seldom seen in nasal or skin clip smears. (5) Their not producing leprosy lesions in laboratory animals when inoculated with them.

Though many attempts have been made to culture the organism by many workers with claims of success by some, the successful cultivation of *M. leprae* is not yet considered accomplished.

Pathogenicity.—With the exception of man, *M. leprae* are not pathogenic to any other animal high or low. Rao and co-workers were able to transmit the disease to *Macacus* monkeys by "an intensive method of several intraperitoneal injections". In rodents, an analogous disease, rat leprosy exists and the acid-fast bacilli, *B. Stefansky*, which causes it, does not seem to be transmissible either to other animals or to man. Recently, Lobel (1934) discovered a bovine type of leprosy in water buffaloes in the Netherlands, East Indies and named it "*Leprae bubalorum*" which, as the editor of the International Journal of Leprosy (January-March 1936) thinks "both in its gross manifestation and in the laboratory findings, bacteriological and histological, seems fully to justify the name given it".

Methods of demonstration of *M. leprae*.—The common methods adopted for diagnostic purposes are :—

- (1) Nasal smear.
- (2) Smear from ear lobe, a cutaneous patch or a nodule, and
- (3) Gland puncture.

The organisms of leprosy can also be demonstrated in smears taken from the affected nerves, but this method is rarely attempted.

1. *Nasal smear*.—With a blunt scalpel, the mucous membrane covering the anterior third of the septum, floor and the inferior turbinate bone is scraped till blood staining is seen on the scalpel, and smears are made. The organisms are in the mucous membrane and so it must be lightly scraped till slight oozing of blood takes place. The writer uses a lupus scraper.

2. *Smear from the ear lobe of skin patch*.—This can be done in three different ways.

(a) *Skin clip*. The ear or patch from which the clip is to be taken is painted with Tincture of Iodine and held tight between the thumb and forefinger of left hand and a fine clip, a few millimeters long, is made with a sharp curved scissors. The clip should be only just skin deep. If properly done, the patient suffers no pain, there is hardly any bleeding, the bacilli, if present, are easily demonstrable and there is no scar left. The clip is transferred with a pair of forceps to a slide with the cut surface on to the slide and smeared a number of times at the same time crushing it as much as possible. The ear is particularly selected as it is easily accessible, hidden from the patient's sight and is a common habitat of *M. leprae* even if it looks apparently normal.

(b) *Slit method*. This is preferable in areas such as the back where it is not easy to take a clip. With a fine sharp knife a tiny incision is made skin-deep at the growing edge of a patch, ear lobe or border of the ear. The incision should be so superficial as not to produce any bleeding but only serum should ooze. The base of the wound may be scraped and smear made or the drop of serum that collects in the wound may be taken to make the smear.

(c) *Raising a blister*: The fluid in the blister is used to make the smear.

3. *Gland puncture*.—A hypodermic syringe with 0.1 to 0.2 c.c. of sterile normal saline is taken. The skin over the enlarged gland is sterilised and with the needle of the syringe, after steadying the gland, the skin is first punctured and then the gland. The saline in the syringe is then injected into the gland which is lightly massaged and aspirated. Enough material for making a few smears can thus be easily obtained.

The instruments and slides used for making the smears should be clean and flamed to exclude the possibility of having any other acid-fast bacilli in them.

Modes of transmission.—The experimental transmission of leprosy to laboratory animals and even to human beings have been failures with the doubtful exception of Arning's case in a Hawaiian convict. The house fly, the itch mite, the louse, the bed bug, the tick, the mosquito, the flea etc., have all been incriminated. Although insect transmission is possible, Lebouf, after a study of the literature and after extensive observations, concluded that the house-fly may convey live bacilli in their intestines and deposit them on the injured skin or mucous membrane of the nose of a healthy person. But the evidence implicating the blood-sucking insect is not satisfactory. Transmission by intimate personal contact or by using infected clothing, bedding etc., is considered to be the most likely method.

How the bacilli enter the body.—Many think that the bacilli gain access to the body by inoculation, ingestion and sexual intercourse. But the consensus of opinion is in favour of entrance through a minute breach of surface of the skin, nasal, buccal or pharyngeal mucous membrane by intimate, prolonged and continuous contact with an infectious leper or infected articles particularly during childhood. After the bacillus has gained admission into the body, as in the case of any other organism, one of three things may happen: (1) The body tissues may fight the intruders and succeed in destroying them, (2) The organisms may lie latent and wait for an opportunity till the resistance of the individual is lowered or (3) They may multiply and overcome the fight put up by the body tissues, invade and colonise other parts of the body. The bacilli at the site of entry multiply and irritate the tissues to activity and produce the primary lesion. From here the bacilli may spread to other parts of the body by three routes, (1) through the lymph spaces and lymphatics, (2) through the blood stream and (3) by auto-inoculation.

The organisms of leprosy, after establishing themselves on the skin, always spread radially producing circular, oval or

elongated patches. This radial spread can at times be seen by the formation of small specks radial to a central, small patch (See Fig. 1.). Larger and irregular patches are formed by the coalition of smaller ones. Some of them are so large as to cover a whole cheek, large part of chest, back, buttocks and limbs. The usual leprotic lesions are, (1) erythematous, raised, infiltrated, non-pigmented anaesthetic or non-anaesthetic patches and (2) thickened infiltrated nerves. Whatever the nature of the lesions and where-ever they are present, the pathology is the same. This consists of multiplication of connective tissue cells, proliferation of fibrous tissue cells depending upon the chronicity of the lesion, migration of large round cells and cells resembling lymphocytes, dilation of the capillaries whose walls in course of time show hypertrophy and comparative oedema of the affected area.

Incubation period.—It is very difficult to fix an incubation period for leprosy as there are no data. Even animal and human inoculations with the bacillus of leprosy have been failures. By a consideration of the data, available in leper asylums and settlements of children of leprosy parents, it is reckoned that the incubation period is from two to four years. The writer has come across two cases in children, one of eight months and the other of seven months of age with a number of hypo-pigmented patches. The mother of the eight-months-old child did not show any signs of leprosy and the father also was said to be free. In the seven-months-old child, the patches have been noticed for three months *i.e.*, when the child was only four months old, and the mother showed lesions of a mixed type of leprosy. Even before conception she had been suffering from leprosy and the disease advanced rapidly during pregnancy and puerperium. From these two cases the incubation period should be from 4 to 8 months as leprosy is not considered to be inherited.

Modes of onset.—There are two modes of onset;

- (1) Acute, and
- (2) Sub-acute or chronic.

1. *Acute onset*:—When a large number of patches appear on the surface of the skin distributed symmetrically, bilaterally, or irregularly the onset is considered to be acute. In these cases wherever the initial lesion be or whatever the portal of entry be, lesions have to be considered to be metastatic or blood-borne. In an acute onset there may be prodromal symptoms as irregular fever, general malaise, nausea, loss of appetite, pain in the course

of the superficial nerves, numbness and tingling of pins and needles in the extremities. After a time the patches become manifest. They may be raised, red, non-anaesthetic patches and showing *M. leprae* in clip smears or a mixture of the two types. In anaesthetic type the superficial nerves and sometimes the fine cutaneous nerves supplying the patches may become painful, tender and swollen.

Another kind of acute onset though not so common is to begin with high fever, headache, pain all over the body, running from the nose, loss of appetite and great prostration. In short, it is a leprosy reaction or leprosy fever. With the fever a large number of erythematous infiltrated patches and small temporary nodules on the skin or subcutaneous tissues develop. The face is swollen and red, the conjunctiva is injected and the ears are thickened, enlarged and elongated. Within a few days the acute symptoms subside including all or most of the patches, infiltrations and nodules. In some cases one or two patches are left behind. Such acute attacks are repeated a few times at varying intervals, the duration of which varies before the disease manifests itself.

2. *The subacute or chronic onset.*—Here the patches appear slowly in different parts of the body taking usually months or years for the development of a number of patches. The patches are of various kinds and shapes. Adjacent patches coalesce and form larger ones sometimes covering large areas of the body. The kinds of lesions met with are:—

(1) Hypo-pigmented patches with or without disturbance of sensation and absence of leprosy bacillus. (Fig. 1.)

(2) Red raised or infiltrated patches with no disturbance of sensation but showing *M. leprae*. (Fig. 2.)

(3) Patches showing three different zones, (a) a hypo-pigmented anaesthetic centre, (b) a raised red non-anaesthetic middle zone and (c) a hypo-pigmented anaesthetic border. (Fig. 3.)

(4) Hypo-pigmented rings with or without disturbance of sensation with unaltered skin in the centre.

(5) Faintly hypo-pigmented area presenting goose skin, nutmeg grater or keratosis pilaris appearance.

(6) Blisters on the fingers or toes with or without sensory disturbances of the affected area.

(7) Muscular paralysis and deformities, *e.g.*, wrist drop, ankle drop, wasting of the intrinsic muscles of the hand etc. The nerve supplying the affected muscles is found thickened.

(8) Acroteric lesions with no patches. These are seen in the extremities with a tendency to centripetal spread and

characterised by "alterations of sensation, trophic disturbances in the skin, bones and muscles and paralysis."

Classification.—There are two main types of leprosy;

- (1) Neural and;
- (2) Cutaneous.

Where both types coexist, it is known as mixed type.

- I. *Neural* (N). All cases that show evidence of actual or previous nerve involvement. There are no leprotic changes in the skin.

Sub-types: Neural-1 (N1). Slight neural. Few small areas of disturbed sensation and pigmentation.

Neural-2 (N2). Moderately advanced neural. Extensive or numerous areas of disturbed sensation with paralysis and/or trophic disturbances.

Neural-3 (N3). Advanced neural. Extensive areas of anaesthetic and marked motor and trophic disturbances.

- II. *Cutaneous* (C). Cases showing leprotic lesions in the skin.

Sub-types: Cutaneous—1 (C1). Slight cutaneous. One or few leprotic macules, infiltration or nodules.

Cutaneous 2 (C2). Moderately advanced cutaneous. Numerous actively leprotic macules, infiltration or nodules with lesions of the mucosa.

Cutaneous 3 (C3). Advanced cutaneous. Numerous or marked leprotic lesions of the skin and mucosa.

Secondary Neural. Neural cases that were formerly cutaneous but from which the active leprotic lesions have disappeared.

Burnt-out cases are those arrested cases that show mutilations, contractures etc. This term is now replaced by "arrested with deformity."

Open cases are those in which organisms can be found in the skin or mucous membrane and also at times on the surface. Such conditions favour dissemination.

Closed cases are those from which no bacilli can be obtained from the accessible lesions by the ordinary methods of examination. These were formerly known as "bacteriologically negative."

Symptomatology.—*Age*—Children are very susceptible to leprosy infection particularly children from three years and upwards and young adults up to 20 years of age. But no age is

exempt though rare in old age. The period of pubescence is another susceptible period. In a series of 1837 cases that came under my observation, the youngest were two babies of 7 and 8 months old, the former having had the disease from the fourth month. The oldest in the series was 85 years old. In the above series, 546 cases were from the Vizagapatam Municipality. An analysis of the age period of the onset of these cases showed that 41 cases (7.5%) were 5 years and under, 115 (21 %) were 10 years and under, 347 (63.5%) were 20 years and under and 457 (83.7%) were 30 years and under, thus showing that leprosy is practically a disease of children and young adults. (See diagram in next page.)

Sex.—Males are more affected than females. Of the 546 cases in Vizagapatam, 380 were males and 166 females. *i.e.*, in the proportion of 2.3:1. The sex incidence in the census report of 1931 is 3:1. The proportion generally given by other observers is 2:1. The lesser incidence in women may be due to concealment, less of free movement and so less chance of coming in contact with persons suffering from infectious leprosy, more clothing and so less exposure of parts to infection and the development of the female sex hormones which may have a deleterious effect on the development of leprosy.

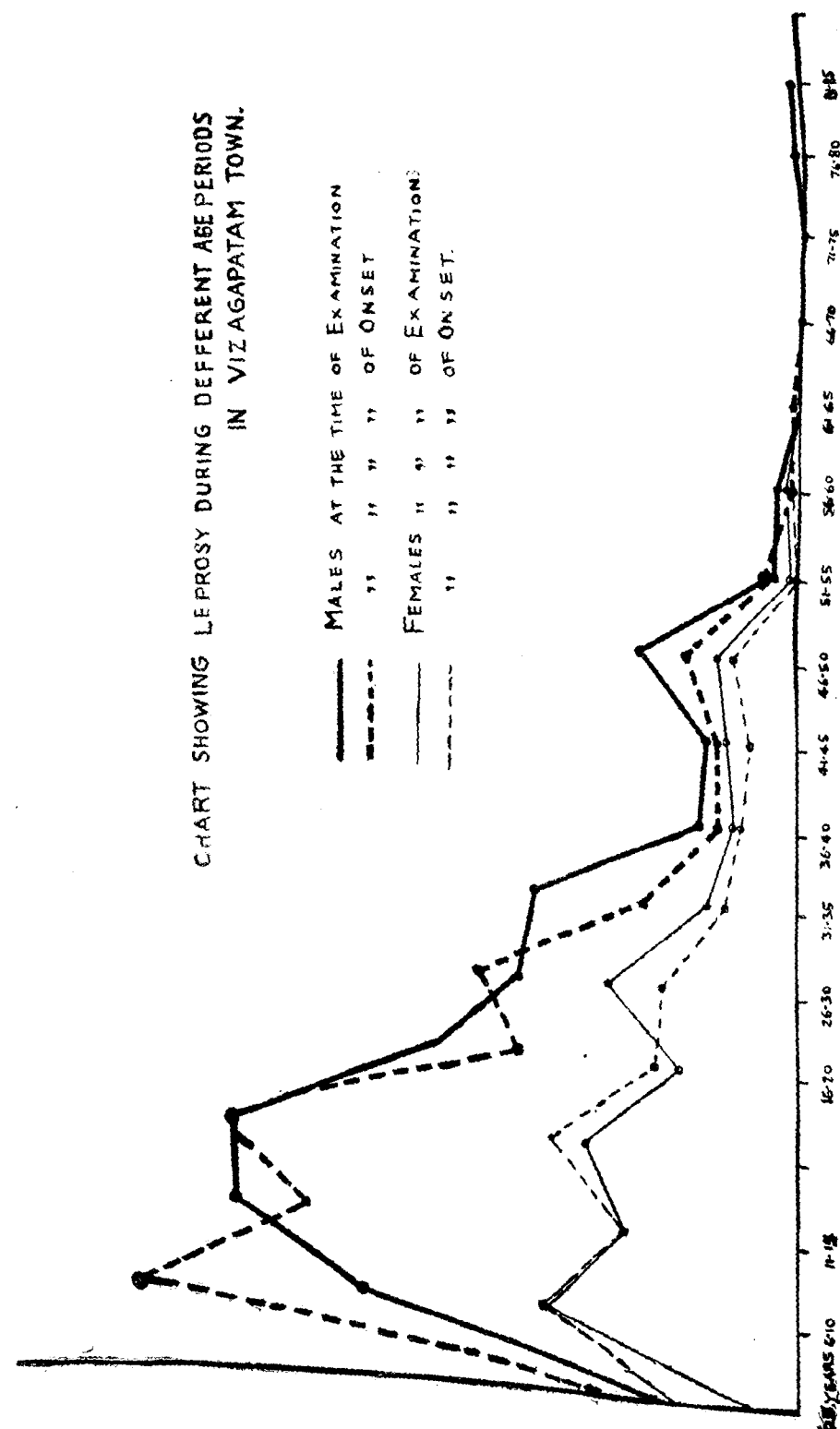
Race.—All races are affected, though at present it is more prevalent among the Orientals, Africans and Polynesians. Among the 646 Vizagapatam cases, 503 are Hindus, 22 Mohammedans, 21 Christians in a population of 51,299 Hindus, 2,955 Mohammadans and 3,011 Christians.

Social status.—Though leprosy is no respecter of wealth or social position, still it is more a disease of the poor and the lower strata of society, as they live in unhygienic houses with insanitary surroundings and on ill-balanced, non-nutritious food with more opportunities for coming in contact with infectious lepers.

Occupation.—Leprosy is found in all occupations. But persons with leprosy who come in contact with healthy individuals may disseminate the disease.

Clinical aspects.—Clinically leprosy can be primarily divided into two types. (1) The cutaneous and (2) The neural. There is also a third type, the mixed type, where there is a mixture of both cutaneous and neural lesions.

Cutaneous type.—As already referred to, the onset of leprosy may be of two kinds—the acute and the subacute or chronic. In either case a fully developed patch shows infiltration or thickening of the skin with or without disturbance of pigment. The patches



may be regular or irregular in outline, more often the former. They are round, oval or elongated, slightly raised above the surface and red or of a coppery colour. They may be single or multiple, unilateral or bilateral and symmetrical. The patches may show only infiltration and at times are very extensive and appear like large plaques. In these infiltrated areas and also unconnected with them, nodules may develop. The nodules may be temporary and resolve fairly quickly or be permanent. The skin over these nodules and proliferations is hyper-aesthetic first, then hypo-aesthetic and finally becomes anaesthetic. When the infiltrated areas and nodules resolve and completely disappear, the skin becomes wrinkled and atrophied. Some of the nodules may soften and ulcerate discharging *M. leprae*, and so they are infectious unlike the trophic ulcers. The lesions may appear in any part of the body but they are more common on the face particularly cheek and other exposed and laid on parts and external and extensor aspects. The ears become thickened, infiltrated, nodular, elongated and enlarged. In advanced cases, due to the leprotic infiltration of the skin, the face becomes glossy, thick, furrowed, dull, gloomy and expressionless which is known as "Leonine facies". (See Fig. 4.) The mucous membrane of the nose and mouth become infiltrated and ulcerate disseminating the bacilli. The pharynx and larynx also become involved producing huskiness of voice, aphonia and dysphagia. The hairy scalp is rarely affected in this country but frequently seen in Africa and Japan, with alopecia. The eyes are also affected, iritis being the most common complication. The mammary glands in the male are enlarged in a fair proportion of cases (Fig. 5.) and in equal number, the nipples are infiltrated and they stand out prominently being thickened and elongated (Fig. 2.). Leprotic infiltration of the testes and ovaries with consequent sterility is not uncommon, though more common in the male than in the female. The internal viscera also do not escape the infection. Liver, spleen and kidneys show leprosy deposits. Lungs are not so uncommonly affected as generally believed to be, though tuberculous infection of the lung in a leper is more common. Although lepromatous infiltration of the heart is not seen, it is affected by the toxin. Now and then patients with leprosy reactions die suddenly with substernal pain, blueness in the face and dyspnoea. The toxæmia and bacillaemia caused by the leprosy fever put the heart to great strain having poisoned it. So it gives way and the patient dies suddenly. Generalised lymphadenitis is frequent and as already mentioned the femoral glands and a small gland at the

emergence of the great auricular nerve are commonly enlarged. The glands are slightly hard, freely movable but at times with slight peri-adenitis. Bones, joints and even nails do not escape. Leprous osteitis, osteomyelitis and arthritis especially of the bones, of the hands and feet are rarely met with. Changes are noticed on the fingers also, producing alterations in the finger prints. Recently a case was reported where this alteration of the finger print led to the case being diagnosed as leprosy. In this type attacks of fever occur now and then. These attacks are known as lepra reactions or leprous fever. The fever may precede or accompany an out-break of new lesions and with the subsidence of the fever, the lesions also resolve.

Neural or nerve leprosy.—There are two kinds of infection in this type, the ascending and the metastatic. In the ascending type the bacilli after entering the skin select the sensory nerve endings for their points of attack and slowly ascend the terminal twigs to the nerve trunks. In the metastatic type the bacilli form an embolus which blocks the vasa nervorum thus affecting the nerve trunk. In either case the same proliferative reaction takes place in the sheaths and coverings of the nerves. This results in pressure on the nerve fibres producing their degeneration and atrophy. There are also two kinds of onset, the acute and the chronic though the latter is more common. The acute onset is not usually accompanied by constitutional disturbances as in the cutaneous type. Before the development of the macules there are prodromal or antecedent symptoms as numbness and tingling, itching and burning of the extremities, and shooting pain and tenderness along the course of the superficial nerves. Then macules begin to appear. They are generally hypo-pigmented and there is no de-pigmentation as in leucoderma. In dark individuals the hypopigmented areas look reddish instead of whitish and when the areas are long-standing they appear crinkled as cigarette paper. On account of this reddish colour and the crinkled nature, the macules may at times be mistaken for leprous patches unless one is careful. The only way of avoiding the mistake is to palpate the areas when the macules will be found to be neither raised nor infiltrated as cutaneous patches. Besides the altered pigmentation, the macules show also altered sensation. They may be hyper-aesthetic, hypo-aesthetic or anaesthetic to light-touch and in marked cases there may be anaesthesia to pain, heat, and cold. If the macules are in the hair bearing areas, the hairs become thin and brittle and later break or completely fall off. These areas are hyper-hidrotic for a time or show bouts of

sweating before becoming hidrotic. The skin also appears thin, atrophic and glossy or harsh, scaly and para-keratotic. The superficial cutaneous nerves supplying the macules become thickened, hard and sometimes painful. In general, the superficial nerves also become thickened, hard, painful or painless, easily palpable and freely movable. The commonly affected nerves are the great auricular, ulnar and peroneal and less frequently radial, median, supra-orbital, facial and internal tibial nerves. Of the cranial nerves, the fifth and the seventh (Fig. 6) are commonly affected. The nerve trunks may be uniformly thickened or be beaded, fusiform or spindle shaped. The nerves are at first swollen, painful, thickened and tender. They then get hardened and in extreme cases feel like cords or iron rods, and become insensitive. In advanced and long standing cases the nerves again become thinner, but insensitive. Abscesses develop in the nerve though none were seen here. The nerve abscess seems to be peculiar to certain localities and when it is found it is seen in cases with affection of one nerve only. When the nerves in the limbs are affected there is weakness, atrophy and paralysis of the muscles. In the hands the power of grip is diminished, intrinsic muscles atrophy and waste. The usual eminences disappear, the interspaces between the metacarpal bones are very much deepened and the fingers are contracted (Fig. 7). The hand is very much wasted, the wrist and the proximal phalanges of the fingers are extended with the middle and distal phalanges acutely flexed producing the "ape hand" or "leper claw". All imaginable types of deformity develop beginning with slight contraction of the little finger to the classical "main en griffe." In the feet the toes may become flexed—the patient walking on tip toes. When motor nerves are affected paralysis of the muscles supplied by them results. Wrist and ankle drop and facial paralysis (Fig. 6.) are the usual ones.

Trophic disturbances are common and the changes noticed are in all the structures, from the skin down to the bones producing bullae or blisters, pigmentation, glossiness or para-keratosis of the skin, (Fig. 9.) ulceration of the soft tissues, perforating ulcer, gangrene, rarefaction, necrosis or absorption of the bones. (Fig. 10.) Trophic ulcers on the sole of the foot (Fig. 8.) are very common, the usual sites being the area just below the interspace between the second and the third toes, the ball of the great toe and the heel. Mutilation of the fingers and toes (Fig. 8.) are not uncommon. Whole fingers and toes may get absorbed with the exception of the nail and in extreme cases everything up to the

wrist is absorbed leaving the nails attached to the wrist. Effusion into a joint with or without the destruction of the cartilage and bony ends; and a condition resembling charcot's joints may develop. In this type also eye changes are seen, but due to the affection of the fifth and seventh nerves producing ulceration of the cornea, ptosis, paralytic ectropion etc. In extreme cases the skin as a whole becomes glossy and the extremities particularly the hands and feet become oedematous. (Fig. 9.) This oedema is solid and is known as leprous oedema. The skin is ichthyotic, and palms and soles become hyperkeratotic with cracks and fissures. The latter is more common in the feet and some of them are deep, painless and difficult to heal and at times are the beginnings of perforating ulcers. Alopecia of the eye brows commencing from the outer end is common. But loss of hair is also met with on the face, eye lids and other parts of the body, either as a result of the development of macules or generalised trophic disturbances. Alopecia and lesions of the scalp do exist but are not seen in these parts.

Though these two types are seen separately, they are also seen fairly commonly together. There is some inter-relationship between the cutaneous and the neural types when they occur together. The more the skin is affected, the less the nerves and *vice versa*. The lesions are met with in all parts of the body but here they are not found on the scalp and are seen only rarely on the palms, soles and genitalia. The face, buttocks, around the elbows and knees and outer aspects of limbs are the common sites.

Leprous reaction of leprous fever.—This is a common occurrence whether the patient is undergoing treatment or not. The reaction and the fever also subside with or without treatment. This peculiarity should be remembered as some of the vaunted cures of leprosy are those drugs that were administered during a reactionary phase of leprosy. Another feature is that it occurs more commonly in cutaneous types. When it occurs in neural types, sometimes cutaneous lesions develop for the first time, thus showing that a neural case is not always non-infectious and that at any time it may become infectious. Very recently an antenatal case was attending the venereal department for anti-syphilitic treatment as the patient's blood Wassermann was strong positive. She had a very suspicious leprotic facies characterised by glossiness of the face and also of the extremities. There were no other incriminating signs or symptoms, no macules or cutaneous patches, no thickening of the superficial nerves. She was given two injections

of neo-salvarsan, 0.3 g. each, when she developed a reaction with development of red, raised infiltrations particularly marked on the face, ears and arms. Clip smears taken from the ear and the infiltrations were positive for *M. leprae*. The reaction usually sets in with prodromal symptoms as malaise, lassitude, numbness, tingling, burning and itching in the extremities, peculiar unpleasant sensation or pain along the course of nerves, loss of appetite and disinclination to work. Then the patient gets an attack of fever preceded by chills, pain in the joints or generalised all over the body. The temperature rises up to 103° to 104° F., the face is red, eyes are congested and watery and the patient looks ill and toxic. He prefers not to be disturbed and likes to lie down with the limbs flexed and eyes closed and covered with a sheet of blanket. He shuns light particularly the bright sun light. The appetite is lost and nourishment is taken indifferently. The existing leprous lesions become swollen, congested and more infiltrated. The thickened nerves become painful, tender and more swollen, unaffected nerves become affected and new erythematous raised patches and infiltrations appear. In some cases, the new patches are profuse and widely distributed but in others few and discrete, depending on the severity of the reaction. The ears become swollen, red and enlarged, the superficial glands inflamed, painful and in some of the neural cases, trophic blisters also develop. The temperature keeps up for a few days and then gradually comes down. The blood smear particularly a thick smear, often shows *M. leprae* and the sedimentation index is high. With the subsidence of fever, the patient regains his appetite and slowly returns to his normal state. The old lesions regress to their previous state and the new lesions disappear with desquamation of the skin covering them. The nerves also resume their previous size and state. In some cases the original lesions do not regain their original condition and some of the new lesions may not disappear. In some other cases these reactions are beneficial in as much as the original lesions completely disappear and the patient appears apparently well.

How these reactions are caused is not definitely known but, it is considered.

(1) That it is an allergy or protein sensitization phenomenon.

(2) That it is due to the simultaneous breaking down or lysis of a large number of lepromata or leprous granulation with subsequent release of a large number of bacilli and their toxin producing a bacillaemia and toxæmia.

Changes in blood.—No definite changes of a diagnostic or prognostic value are noted either in the cellular elements of the blood or in its serum constituents.

Diagnosis.—There is no difficulty in diagnosing well advanced cases of leprosy. The difficulty is with very early cases, particularly of the neural type, where hypopigmentation is slight, anaesthesia not yet developed, the superficial nerves absolutely normal, no *M. leprae* to be demonstrated in the nose, ear or suspected macule. The ignorant and illiterate villager suspects leprosy in its very earliest stage and very early cases are seen now and then coming to the clinics for treatment which even medical men find difficult to diagnose. A certain diagnosis depends on either of the two factors or of both.

- (1) The presence of hypopigmented anaesthetic macules and ;
- (2) The demonstration of *M. leprae* in the nasal or skin clip smear. To make a diagnosis, it is not enough to look for hypopigmented anaesthetic macules or leprous patches, thickened nerves, trophic disturbances and deformities but a thorough clinical examination of the whole patient is very necessary, with special reference to the following points :—

Search for any macule with altered pigmentation and sensation or areas showing a goose skin appearance ; thickened, hard, freely movable, painful or painless superficial and cutaneous nerves ; alopecia of the eye brows and eye lashes ; thickening of the ears ; ulceration of the nose ; infiltration of the nipples and enlargement of the mammary glands in males ; wasting of the intrinsic muscles of the hands, paralysis, contractures and deformities ; glossiness of the skin, generalised or in parts, solid oedema of the hands and feet, parakeratosis of the skin, hyperkeratosis of palms and soles with cracks and fissures ; trophic ulcers and blisters, necrosis and absorption of bones and lymphadenitis.

Neural lesions have to be diagnosed from :

Syphilis.—Syphilis is capable of producing both hyperpigmentation and hypopigmentation. Hypopigmented, discrete, macules appearing on the triangles of the neck and anterior axillary folds, particularly in women are pathognomonic of syphilis.

Pityriasis versicolor.—The lesions may be hypo or rarely hyper-pigmented. The macules are very superficial and limited to the lower part of the face, neck and upper part of chest, shoulder and arms, finely scaly and non-anaesthetic. The diagnosis finally depends on the demonstration of *Microsporon furfur* in the scrapings from the lesions.

Seborrhoea.—There is often an associated seborrhoea of the scalp. The skin is greasy, the scales are fine and they show *pityrosporon ovale*.

Vitiligo or leucoderma.—Except in the very early stage of development, there is no difficulty in diagnosis. In vitiligo the loss of pigment is complete and the macules are white and not anaesthetic.

Birth Marks.—Their presence from birth, their not increasing in size and the absence of anaesthesia are characteristic enough.

Morphoea.—The macules are ivory white in colour and the skin is sclerosed but not anaesthetic.

Dermal Leishmaniasis.—Hypopigmented patches with no anaesthesia, smears and cultures from the patches will show *Leishmania Donovanii*.

Trophic lesions and nerve affections have to be diagnosed from :

Syphilis.—Perforating ulcers of the feet are seen in neurosyphilis particularly *Tabes dorsalis*. Loss of ankle and knee jerks, Rombergism, Argyll-Robertson pupils and positive Wassermann reaction of the blood and cerebro-spinal fluid are in favour of syphilis. The changes in C. S. F. as increased globulin, albumin and cells and gold sol curve are not seen in leprosy.

Syringomyelia.—Here the thermal and deep pain sensations are lost but superficial sensation is intact, whereas in leprosy the superficial sensation is lost.

Raynaud's disease.—This condition tends to be bilateral and symmetrical, very rare, except in winter and anaesthesia is limited to the gangrenous area only. The evolution of the disease is also characteristic. Local syncope, local asphyxia, active hyperaemia and finally gangrene.

Hypertrophic interstitial neuritis.—In leprosy, trophic ulcers and ankle or wrist drop are common, whereas deformities such as claw hand and club foot are common in this condition. Anaesthetic macules do not develop. The nerve is very much thickened and the diagnosis depends on biopsy. In leprosy the nerve fibres are atrophied and degenerated but in neuritis there is hypertrophy of both the interstitial tissues and nerve fibres.

Bernhardt's syndrome.—This is characterised by an area of anaesthesia of the upper and outer part of the thigh in the distribution of the lateral femoral cutaneous nerve. Absence of any other changes in favour of leprosy and presence of conditions as syphilis, gonorrhoea or local sepsis which may cause fibrositis of the muscle and constriction of the nerve are in favour of the syndrome.

Progressive muscular atrophy.—Usually a disease of adults; bilateral though one side is a little more advanced than the other; progress of the disease is characteristic first, hand, then forearm, finally upper arm and shoulder; fibrillary tremors and no disturbance of sensation.

Diphtheritic Neuritis.—The history of diphtheria and the absence of thickened nerves and other signs of leprosy.

Peripheral or multiple neuritis.—Usually bilateral and symmetrical with loss of power, stoppage gait, tenderness of the calf muscles and evidence pointing to aetiology other than leprosy such as toxic, infectious, diabetic, beri-beri etc.

Cervical ribs.—Pain, numbness and tingling along the ulnar border of the forearm, wrist and fingers, slight sensory changes, wasting and paresis of intrinsic muscles of hand and weak radial pulse of the affected side. Skiagram will reveal the ribs which are usually bilateral though symptoms are unilateral.

Ulnar paralysis—due to injury or wound.—history of accident, claw hand, muscular wasting and sensory changes in the distribution of the nerve.

In all these conditions, the nerves are not thickened and there is no other evidence of leprosy.

Cutaneous lesions including nodules have to be diagnosed from:

(1) *Syphilis*. (a) *Secondary lesions*.—A generalised papulo-squamous rash may be mistaken for generalised erythematous infiltrated lesions of leprosy. In leprosy, *M. leprae* can be easily demonstrated in smears but in syphilis *sp. pallida* are in evidence in the serum from the lesions. Other signs of syphilis and the presence or history of a primary lesion and a positive Wassermann will be of help.

(b) *Nodular cutaneous*.—These syphilitic lesions are characteristic in their arrangement in circles or arcs of circles. No *sp. pallida* can be demonstrated, but leprous nodules will easily show *M. leprae*.

(c) *Broken down gummatous ulcers*.—These have punched out or clean cut edges with wash leather sloughy base. An ulcerated leprous nodule is a superficial ulcer showing *M. leprae* in smears and there may be other nodules or lesions of leprosy, in other parts of the body.

(2) *Ring-Worm*.—In the early stage the patches are very small and rapidly spreading and if of long duration, they assume large sizes. They always have raised, red, itchy, advancing borders

with vesicles and papules and clear or clearing centres. Scrapings from the advancing border will show the fungus.

(3) *Psoriasis*.—The sites of election:—anterior aspect of knees, posterior aspect of elbows, scalp and sacral region—and patches typical, large, silvery scales, minute haemorrhagic points on complete removal of scales, and slight infiltration. Absence of anaesthesia, absence of thickened nerves and absence of *M. leprae* in smears are favourable negative findings.

(4) *Lupus erythematosus*.—This is practically confined to face, ears and hands. It is a disease of adult life, and the patches show a stippled appearance and fine scaling.

(5) *Lupus Vulgaris*.—This is also confined to the face and upper part of the chest. The patches have a tendency to heal in the centre and spread at the periphery, characteristic apple-jelly nodules and tendency for healed parts to break down. Biopsy will show giant cell systems and tubercle bacilli.

(6) *Yaws*.—Here again the diagnosis depends on negative findings and the demonstration of *Treponema pertinax*, positive blood Wassermann and therapeutically the lesions disappear with the exhibition of one or two doses of neo-salvarsan.

(7) *Dermal Leishmaniasis*.—Infiltrated and nodular lesions are seen. Smears will show *Leishmania Donovanii*.

(8) *Neuro-fibromata or molluscum fibrosum*.—Soft fibrous nodules on the skin, sessile or pedunculated and varying in number and size. There may be patches of pigmentation and pigmented naevi. Biopsy will settle the diagnosis.

(9) *Erythema Nodosum*.—An acute onset, tender, hard, erythematous painful swelling confined to the front of legs. After a day or two the nodules soften and change colour as a bruise. The course of the lesion lasts only a week or ten days but fresh crops may appear.

Certain supplementary tests are of use in doubtful cases but they are not diagnostic. They are (1) Erythrocyte sedimentation test. (2) Immunological or specific tests. (3) Leprolin test or Mitsuda's skin reaction. (4) Histamin test.

Prognosis.—It is very difficult to give a correct prognosis in leprosy. Prognosis depends on many factors such as age of onset, resistance of the individual, freedom from other diseases, intelligent and willing co-operation of the patient, stage and phase of the disease, duration and nature of development of the disease, reaction of the tissues to *M. leprae*, and the mental outlook of the patient. In short, the prognosis is good in an early case of leprosy who is an adult of 20 to 30 years of age, who is active in

his habits, who lives under sanitary conditions, who takes regular and regulated exercise, who takes particular care of personal hygiene, who avoids all excesses, who takes a wholesome and well balanced diet rich in vitamins, who has a good general health and high resistance, who tolerates and thrives on the treatment, who co-operates intelligently and willingly with the physician and who has a cheerful and hopeful mental outlook.

Treatment.—Cases should be selected for treatment if possible. Early cases having a fair chance of recovery should only be selected. Early neural and cutaneous types can be treated in out-patient clinics. Mutilated and deformed cases, unable to move about and look after themselves, may require to be lodged in asylums and cared for. Highly infectious and advanced cutaneous cases who are able to do some work and look after themselves may be settled down in colonies or segregated in villages with arrangements for free treatment in the colonies or settlements. Patients with trophic ulcers, necrosis of bones etc. who require surgical interference and those with leprosy reactions require hospitalisation.

Personal Hygiene.—Leprosy patients should live in well ventilated and well lighted houses with sanitary surroundings and preferably isolated in separate rooms or huts. They should keep themselves clean as they are more susceptible to skin diseases such as scabies, ringworm, coccal dermatitis etc. They should have at least one bath a day. Their bedding, clothing and toilet appliances and eating utensils should be kept separate. They must also have separate arrangement for bathing and washing. Bowels should be kept regular as auto-intoxication worsens the condition. Regulated exercise, if possible under supervision, is essential. Regular hours should be kept and all excesses avoided.

Diet.—Diet is at least as important as the medicinal treatment. This point is often neglected and so may be stressed particularly. Many of the patients who attend the ordinary leprosy clinics are emaciated, miserable and dirty with the minimum of clothing. They are also under-fed—even starving at times—undersized and of underweight. It is no use injecting lepromolytic drugs to such patients without at the same time feeding them properly. Treatment in some such cases may even be harmful. It is very essential that leprosy patients should be given a balanced diet with fresh vegetables, milk and butter. No decomposed food should be allowed. Vegetable proteins, vitamins, mineral salts and fats, all should find a place in their dietary. In connection with out-patient clinics, arrangements should be made

to feed those patients attending the clinics from distant places at least once on treatment days.

The Associated or co-existing diseases.—These should, if possible, receive prior treatment or at least conjointly with leprosy. The common conditions met with are:—Skin diseases such as scabies, fungus and coccal infections etc.; gastro-intestinal disturbances, intestinal parasites particularly ankylostomes and round worms (more than 8% of the population are infested with these parasites); malaria, kala-azar, dysentery; focal sepsis particularly oral sepsis and syphilis. All these conditions should receive appropriate treatment. It is also advisable that the treatment should be given before commencing the anti-leprotic treatment as that may influence the resistance of the patient. In the case of syphilis, as prolonged treatment is required, it is not absolutely necessary to wait until anti-syphilitic treatment is completed. If at all, one course of about 8 to 10 weekly injections of neosalvarsan and an equal number of bismuth or mercury may be given prior to the beginning of anti-leprotic treatment. Otherwise both the treatments may be given concurrently as it is done in the clinic here. In cases of leprosy with associated syphilis, mercury is preferred to bismuth as the former can be given along with the hydnocarpus oil or its ester. Avenyl, a proprietary mercurial compound is used, 0.25 g. of it is dissolved in 100 c.c. of the creosoted oil or ester and the same can be given intramuscularly bi-weekly, the dose being 2 to 5 c.c. A course is about 15 injections. Avenyl does not dissolve in cold oil, so heat the oil until it is completely dissolved, filter and then sterilise.

Anti-leprotic treatment.—This treatment of leprosy is as old as the disease itself. The very same drug as used by the celebrated Ayurvedic physicians of old still holds the field with modifications and alterations to suit the modern tastes and convenience. Chaulmoogra fruit and its oil is the drug under reference. In olden times when time was not a very important consideration, people were sent to the forest where Chaulmoogra trees grew wild. There they lived a natural life with plenty of fresh air, sunlight and fresh food and in addition, ate the fruits of Chaulmoogra. It is a familiar story to all that a king of Benares abdicated in favour of his son as he was afflicted with leprosy, went to a forest full of Chaulmoogra trees, lived there comfortably, free from all worries, and got better by eating its fruits.

The oil used for treatment of leprosy is extracted from the seeds of *Taraktogenus Kurzii* which grows in Assam and Burma *Hydnocarpus Wightiana*, which grows in Kerala and *Hydnocarpus*

Anthelmintica which grows in Indo-China and Siam. The seeds should be properly selected using fresh and fully ripened ones only. The oil is extracted by cold process. It is filtered till clear and bottled with no air space and kept tightly corked. The oil is sterilised by heating to a temperature of 120° C. for 30 minutes. Esters and soaps from the oil are also in use. To the oil and ester 4% double distilled creosote is added before sterilisation.

Modes of administration.—The oil can be administered orally, rubbed externally or given by injections intradermally, subcutaneously, intramuscularly or intravenously. The oral administration and inunction are the old methods. Besides its slow action, it is unpleasant and nauseating to take the oil which is not tolerated in large doses which are necessary to be of any use. It can be given in gelatin capsules after meals and administered up to tolerance. Tourtoulis (1894) was the first to give the oil intramuscularly and it produced severe pain and local reaction. Then mixtures containing chaulmoogra oil were tried and one such mixture was Mercado's mixture consisting of 60 c.c. each of chaulmoogra and camphorated oil and resorcin 4 g. Very encouraging results were reported from its use. Rogers (1915) prepared sodium salts of chaulmoogra and hydnocarpus oil which are used, both intramuscularly (3% solution) and intra-venously (1% solution). Rogers also made ethyl esters of these oils which were improved by Deane and McDonald, who found the ester of the whole fatty acids more effective.

Intradermal injections are given into the patches direct.—There are special small intracutaneous needles which fit into the record syringes, and can be used with or without adapters to suit individual convenience. A record syringe with finger rest is preferred. A few drops only can be injected with every prick and a large number of pricks have to be made to cover a small patch. With every prick a small wheal is raised and yet another one adjacent to it so that both may overlap each other. This is painful to the patient and difficult to the Doctor with a large number of out-patients though it is more effective. The patches clear sooner. The pigment re-appears earlier. The oil acts as a local counter-irritant, besides its action, if any, on the bacilli locally.

Subcutaneous injections underneath the patch.—Here a fine long needle is necessary. After plunging the needle subcutaneously at one border of the patch, a small quantity of the oil is injected, then the needle is withdrawn almost completely and re-entered in another direction injecting at the same time, and so on till the whole affected area is covered.

Intra-muscular injection.—This is the method of choice. The upper and outer quadrant of a buttock is the site selected. Injections are given bi-weekly on either buttock alternately.

Intravenous injections.—Oil injections by this route are given with great reluctance for fear of producing pulmonary embolism. Intra-venous injections of the chaulmoogra oil are now commonly given as neutralised or emulsified with no bad effects. Some even think that this is more efficacious than any other method of administration.

Dose:—The hospital going patients, as already referred to, are undersized, underfed and of underweight. The average weight is below 100 lbs, and so the dose must necessarily be small. It is advisable to begin with 1 c. c. and slowly increase it by $\frac{1}{2}$ to 1 c.c. every time up to 5 c. c. twice a week. After reaching the maximum dose the same may be kept up. In the case of children, the dose is $\frac{1}{2}$ to 3 c. c. bi-weekly according to age.

Mode of action.—How these oils and their preparations act is not definitely known. Clinically and bacteriologically, the patient shows improvement. It is considered that the injections of fatty acids increase the lipase content of the blood which in turn acts on the lipoids of the bacilli, which are broken and destroyed. In support of this view, Rogers and Muir have found that the lipase content of the blood after a reaction, when there is great destruction or granulation of the bacilli, is very much reduced. The lipase is again raised to its usual level when the patient regains his normal state. Experimentally it has been found that the growth of acid-fast bacilli in the presence of these oils and their preparations, are inhibited. Further they are lepromalytics, as lepromata are found to be softened and reduced in size and as they also produce reactions.

Other treatments.—A large variety of drugs, vaccines, sera, antigens etc. have been used with doubtful benefit to the patient. Some of them may be useful in conjunction with the oil and at times as an alternative. Some of them are mentioned here.

Heavy metals. (1) *Antimony.*—Potassium or sodium antimony tartrate, colloidal antimony, stibenyl, urea stibamine etc. Muir advocates intravenous injections of antimony tartrate 0.02 to 0.04 g. on alternate days to control the reaction and reduce the high sedimentation index.

(2) *Gold.*—Sanocrysin, krysolgan, triphal, aurophos, solganol, solganol B oleosum and gold sodium thiosulphate. Feldt

considers that they act "by stimulating the natural defensive process through the reticulo-endothelium."

(3) *Copper*.—Cyano-cuprol, colloidal copper, copper cyanide, cupriiodose, copper citrate etc. Reduced copper is a recognised treatment according to the ancient Ayurvedic system for incurable diseases including leprosy.

(4) *Mercury*.—Perchloride of mercury, calomel cream, avenyl, and mercurio-chrome. These are particularly useful in those cases complicated with syphilis. Mercurio-chrome 1% in normal saline 2 to 10 c. c. intravenously is useful in controlling lepra reactions and those complicated with sepsis.

(5) *Arsenic*.—Atoxyl, sodium cacodylate, arrhenal, eparseno, salvarsan, neosalvarsan, silver salvarsan etc. These are useful more in cases associated with syphilis.

(6) *Lead and Silver*.—In general, the heavy metals in large doses stir up lepra reactions; in well regulated doses they may be beneficial; and in small doses they control the reactions.

Other drugs.—Potassium chlorate in large doses, thyroid extract, sodium salicylate, sodium thiosulphate, creosote, thymol, guaicol, resorcin, salol, etc. are also tried with doubtful benefit.

Aniline dyes.—Of late, all kinds of dyes are being tried. Trypan blue, methylene blue, brilliant green, methyl violet, eosin, fluorescein and many others. Methylene blue neutral is more extensively used. Montel recommends 1% solution of it intravenously on alternate days sterilised at 80° c. in a water bath for one hour at a time for three consecutive days. The dose is 5 c. c. to begin with and gradually increased to 25 to 35 c.c. for about 20 weeks, when the oil may be given. The cutaneous lesions are stained by the dye and so they stand out clearly. Thus the treatment is also of diagnostic value. The injections control the reactions; neuralgic pains, headache and general malaise pass off; appetite gets keener, general condition improves, the patient sleeps better and feels generally well. Besides, he has a mental satisfaction as he sees the cutaneous lesions becoming blue.

Iodine.—Iodum is administered dissolved in the oil or ester. It was so used first by the Philippine workers to make the oil and ester less irritative and less painful. Potassium iodide was first used for diagnosis and treatment by Danielssen in 1886. Its use is very much limited now, as it is only used as a test of cure and that in large doses. Potassium iodide also sensitizes the patient. It produces lepra reactions when given in conjunction with other

anti-leprotic drugs which, by themselves even in large doses, are not capable of producing the reaction.

Sera and Vaccines have also been tried. The following are some of them:—Normal horse serum; serum prepared after injecting horses with blood from leprosy patients, leprosy juice, snake-venom, etc. antivenine: tuberculin—original and autolysed; nastin, an ethereal extract of cultures from a non-specific acid-fast streptothrix cultured from leprosy lesions; anthrax vaccine; leprolin etc.

Pyrexial treatment.—Injections of turpentine, sulphur-in-oil, milk and its preparations, T. A. B. vaccine intravenously.

External Applications.—(1) Injection of chaulmoogra, hydnocarpus, gurjan, kuranjee and neem oils; and salicylic acid, ichthyol, resorcin and pyrogallol acid in the form of ointments.

(2) Caustics.—Trichloroacetic acid 1 in 1 to 1 in 5 is painted on the patches depending on the site, weaker solutions being used on the face and other areas of soft skin, carbondioxide snow, marking nut juice (*semicarpus anacardium*).

Treatment with light and electricity.—General or local irradiation with ultra violet rays. Irradiated hydnocarpus oil will be more effective. X-rays, radium, galvanism and high frequency currents have also been tried.

The Routine treatment in the Vizag Clinic.—For the last five years, the treatment adopted here is intramuscular injections of equal parts of oleum hydnocarpus and its ester with 4% double distilled creosote. The Madras Medical Stores supplies both the oil and its ester with 4% double distilled creosote sterilised at 120° C for one hour. These are mixed in equal parts and filled in two ounce stoppered bottles and then autoclaved. Two ounce bottles are particularly selected so that much of the mixture is not left behind for another treatment day, after it is once opened. This is important as by keeping open a two-pound bottle for a long time using only small quantities at a time, the sterility of the oil may be doubted by the time it is finished. Creosote is added to make the injections less painful and not to keep the oil or ester sterile. Small bottles are just warmed before giving the injection and emptied into a warm sterile porcelain or glass dish from which the mixture is drawn into a syringe. A steriliser is kept going the whole time injections are being given. The needle after every injection is sterilised and from the steriliser it is transferred to a dish containing rectified spirit from where the needle is taken and held between the thumb and fore-finger, then stabbed on to the upper and outer quadrant of one of the buttocks of the patient who is standing leaning on a table. The injection area is painted

with tincture of iodine. The syringe with the required quantity of the oil-ester mixture and one c.c. of air is attached to the needle and injected slowly. The air clears the needle of the mixture. While the needle is being withdrawn, the skin and the subcutaneous tissue around it is pinched so that the needle track becomes obliterated soon after the needle is removed, thus giving no chance for the material to escape. The part is again swabbed with tincture of iodine and thoroughly massaged with a pad.

For a change, 3% alepol with 0.5% acid carbohc 1 to 5 c.c. bi-weekly is given. Colloidal copper 1 to 3 c.c. three times a week is also under trial. Methylene blue 1%, Mercurochrome 1%, antimony tartrate 2% in 5% glucose are all given intravenously during reactions. Those whose blood Wassermann is strong positive are given anti-syphilitic treatment conjointly with the oil injections. Externally trichloracetic acid in different dilutions is painted over the patches.

Accidents and intolerances to treatment.—(A) *At the time of injection:*

(1) The needle may strike bone. Withdraw the needle slightly and then give the injection.

(2) Blood may come through the needle. This is due to the needle piercing a vessel. This will not be detected if the needle is plunged into the muscle after fixing it to the syringe. So, it is very necessary that the needle should be stabbed first and after a few seconds to see if there is any bleeding through the needle. If there is any, the needle must be removed and stabbed in another place and the injection given. Sometimes blood is seen at the site of puncture when the needle is removed after the injection. This is due to puncturing a superficial vein and no bleeding takes place so long as the needle was in its place. Pressure and massage will stop it.

(3) Patient may complain of pain at the site of injection and radiating along the thigh to the leg. This is due to injury to a nerve twig. Here also the needle should be removed and planted at another site before giving the injection.

(4) The needle may break. If part of the needle is projecting outside or just palpable, attempt to remove the same may be made. If it is deep in the muscle it is better to leave it alone. Attempt to remove the needle by cutting down on it should not be undertaken light-heartedly as the needle may move away by the time it is reached.

(B) *After 8 to 12 hours of injection:*

(1) Pain and infiltration about the site of injection. Hot fomentations and gentle massage will give relief.

(2) Leprea reactions:—Some of the patients now and then get these reactions accompanied by general constitutional symptoms, fever and development of new lesions and accentuation of the existing lesions as already described. This is not due to overdosage or defect in treatment. The reaction occurs whether the patient is under treatment or not. The treatment is stopped for the time being and appropriate treatment for the reaction given.

(3) Development of abscess:—Rarely, at the site of an injection, an abscess develops. It takes a long time for it to point. First there is hardness, infiltration, pain, heat and tenderness over the area. The pain increases, the infiltration and hardness decrease and a soft fluctuating swelling develops. As the focus of suppuration is deep, it takes time for the abscess to reach the surface. Local application of Ichthyol and glycerine or antiphlogistine and hot fomentations will hasten the process. When the abscess has come to the surface, a small incision should be made at the most dependent part and drained. Through the incision a sinus or artery forceps may be introduced and dilated. A long incision is not necessary as healing takes place rapidly. Why this abscess develops it is difficult to say. Only thrice did this complication occur here during the five years, when not less than 25,000 injections were given. Probably the rundown condition of the patient, malnutrition, lowered resistance, overdosage, and trauma at the site of injection, may individually or conjointly be the cause.

(4) Toxic Jaundice:—When the treatment is prolonged and continuous, jaundice develops in some cases. The treatment should be stopped and jaundice treated on usual lines.

Length of treatment.—No hard and fast rule can be laid down. Each case has to be considered on its merits. It must be remembered that leprosy is a very chronic disease and recrudescences other than lepra relations are not uncommon. The treatment should be continuous and prolonged. It should be continued till all the patches disappear, till thickened nerves become normal; till repeated bacteriological examinations for *M. Leprae* are negative. No new patch or infiltration, pain and tenderness of superficial nerves should have appeared during this period. This state of affairs should continue for not less than 6 to 12 months before the patient can be discharged provisionally.

He should be then under observation for another 12 to 24 months. During this period of probation, he should be examined at regular intervals of two to three months bacteriologically and clinically or whenever any new patches or symptoms arise. If everything goes on satisfactorily during these two years then his discharge may be considered as "disease arrested" or "symptom-free."

Treatment of complications and special conditions:—

(1) *Lepra re-action*.—Patient should be put to bed and given a saline purge. Diet must be restricted to fluids. Alkalies and calcium by mouth or intravenously are indicated. The following intravenous injections have been recommended by various authorities. Antimony tartrate 0.02 to 0.04 g. on alternate days, mercurochrome 1% daily 3 to 10 c. c., 2% acriflavine 2 to 5 c. c., 1% methylene blue 5 to 10 c. c., 10% solution of sodium thiosulphate 5 to 10 c. c., saturated solution of sodium bicarbonate 20 to 25 c. c., daily for a few days and 2% calcium chloride 20 to 30 c. c. daily for a week.

(2) *Nerve pains during a re-action or at other times*.—Subcutaneous injection of adrenalin hydrochloride 1 in 1000, 5 m. or ephedrine subcutaneously or by mouth will relieve the pain in about half an hour. Subcutaneous injection along the course of the nerve of sod. bicarb. solution in saline is also recommended. A thickened nerve, particularly the ulnar nerve, is sometimes bound down by adhesions or the swelling caused by the infiltration may strangle the nerve and cause pain. In such cases the nerve should be exposed and the tissues cut down, or a linear incision on the sheath of the nerve may be made and the exudation let out. A nerve abscess has to be opened and drained and the wound sutured as the abscess is not generally infected.

(3) *Surgical interference* may be required when—

(a) The ears have become nodular, enlarged, elongated, and thickened. The ears can be trimmed for cosmetic purposes and there is a special forceps designed for the purpose.

(b) Perforating ulcers and chronic sinuses. If there is a sequestrum or dead bone, that has to be removed and the sinuses scraped. In some cases amputation may also be called for.

(c) When there is obstruction and stenosis of the larynx, tracheotomy may be necessary.

As the affected parts are anaesthetic, no anaesthesia is required, and if at all, local anaesthetic will be sufficient. General anaesthesia should be avoided as far as possible.

(4) *Eye complications*.—It is always advisable to consult an ophthalmologist in all such cases. Iritis and corneal ulcers are the common complications. Both require atropine drops. For iritis, sub-conjunctival injections of 0.1% trypan blue in normal saline is recommended. This treatment was given to three patients here and of these, one recovered completely, another had partial benefit and the third showed no improvement at all. Injections of yeast (*Ascomycetes*) is considered to be very beneficial by some observers.

(5) *Perforating ulcers*.—Any slough, necrosed bone or gangrenous tissue should be removed. Thickened skin around the ulcer is shaved and made level with the ulcer, which is dressed antiseptically. Painting the ulcer with 10% iodoform in acetone is recommended. The patient should keep the weight off his feet and so he must move about only on crutches. In bad cases amputation may be necessary. Peri-arterial sympathectomy hastens the healing of the ulcer which is likely to break down again. Ultra violet ray exposures, general or local, also are helpful. We have been trying the following method of treatment with encouraging results. The ulcer is cleaned and the thickened edges pared or shaved. Then the ulcer is filled with pure cod liver oil and sealed with a paste made of the same oil and an inert powder as zinc oxide or calamine. Sterile gauze is placed over this and a tight bandage or strapping applied. The dressings are changed once in a week and the ulcer heals fairly rapidly. Sometimes the ulcers are infested with maggots. There is no hurry to remove them as they remain there only till the sloughs have disappeared. Ulcers infested with the maggots heal sooner.

(6) *Deformities and contractures*.—These should be corrected by manipulation and appropriate splinting. Encouraging results have been reported by massage, diathermy and electrical treatment.

Prophylaxis.—Compulsory segregation of all leprosy cases is the ideal to be aimed at. But it is not practicable as there are about two million lepers in India. But modified forms of segregation may be practised.

(1) *Home segregation*. Early cases can be segregated in separate rooms or cottages in the patients' own houses with restrictions that they should never handle or come in close contact with children, that they keep separate eating utensils, bedding and clothing and that special arrangements are made for bathing, washing etc.

(2) Establishment of (a) Special hospitals or special wards in general hospitals for patients with perforating ulcers, chronic sinuses etc., that require surgical interference and for those with reactions. (b) Colonies or settlements for able bodied infectious cases with arrangements for free-medical attendants. (c) Asylums for disabled, deformed and maimed cases.

(3) Separation of sexes and prohibition of marriage. But persons suffering from leprosy may be permitted to marry among themselves if the male will allow himself to be sterilised.

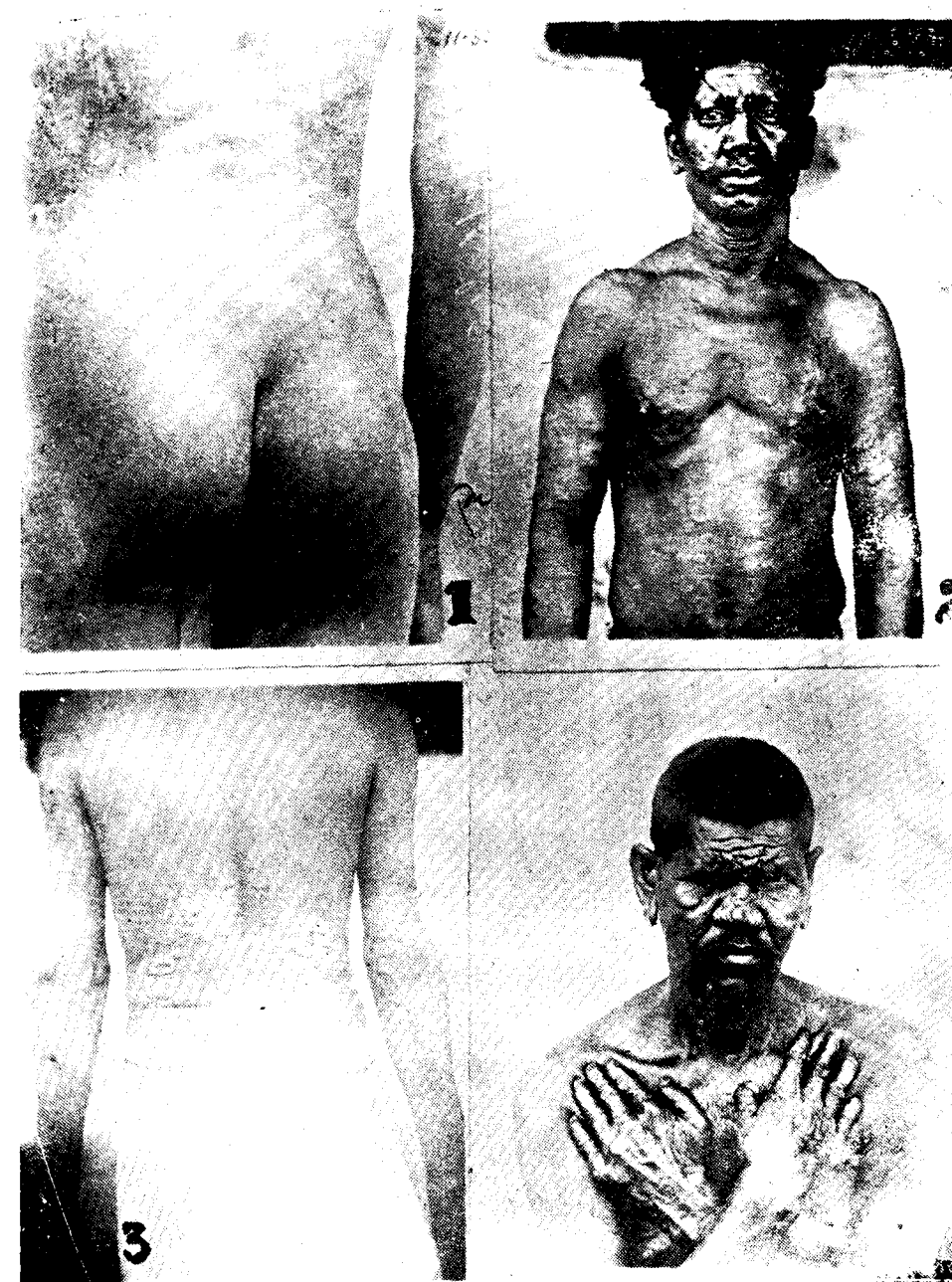
(4) Separation of all children from their leprous parents. The tainted and untainted children should also be kept separate.

(5) Inspection of all school children to detect leprosy. The inactive cases should be periodically observed, active neural cases should receive treatment and infectious children should be prevented from attending schools. Attempt should also be made to inspect the homes of infected children to detect infected contacts. Personally, I would prefer exclusion of persons suffering from leprosy of all types, both teachers and pupils, from all schools and if their number is large enough, separate schools should be provided for them. It is not uncommon to see the so-called non-infectious neural cases suddenly developing infectious lesions and showing *M. leprae* in skin and nasal smears.

(6) For the same reason, persons suffering from leprosy whether neural or cutaneous should be prohibited from being engaged in certain occupations such as handling of foods and drinks, manufacture and sale of clothes, cigars, cigarettes and beedies, care of children and the sick, in those of domestic servants, hospital servants, doctors, nurses, midwives, teachers, lawyers, engineers, ayas, wet nurses, barbers, washermen, prostitutes, temple servants and other occupations which bring them in close contact with the public.

(7) Country-wide organization to treat early cases by well trained *sympathetic* and *willing* doctors. Cases should be selected and only early cases should be dealt with. It is harmful to collect and huddle together all sorts of cases in treatment rooms, highly infectious cases disseminating bacilli every minute of their existence to remain by the side of children who are only doubtful or early neural cases. It is doubtful whether the recognised treatment can cure all cases of leprosy such as the early, the advanced, the burnt out, the deformed and maimed. For the sake of impressing the good effects of treatment, so that it may not fall

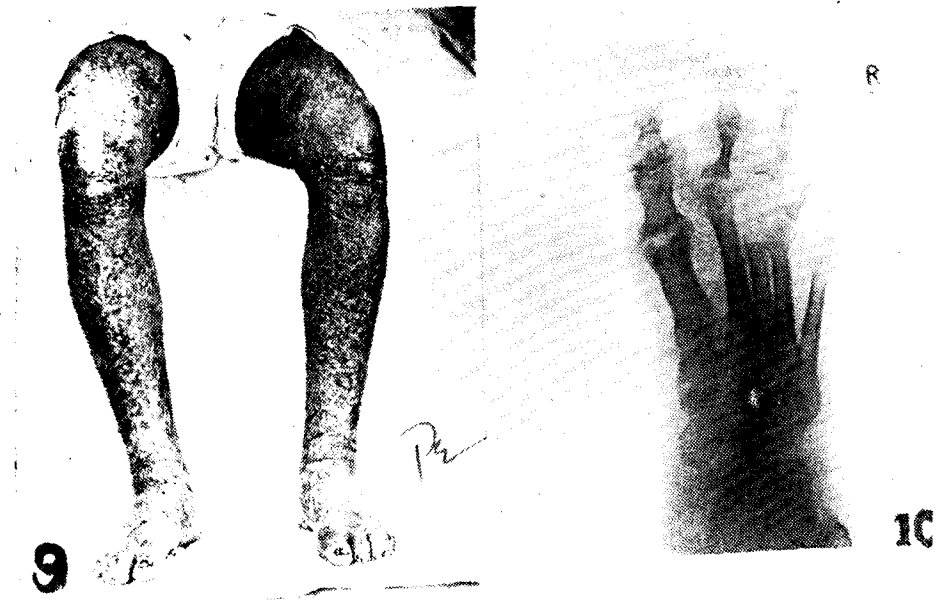
LEPROSY ITS DIAGNOSIS AND TREATMENT—By V. Govindan Nair.



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Illustrations.

- Fig. 1. Hypopigmented anaesthetic patch. Note the radial spread and the goose-skin appearance of the skin beyond the patch.
- Fig. 2. Raised erythematous patches and infiltration of nipples.
- Fig. 3. Lesions showing three distinct zones—hypopigmented anaesthetic centre raised red non-anaesthetic middle zone and hypopigmented anaesthetic border.
- Fig. 4. Showing elongated, enlarged and thickened ears, leonine facies, alopecia of the eye-brows and face, thickened stunted fingers and enlarged mammary glands.
- Fig. 5. Enlarged mammary glands in a male leper.
- Fig. 6. Facial paralysis in a leper.
- Fig. 7. Showing marked wasting of the intrinsic muscles of the hands and contraction of the fingers.
- Fig. 8. Perforating ulcer of the heel. Note the mutilation of the little toe.
- Fig. 9. Parakeratosis of the skin of the legs and oedema of the legs and feet. Note the changes in the nails.
10. Absorption of bones without any external manifestation.

into disrepute, cases for treatment must be selected. The rest may be disposed of as detailed above. Patients come for treatment from distant places and it is highly necessary that some resting place should be provided for them to avoid their wandering about in the locality. Otherwise these treatment centres instead of being healing centres become centres for dissemination of the disease.

8. The feeding of these patients should also be considered as most of them are poor.

9. Educate the public by popular lectures, lantern slides, and cinemas with special reference to infectivity, prophylaxis and early treatment at recognised treatment centres. Similar lectures should also be given in all schools and colleges.

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Some Aspects of Leprosy

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Introduction.—Leprosy and its ravages on mankind have been known since remotest antiquity, mention having been made of this dreaded disease in the Rigveda, Manusamhita, old Egyptian, Greek and Chinese writings. It is a wide-spread disease, prevailing usually in tropical and sub-tropical climate and is one of the major tropical diseases in India, China, Japan, Mexico, Phillippine islands, certain parts of South America, Sandwich Islands, Norway, Egypt and Abyssinia. We do not as yet know definitely why incidence of leprosy should have natural rise and fall in different countries and communities. On account of the mystery surrounding the aetiology and for want of any specific treatment, it has long been dreaded as a loathsome, incurable disease, the morbidity is huge and to the sufferer even death seems better in comparison to the great physical suffering and mental agony caused by it. But in recent times, the disease has been made extinct in many countries in the West by proper segregation etc. Thanks to the inauguration in 1924, of the British Empire Leprosy Relief Association and its branches, it has given rise to a new impetus towards systematic anti-leprosy campaign and we have now better knowledge and experience than before in combating this scourge.

Leprosy is a variety of specific Infective Granulomata of the group of tuberculosis, syphilis, glanders etc., and as such, the lesions caused by it are the result of subacute or chronic inflammatory changes associated with various protective and reparative processes in the tissues involved. The causative organism, *Mycobacterium Leprae*, discovered in 1872 by Hansen, is gram-positive, acid-fast, pleomorphic and of low toxicity. (1.) As the biology and bionomics of this organism are still mysterious, (2) as it does not fulfil two of Koch's postulates (it is not successfully culturable in vitro nor inoculable in any laboratory animal) and (3) as the mode of transmission is still baffling all research work, some workers even go so far as to entertain a doubt as regards the genuineness of *M. Leprae* being the infective organism. The *M. Leprae* have got a peculiar selective action for skin, mucous membrane and nerve tissue. Leprosy resembles to a great extent, its sister disease, tuberculosis, in as much as the morphology and

staining reactions of the causative organisms in both, are to some extent, similar: both belong to the group of specific Infective Granulomata; both are causing havoc in our country, forming a major public health problem; both are usually chronic or subacute processes; both are more or less self-limited diseases without as yet any specific treatment; in both, prognosis is good if detected early and subjected to modern treatment; in both improvement of general body resistance is the sheet anchor in the treatment.

Clinical Types and Classification.—The infection may be latent in a person for a very long time. The signs and symptoms of the disease appear only when the tissues react to the bacilli. Broadly, two types of manifestations are found in Leprosy, (a) Cutaneous or Nodular and (b) Neural, depending upon whether the selective action of the bacilli fall on either of these tissues. Muir classified all cases of Leprosy broadly into Nerve type (A), (cases with anaesthesia) and skin type (B) (cases where lepra bacilli are found). These again were subdivided into A_1 , A_2 , B_1 , B_2 and B_3 , according to severity. The Manila Conference in 1931 preferred to call Neural (N) and Cutaneous (C) types with N_1 , N_2 , N_3 and C_1 , C_2 , C_3 , gradations according to severity. Usually except in the early stage, all cases of Leprosy are however mixed of Neural and Cutaneous types in varying degrees. In Neural type, lesions are (I) Macules which are anaesthetic to light touch, raised and indurated, anhidrotic, depilated, erythematous and Hypopigmented and (II) Thickening of nerves and consequent symptoms of Neuritis. In Cutaneous type, the lesions are (I) Cutaneous macules which are sensitive to light touch, flat and soft, slightly or not at all erythematous and (II) Diffuse infiltration of skin, nodules, ulcers etc.

Infectiousness and transmission.—Though the route through which the organisms enter the human body still remains unknown, there is not the slightest doubt that Leprosy is an infectious disease. It has been proved beyond doubt that contact can certainly transmit the disease from one person to another. The more prolonged and close the contact the more are the chances of infection. The fact that the organism cannot be cultured in Vitro nor inoculated in any laboratory animal as also the long incubation period, greatly stand in the way of solving the problem of transmission. Whether there is any intermediate host or not, we do not know as yet. The neural type of cases are much less infectious than cutaneous cases. Because in the former the bacilli are few and they remain embedded in the nerve

only, whereas in the latter the bacilli are much more and lie on the surface more or less. In the later stages of the disease the so called arrested or burnt out cases are very little infective. Overcrowding, insanitary conditions, industrialisation, modern ways of transport by rail or road, uncontrolled system of begging by frankly open cases of Leprosy, are potent factors in the spread of the disease. Cinemas and restaurants also help in the spread of leprosy.

Some outstanding facts as an outcome of recent researches on leprosy:—

(1) Incidence of the disease is much more than formerly thought of. 'Ignorance of the earliest appearances, shame lest others should know of the presence of this loathsome disease, fear of loss of employment and social ostracism' lead to concealment on the part of the sufferer.

(2) Climate, rainfall, humidity etc. are not primarily responsible for the incidence of the disease.

(3) It is most prevalent among semi-aboriginal tribes with insanitary mode of living. They may again infect better class people.

(4) It is not a hereditary disease; close and prolonged proximity is responsible for infection of children of leper parents.

(5) Children are particularly susceptible to the disease and majority of infections occur in childhood.

(6) Malnutrition and debilitating conditions (often caused in our country by Malaria, Kala-Azar, Ankylostomiasis, venereal diseases etc.) are the most important predisposing causes for the soil to be prepared for implantation of the seed of Leprosy.

(7) Neural type is much more common and benign than Cutaneous or Nodular type which is more infectious and malignant.

*Regional Variations of Leprosy:—*Recent researches have shown that Leprosy like Malaria, has a tendency to show characteristic variations in its manifestations in different regions. This difference has been ascribed by Muir (1934) and Hyashi (1935) to climatic distinction, whereas Cochrane (1935) and Ryrie maintain that racial distinction is responsible. The truth however seems to cover both the factors more or less. Some of the instances of these variations are: (1) Neural type is much more prevalent in India, particularly Calcutta, whereas Cutaneous type

in the Philippine islands; 2) again, differences in type proportions have been shown to exist in different parts of India alone; (3) eye involvement in Leprosy and leprous Alopecia have been found to be most prevalent in Japan. In this connection, it will be interesting to note the following peculiarities of Leprosy in India:—

(1) Neural type more prevalent than Cutaneous type.

(2) Presence of diffuse cutaneous leprosy-bacilli can be found in almost any part of the body though there may be no signs of definite infiltration. (This has been ascribed by Muir to lack of general and local reaction to the bacilli due to lowered resistance).

(3) Presence of Tuberculoid lesions:—(Neural type). A peculiar infiltrated skin lesion in leprosy characterised by:—(a) Negative or scantily positive bacteriological examination, (b) Centrifugal progress of the lesion with abrupt demarcation from the healthy skin, (c) Outward character of the infiltration often raising above the surrounding skin. (Wade)

Diagnosis:—Well developed cases with maimed limbs, atrophied digits, horrible trophic ulcers and disfiguring patches on the body, nodular masses on face and ears, as are frequently seen in most cities in India, are easily diagnosed even by the layman in the street. Difficulty lies in diagnosing early cases. The more early the case, the greater is the difficulty in the diagnosis. But importance of early diagnosis in Leprosy cannot too strongly be stressed, as it is only in the early stage that the disease is most remediable, as also proper segregation in this stage prevents the chance of spread. As already mentioned, ignorance, shame and fear on the part of the sufferer, are great obstacles in early diagnosis of leprosy. The two cardinal points in diagnosis are: (a) finding of *M. Leprae* in the skin or mucous membrane in skin type; (b) presence of superficial anaesthesia in nerve type.

The bacilli can be found out:—

(1) from a nasal smear only in cases where the nose and its surrounding parts are affected;

(2) from thick surfaces of the body e.g. back; a small incision is made (slit method) over the edge of the suspected patch just deep enough for the blood to ooze and not flow; the bottom of the wound is then scraped with the edge of the scalpel and a smear made;

(3) The method of choice, however, is to clip (clip method) a small piece of the skin with a pair of medium-sized scissors curved on the flat and making the smear by rubbing the raw surface of the piece on the slide.

Leprosi bacilli are seldom found in the superficially anaesthetic skin; they are often found towards the edges of fresh active lesion with raised erythematous margin (may or may not be totally or partially anaesthetic). Likelihood of finding the bacilli depends more upon the age of the lesion and its depth in the corium and sub-cutaneous tissue rather than on the amount of swelling and apparent inflammation. To stain the bacilli, Ziehl-Neelsen method is adopted. Presence of superficial anaesthesia can best be elicited by testing the light touch sensation with a folded piece of paper, the patient being blind-folded and asked to point out with his finger the part of the skin touched.

Patchy anaesthesia is most commonly found in the distribution of the ulnar and peroneal nerves. Apart from anaesthesia, Paraesthesia (due to overlapping of the nerve supply of the skin) and Hyperaesthesia (usually preceding anaesthesia) may be detected and are suggestive.

Besides these two cardinal points, there are some subsidiary signs; presence of two or more of these may lead to a strong suspicion. These are, deep analgesia (with or without superficial anaesthesia), loss of sensation of heat and cold, depigmentation, thickening of superficial nerve trunks e.g., ulnar, peroneal, great auricular, supra orbital, etc.; erythematous patches, parakeratosis, anhidrosis, interfollicular swelling, dry rhinitis, irregular pyrexia accompanied by vague nerve pain and trophic blisters and ulcers.

If what is said above is remembered, differential diagnosis of leprosy is not a matter of difficulty at all.

Prognosis:—Except the very few fortunate cases that are early detected and effectively treated, it may be said "Once a leper always a leper". Even the so called burnt out or arrested cases are sometimes seen to relapse subsequently. Though not a very fatal disease, the sufferings are horrible and usually some intercurrent disease carries away the patient. Neural type is relatively more benign than Cutaneous type. Marked thickening of nerve usually indicates good resistance and consequently speaks of good prognosis. When a definite predisposing cause, which can be effectively treated, is found, the prognosis is usually favourable. The blood Sedimentation test helps a good deal; the lower the Sedimentation Index, the better is the prognosis. A strongly positive Leproline test usually indicates a favourable prognosis as it speaks of resistance to the infecting organism.

Modern treatment.—Leprosy is more or less a self-limited disease and we have as yet no specific for it. The long continued

course of treatment, requiring great perseverance, insanitary surroundings, poverty, debilitating conditions etc., are obstacles in treating these cases successfully. Only the early cases are most amenable to treatment which can also ameliorate the sufferings of an advanced case. Like the sister disease tuberculosis, general treatment in the shape of nourishing diet, fresh air, rest, regulated exercise, psychotherapy etc., is certainly more important than special treatment with Hydnocarpus oil and its preparations. Muir thinks that on the whole, treatment of leprosy is, taking cases in similar stages, easier and more effective than that of tuberculosis; but the relative importance of general and special treatment should be considered the same in both the diseases. Treatment of the predisposing and intercurrent diseases, if any, and strengthening the body resistance are of outstanding importance. Neural type of the disease, which is so common in adults responds better to treatment than the cutaneous type.

Special treatment.—A host of remedies have been tried from time to time to combat the disease but only Hydnocarpus oil and its ethyl esters have stood the test of time. 4% creosoted Hydnocarpus oil, from ripe fresh seeds or 4% creosoted ethyl esters once or twice weekly, in gradually increasing doses according to the reaction, (2 to 10 c.c.) intra-muscularly or sub-cutaneously under the lesions, is usually given. Recently introduced Intradermal Injections (Plancha method) have been reported to yield most satisfactory results, but as large quantities are rather difficult to be administered this way, the method of choice is to supplement the intradermal injections by intramuscular or sub-cutaneous route. For the intradermal infiltrations, the esters are injected into the actual lesions in the skin by means of multiple small punctures raising a wheal not more than one centimetre in diameter. The difficulty in putting the oil intradermically due to its viscosity can be overcome by heating the creosoted oil on a water bath at 55° C. before drawing into the syringe. Alepol (B. W. & Co.) (a sodium salt of the low-melting-point fatty acids of *H. Weightiana*) has been reported to have given satisfactory results. Its advantages are, the ease of injection, its cheapness and safety (keeping alepol for a long time or coming in contact with carbolic acid or alcohol may give bad pain and reaction due to precipitation of fatty acids). In treating syphilis in lepers, the organic arsenicals should be given with great caution and judgment as they sometimes give very bad lepra reaction, causing a flare up and may be very dangerous to the eyes particularly.

Painting the local lesions with trichloroacetic acid is usually

beneficial. Complications *e.g.* ulcerations etc. should be met with the usual surgical procedures according to necessity.

Control of Leprosy in India.—In our country, ignorance, poverty, peculiar social customs, unhygienic conditions, debilitating diseases *e.g.* Malaria, Kala-azar, Ankylostomiasis etc. form great obstacles in the way of prophylaxis of leprosy. Education of the public regarding sanitary measures in general and the recognition that a transmissible disease like leprosy can be controlled by preventing contact with infectious cases, are essential. This can be, to some extent, done by systematic propaganda by posters, pamphlets, lantern lectures in vernacular etc. A survey of the incidence of the disease is of outstanding importance before an effective anti-leprosy campaign can be started. Research into the still unknown aspects of the disease is also as important in such a campaign. Training of Doctors in regard to early diagnosis, prevention and modern treatment of the disease forms an important item. A successful anti-leprosy campaign needs the sympathetic and active co-operation between the Government, Public Health Department, Local Bodies and the Public, as also the voluntary co-operation of the sufferers. The Indian Council of the British Empire Leprosy Relief Association has been doing very useful work in these lines to stamp out leprosy in this country for the last one decade or so of its existence. In endemic areas prevention of leprosy should form a major problem like tuberculosis or malaria, to the Public Health Department. Diagnosis and treatment of early cases is of paramount importance. Open or frankly infective cases are a positive danger to Society. Control of such cases coming in close contact with healthy individuals especially children that are particularly susceptible to the disease pivots round the problem of the prevention of leprosy. Spread of leprosy can, to a great extent be controlled if open cases can be kept away from healthy persons. In other words, their utensils, beddings, clothings, furnitures, and bedrooms must be kept separate. In our country, due to ignorance, superstitions and social ostracism, sorting out of symptomless open cases is very difficult and such strict isolation of all those cases is even more difficult and that is the reason why incidence of leprosy is so alarmingly high here. Legislative control urging compulsory segregation is practicable only when the disease is limited and as such is not feasible in a vast country like ours. Difficulties in such a procedure will be:—(i) huge expense involved, (ii) difficulty to sort out all open cases and (iii) chances of concealment.

As to the very important question of segregation, general

isolation of all lepers is not practicable in a vast country like India. The wiser method of isolation and treatment may be carried out in the following three different types of institutions:—

(1) *Out-patient clinic.*—Prevention should be the first object and treatment, the second. Treatment should be given to only early cases and not to advanced reacting cases, as the latter are not likely to be much benefited and may bring on a bad reputation. Examination of contact, survey and propaganda work should better be carried out under the influence of these clinics. The number of these clinics in a province should vary according to the proportion of incidence of the disease.

(2) *In-patient Hospitals.*—For the purpose of isolation of open cases amenable to treatment and for the treatment of active neural cases in children, a sanatorium sort of arrangement such as good food, fresh air, rest and regulated exercise can be arranged here. These institutions may be few in a province according to necessity and may have a controlling and associate influence on the out-door clinics. Advantages of these are:—

(a) Segregation of infectious cases.

(b) Arrangement for better treatment including that of complications.

(c) *Psychotherapy.*—By encouragement and suggestion an atmosphere may be created where the patients may forget the gloomy view of the disease and bring about their moral and mental welfare.

(3) *Leper Homes.*—Advanced cutaneous and chronic mutilated arrested cases may be isolated in a colony (solely philanthropic for the helpless and destitutes, similar to the Mission Homes).

Farm colonies for arrested cases without any disabling deformities may be started also.

Infants and children of infectious leper parents ought to be isolated as soon as possible and helpless ones may be accommodated in a separate "Home", where they can be kept under proper care and observation.

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Prevention of Leprosy

BY

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THERE are still many aspects of leprosy which have yet to be elucidated. But we can take a birds'-eye-view of the problem in a way which was impossible ten or fifteen years ago. There has evolved a new outlook as a result of finding increasing number of cases with early and slight lesions, for example, to-day the general trend of opinion amongst leprosy workers is:—

(1) That leprosy is an infectious disease and is acquired by the non-leper, from a leper, by close contact with the sufferer of this disease or by the use of articles soiled by the patient.

(2) That only certain stages are infective.

(3) That the healthy adult is highly resistant to leprosy.

(4) That when leprosy is acquired in childhood or early adolescence, it is most liable to spread and set up the active phase of the disease.

(5) That the cases of leprosy in whom the fingers and toes have fallen or have disappeared and the general appearance has become deformed, are the cases in the last stages of the disease or are usually those in whom the disease has died out, leaving permanent marks behind.

(6) That the climate of a place has got its effect on the infectivity of leprosy bacillus.

(7) That the best results of treatment are in cases that are in early stages.

On the basis of the above points, the line of prevention should be as follows:—

(1) Complete house to house and village to village survey of the infected area with reference to age and type of the cases, which will give the following:—

(a) The incidence of leprosy.

(b) Factors contributing to the high or low incidence.

(c) Early cases.

(d) Infectious cases.

(e) Cases in whom the disease has died out.

(2) So, isolate the open cases in houses, villages or institutions. Though the house or village isolation is not going to be so

effective as the institutional one but it is economical and has got its importance in village propaganda, so necessary in the present day conditions of the country.

(3) Separate the children born of lepers at birth.

(4) Reobserve in the community the cases in whom the disease has died out.

(5) Put all the early cases under observation and those who need treatment, should be treated regularly, with the object of preventing them from progressing to the infectious stages.

(6) Examine the contacts at an interval of every six months for five years.

(7) Lepers should be prohibited from engaging in occupations such as:—

(a) Preparation and sale of food and clothes.

(b) Domestic work and care or instruction of children.

(c) Nursing or wet nursing.

(d) Hair dressing.

(e) Prostitutes infected with leprosy are a potent means of the spread of the disease.

8. *Propaganda.*—An intensive and sustained propaganda should be carried on, emphasizing that:—

(a) Leprosy is a disease communicated by close contact, from a sick person to a healthy one.

(b) Only certain stages are infective.

(c) Every case does not need treatment.

(d) From amongst those who need treatment, it is the early ones that have got the best chances of recovery.

(e) Only dangerous cases need be isolated, others should be treated as out-door.

(f) In early non-infective stages, children need not leave their schools and workers, their employment, though they should be examined periodically.

(g) All contacts should be examined periodically.

(h) The development of public health sense is one of the greatest factors against the spread of leprosy.

(i) Healthy body is the greatest safeguard.

Signs and Symptoms and Differential Diagnosis of Leprosy

BY

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LEPROSY is a chronic infectious disease. Children are more prone to infection than adults and the disease is more often contracted before the age of thirty and rarely in infancy. The disease is not congenital and heredity seems to have but little share in its occurrence.

In about 80% of cases, leprosy starts with one or more depigmented or hypopigmented patches with or without anaesthesia. The most common sites first affected are the face, the gluteal region or the extensor aspects of the upper or lower extremities and the back. Superficial nerves are sometimes found thickened. Those most commonly affected are the ulnar, the greater auricular and the peroneal. These nerves are often painful and causes tingling and numbness when they are percussed lightly with a finger.

Sometimes, instead of anaesthesia, we find other disturbances of sensation as, paraesthesia due to over-lapping of nerves or hyperaesthesia due to irritation. Loss of sensation to heat or cold is sometimes noticed. This latter symptom is more pronounced in some other diseases of the nervous system *e.g.*, Syringomyelia, but in syringomyelia there is no loss of sensation to light touch. Sometimes we find disturbances of sensation in certain parts of the body due to local injury to nerves. I have seen a case of anaesthesia over the extensor aspect of the left thumb and index finger due to injury of the superficial radial nerve by wearing a tight wristlet for a long time. Such mistakes have to be avoided in diagnosis.

Likewise, depigmented or hypopigmented patches of a common innocent skin disease technically called *Tinea Versicolor* should not be mistaken for the early sign of leprosy. A depigmented patch with anaesthesia is a sure sign of leprosy, but anaesthesia is never found in *Tinea*. When any doubt arises, a scraping with a little saline added and examined under the low power of a microscope will show the spores of the parasites called "*microsporum furfur*" in cases of *Tinea Versicolor*.

In anaesthetic leprosy, an old term yet a self-explanatory

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one, now classified as A or N, we fail to find any bacilli (acid fast) by any known method of staining or culture. Such cases are considered non-infectious and termed "closed".

If leprosy in this stage is left untreated, it more often advances and the lowered resistance of the patient helps to worsen his state. More evident signs begin to appear. From the nerve stage, it goes on to the skin stage. This type of leprosy was formerly called tubercular, later as B stage and at the present day as the Cutaneous type (C). When leprosy has reached this stage, we can easily diagnose it by bacilliary findings on microscopical examination of a skin clip obtained from the affected part. Sometimes, we find the nasal mucous membrane affected without any other evident signs of leprosy. But, in such cases we get a history of occasional epistaxis and dry rhinitis with blocking of the nose.

In skin-leprosy, if any nodules or macules are present, a clip may be taken from any of them. On the other hand, if there is only a general thickening of the skin, the most suitable place to look for bacilli is the edge of the lobule of the ear. In skin leprosy there is usually analgesia, but no superficial anaesthesia. In taking a clip, a curved pair of scissors is to be used and the depth to which the clip has to be taken is about 2 m.m. and the raw surface of the cut clip is to be pressed and rubbed on a clean slide. The bacilli are located in the corium and hence the depth suggested is essential for successful result. The smear thus obtained is dried and fixed by heat and stained by Ziehl-Neelsen's method. In taking the nasal smear, one has to obtain the scraping from the anterior part of the nasal septum. The point of a sharp knife has to be used until a little blood appears. The material thus obtained is smeared on a slide so as to make a thin film, fixed and then stained and examined with 1/12" oil immersion lens.

Thus the two cardinal signs of leprosy are the depigmented anaesthetic patch and the presence of lepra bacilli in the skin or mucous membrane.

A few peculiar features of skin and nerve leprosy are worth noting here. In skin-leprosy, bacilli are not found in the epithelium and therefore a scraping of the skin surface for examination is useless. A clip from an anaesthetic patch is not useful as bacilli are not found in it. However, a patient may show anaesthetic as well as non-anaesthetic patches, the former giving negative and the latter positive results on examination.

In the early stage of nerve-leprosy, we sometimes find hyperaesthesia and later anaesthesia and if at this stage, the disease is

left untreated, it usually passes on to the skin stage and sensation returns. Perhaps, this peculiar phenomenon is due to the disappearance of leprotic infiltration of the nerves and nerve sheaths and the transmission of the causative factors to the skin surface. The absence of bacilli in nerve leprosy is probably due to our inability to detect them by any known method. They may be present in a non-acid-fast form.

There is still another form of leprosy which cannot be classified in any of the above category due to its histological and pathological characteristics, which are different from the ordinary lepromatous tissue, but akin to tubercles of tuberculosis. Instead of lepra cells with large numbers of bacilli, epithelioid cells are seen with necrotic changes similar to tubercles. It is undoubtedly not a real tubercular affection, but as there is no other appropriate term available, it is at present termed "tuberculoid" form of leprosy. Another characteristic of tuberculoid lesions is that many do not show bacilli on examination of the clip, or if they are found at all, they are only very few in number. The cause of the occurrence of such lesions is not probably due to the existence of non-acid-fast form of the bacilli, but due to the quality of the soil in which the bacilli are unable to multiply in large numbers on account of the resistance of the patient, unlike the cutaneous type of leprosy, where the lowered resistance of the patient favours the growth of the bacilli. It is important to distinguish between the tuberculoid and the cutaneous types of leprosy; for the prognosis in the former is more favourable than in the latter type.

A few of the chief subsidiary signs of leprosy are also worth noting:

(1) Dry rhinitis is sometimes the only first sign noticed with ulceration of the mucous membrane of the nose. The patient complains of blocking of the nose. He sometimes gives history of occasional epistaxis. In such cases we are able to detect bacilli in the scrapings.

(2) Hyperaesthesia is occasionally found which gives place later to anaesthesia.

(3) Depigmented spots or patches in an individual is a suspicious sign and more so when he gives a history of contact. Such spots or patches are to be carefully distinguished from tinea versicolor already described. In Dermal Leishmanoid also, we see a similar condition and the differential diagnosis will be given later.

(4) Analgesia is often found in cutaneous leprosy due to the affection of the deeper layer of the corium. This sign can be elicited by a pinprick selecting suitable areas of skin affected and not affected and comparing the relative effects of the pricks.

(5) Erythematous patches are sometimes the earliest sign of leprosy. This, in my opinion, is doubtful, as I find that such patches have been depigmented in the first stage of their appearance which later assume the red colour. This is further corroborated by the fact that such erythematous patches during proper treatment become depigmented and hypopigmented before they finally disappear.

(6) Thickening of superficial nerves to a considerable degree is in favour of leprosy. Those most commonly affected are the ulnar, the peroneal, the great auricular, the supra-orbital and the radial. In some rare cases, we come across nerve abscesses.

(7) Anhidrosis is also one of the signs of leprosy. In some patients we can see such areas of skin devoid of sweat when they are made to take vigorous exercise while the surrounding skin sweats profusely in contrast.

(8) Glossiness of the skin and tiny black spots where hairs commonly grow *e.g.*, the lateral aspect of the thigh are sometimes noticed. These black spots are hairs lying curled up within the openings of the hair follicles and can actually be pulled out.

(9) Swelling of the interfollicular ridges of skin due to affection of the corium is another important sign in leprosy. The pores of the hair follicles stand well depressed and give a peculiar mosaic appearance. A clip taken invariably shows bacilli. This interfollicular swelling with redness simulates Lichen planus but the latter is distinguished from leprosy by its lilac colour and absence of bacilli.

(10) Blisters of the fingers and toes etc. and perforating ulcers of trophic origin are often noticed in secondary nerve-leprosy. They contain no bacilli unlike the ulcerating nodules of leprosy which discharge innumerable bacilli.

(11) In endemic areas irregular fever with vague nerve pains are suspicious signs. Such conditions are often mistaken for rheumatism. Superficial nerves have to be examined for thickening.

(12) Burning sensation of the extremities has been observed in some cases as the first symptom of leprosy and followed later by depigmented anaesthetic patches.

So varied are the signs and symptoms of leprosy. The two cardinal signs are depigmented anaesthetic patch and presence of mycobacterium leprae. If one finds any of these in a patient under observation, he can safely give the diagnosis as leprosy. To classify one as a patient of leprosy without any of these two signs, is a grave injustice. On suspicion, the patient may be safely treated for leprosy but the diagnosis should on no account be given until and unless the cardinal signs of leprosy appear.

Differential diagnosis.—

I. *Tubercular affection of the skin*.—Sometimes simulates leprosy but the absence of the two cardinal signs helps to differentiate it from leprosy.

II. *Psoriasis*.—Anaesthesia and bacilli are absent. Section of the skin in psoriasis shows exaggeration of papilli whereas in leprosy they are flattened out.

III. *Raynaud's disease*.—Resembles secondary nerve leprosy. The acroteric lesions are misleading, but in this disease the anaesthesia is confined to the gangrenous areas.

IV. *Dermal Leishmaniasis*.—The cardinal signs of leprosy are absent although there are depigmented patches found in this disease. We get a history of Kala-azar and by cultural method from the materials obtained, L. D. bodies are found.

V. *Leucoderma*.—Here depigmentation is more complete. There is no anaesthesia, no acid-fast bacilli and no thickening of the nerves.

VI. *Yaws*.—Cardinal signs of leprosy are absent and bacteriological examination shows Treponema Pertenu.

VII. *Syringomyelia*.—Wasting of the small muscles of the hand and loss of sensation to heat and cold simulate leprosy, but there is generally no loss of sensation to light touch.

VIII. *Syphilis*.—Corium of the skin is affected in both. Erythematous and depigmented patches are also present in both but the cardinal signs of leprosy serve to distinguish it from syphilis. But, often both the diseases co-exist in one and the same individual when Kahn test will settle the diagnosis.

IX. *Lead palsy*.—Paralysis is more marked than loss of sensation in lead palsy. History and the "blue line" on the gums will also help to clinch the diagnosis.

X. *Birth marks*.—These should not be mistaken for the depigmented patches of leprosy. History and absence of anaesthesia will serve to distinguish them.

XI. *Trauma* of cutaneous nerves sometimes shows loss of sensation to the parts of the skin supplied. Scars should be looked for. History of trauma also helps to avoid any error in diagnosis.

XII. Conditions causing thickening of the skin such as *Sclero dermia* and *keratosis* cause impairment of tactile sensation. In such condition, one has to look for other signs of leprosy as thickening of nerves, depigmentation, erythema, anhidrosis and presence of bacilli etc.

XIII. Various kinds of *tinca* such as T. Saigurata, T. Circurata, T. Cycosis etc. The presence of fungi and the absence of L. Bacilli will settle the diagnosis.

Treatment of Leprosy

BY

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THE successful treatment of leprosy depends largely on the combination of both general and special measures. These measures should be adopted according to the type and nature of the individual case and not applied indiscriminately.

General Measures.—It is proved by physicians experienced in leprosy that careful and persistent efforts to find out and treat intercurrent diseases which tend to reduce the general resistance of the patient is essential to successful therapy.

Adjustment of both the quality and quantity of the diet helps the patient to have a better control over the disease. Other general measures such as personal hygiene, graduated physical exercise, the stimulation of moral and mental happiness are also essential for the successful treatment of leprosy. Constipation if present, should be remedied by diet regulation and exercise but if this fails drugs must be used. Such skin diseases as tenia, scabies, staphylococcal and streptococcal dermatitis should be cleared up.

Last but not least is perseverance in treatment. This is very

essential as many cases of leprosy, especially advanced cutaneous cases, do not persist in the treatment as required. Cutaneous cases require to be under treatment for many years and this long continuous treatment is considered difficult and impossible by many.

Special Measures.—Hydnocarpus oil and its derivatives are the drugs that are most commonly used for the treatment of leprosy. Of all the numerous drugs that have been tried, these have given the most satisfactory results. The sole disadvantage is that oil is difficult to inject and slow to absorb when injected subcutaneously or intramuscularly. This can be overcome by heating the oil to body temperature, before injection.

The derivatives of the oil are the Ethyl Esters of the Fatty acids and the soaps of these acids. The least painful of the drugs to be administered is the pure oil. Esters are slightly more painful and Alepol is the most painful. It is found by experience that the pure oil is decidedly less reactive than the Ethyl Esters or Alepol.

Advanced Cutaneous (Nodular) cases stand the oil injections better. But such cases when given injections of Esters are likely to develop leprotic reactions.

The following preparations are found effective in the treatment of leprosy.

(1) Pure Oil.....with 2% doubly distilled Creosote.

(2) Ethyl Esters of Hydnocarpus Oil in different dilutions, such as (a) Ethyl Esters with 50% Olive oil and 2% doubly distilled Creosote; (b) Ethyl Esters with 25% Olive oil and 2% doubly distilled Creosote; (c) Pure Esters with 2% doubly distilled Creosote.

(3) Alepol is a soap prepared from certain fractions of hydnocarpus oil.

The method of injection may be subcutaneous, intradermal, intramuscular or intravenous. Oils and Esters should not be given intravenously. We obtain best results by intradermal injection of Esters into skin-lesions especially in cases with a limited number of lesions. With a special intradermal needle, the esters are injected into the corium and not into the subcutaneous tissue. Sufficient is injected to raise a wheal $1\frac{1}{3}$ " in diameter. All kinds of lesions can be injected; nodules, macules, infiltrations in ears and face. The Esters best suited for this purpose consist of 75% of Esters of Hydnocarpus Oil and 25% of Olive Oil (fatty, acid-free) and 2% doubly distilled Creosote. (Dose from 1 to 5 c.c. in

adults and 1 to 3 c.c. in children). Pure Hydnocarpus Oil with 2% doubly distilled Creosote can be used for subcutaneous purposes. Injections may be given once or twice a week. As a rule, small doses should be given twice a week. Later when the patient gets more used to it, larger doses to the extent of the patient's tolerance may be given and these only once a week. (Dose 1 to 10 c.cs., 10 c.cs. repeated 10 times followed by rest for a fortnight). In children, the dosage should be adjusted according to age.

Suitable sites for subcutaneous injections :—

- (1) Outer aspect of both arms.
- (2) Front and outer aspect of both thighs.
- (3) Both buttocks.

As a general rule more than 3 c.c. should not be given in any one area. If the dose is above 3 c.c., it should be divided and given in two different places. This facilitates rapid absorption of the oil or esters when injected and prevents inflammation or swelling at the site of injection.

Alepol.—This can be given intravenously or intramuscularly. From our experience, we can say that this drug has no exceptional advantage over the pure oil or the esters except for the short rest made possible from the most painful intramuscular, or subcutaneous, or intradermal infiltrations. A 3% solution in distilled water with $\frac{1}{2}$ % carbolic acid is generally used for intramuscular injections and a 2% solution with $\frac{1}{2}$ % carbolic acid intravenously. Dose in both methods is 1 to 10 c.cs. and 10 c.cs. to be repeated 10 times followed by rest for a fortnight.

Painting of lesions with 1 in 3 Trichloroacetic Acid in distilled water helps in some cases to clear them up. This should be done once a fortnight.

Leprotic reactions.—Reactions in leprosy are very common. They are of two kinds, nodular (cutaneous) and neural (nerve):

(1) *Nodular reactions*—Are accompanied by a rise of temperature, appearance of fresh macules or nodules with general aggravation of the disease.

(2) *Nerve Reaction.*—This is characterised by nerve pain, in one or more nerves. The pain will be very severe in some cases and less in others. The nerves that are most commonly involved are one or both ulnars, one or both peroneals, or one or both great-auriculars. Sometimes diffuse pains occur in the deep as well as the cutaneous nerves.

The above condition presents a great difficulty to those who

are not experienced in leprosy work. I have often come across cases of leprotic reactions in whom the anti-leprotic treatment was given more vigorously during the reaction phase with the result that their condition was made worse. Such a condition can be brought under control by the following method of treatment.

Nodular reactions. Stop all anti-leprotic treatment.

A course of 6 injections of potassium antimony tartrate .04 gm. at each injection should be given every other day, intravenously with the following mixture orally :—

R Soda Bicarb	...	3 ss.
Phenazone	...	grs. iii.
Soda Salicyl	...	grs. vi.
Mag Sulph	...	3 ii.
Spts Ammon Aromat	...	m x.
Aqua distillate ad.	...	3 i.

Oz. i. three times a day.

Sometimes, a 2% solution of Mercurochrome given intravenously once a week brings down a reaction. But this is found to be useful only in leprotic reactions due to any septic foci. (dose 1 to 5 c.cs.).

Nerve reactions.—There is no satisfactory treatment for this condition. Generally, the pain is relieved by subcutaneous injections of $\frac{1}{2}$ c.c. adrenalin chloride 1:1000 in about 1 c.c. of saline along the course of the nerve, and the following can be taken orally three times a day.

R Aspirin	...	grs. 5.
Caff. citras	...	grs. 2.
Phenacetin	...	grs. 3.

One dose three times a day.

In some cases, a 2% solution of Fluorescine 1 to 5 c.c. intravenously twice a week brings about a great relief from nerve pains followed by drachm dose of Soda Bicarb by mouth, three times a day.

In conclusion, I would say that successful treatment of leprosy depends largely on the type and stage of the disease under treatment. The greatest prospect of favourable recovery lies in early treatment.

My thanks are due to Dr. R. G. Cochrane, M.D., M.R.C.P., D.T.M. & H., Chief Medical Officer, Lady Willingdon Leper Settlement, Chingleput, for his kind permission to publish this article.

Complications of Leprosy and their Treatment

BY

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COMPLICATIONS in leprosy are very common and their treatment requires skill and care. This treatment is, unfortunately, often rendered more difficult, because of the negligence of the patient and his failure to realise the importance of early medical aid.

Among the complications, which the general practitioner is likely to see, are the following :—

(A) **LEPRA REACTION.**—Lepra reaction may arise from any debilitating condition which in anyway lowers the resistance of the patient. Such factors as unhealthy conditions of life, coincident diseases, excessive intoxications, defective foods etc. may precipitate the reaction.

The reaction may occur in any stage of the disease ; but is more common in the cutaneous type. Reaction varies in severity ; In some cases, there is slight fever, neuritis and possibly one or two reacting nodules. In other cases, however, the reaction may be severe and produce great prostration, but it practically never causes death.

The condition of reaction is usually temporary, subsiding within a few days or weeks, but some of the reacting lesions, particularly neural lesions are permanent. Reaction and its significance need recognition as they greatly affect treatment and prognosis, and failure to recognise them may give rise to very misleading ideas regarding the course of the disease and the effects of treatment. Short reactions do good but prolonged reactions are never beneficial.

Lepra reactin is a toxæmia associated with the dissemination of mycobacterium lepræ throughout the body. This may be caused by the multiplication or by the liberation of bacilli from the tissue where they have been previously shut up or both processes may occur.

It is difficult to explain the toxæmia, as lepra bacilli produce no toxins. It is probable that the reaction is an *allergic phenomenon* and this may explain the toxins.

The commonest signs and symptoms of reaction are:

Rise of sedimentation index is common in every type of reaction and there may be fever in certain cases upto a temperature of 104 to 105° apart from the following:—

Neural.—Pain, tenderness, swelling (occasionally abscess-formation), rapid extension of anaesthesia and development of trophic lesions.

Joint pains.—Joint pains are among the commonest symptoms of reaction here. In such conditions gonorrhoeal arthritis must be considered; for, no improvement can be effected until the gonorrhoea is treated.

Bone pain.—Is one of the symptoms of reaction but is not so common here, as joint pains. In these cases, the patient complains of severe pain and tenderness and in our experience we noticed rarely periostitis of the bone.

Cutaneous lesions.—Erythematous painful nodules varying in size, may appear all over the body. Thickening of existing lesions and erythema of pre-existing lesions may also occur. Oedema of feet, hands, face and lips etc. are also common cutaneous reactionary signs apart from rise of temperature upto 104 to 105° and rise of sedimentation index.

Mucous membrane lesions.—There is often increase of lesions in the nasal mucous membrane causing profuse purulent discharge, epistaxis, blocking of nares: swelling of the larynx causing severe respiratory obstruction, ulceration of the pharynx, and oedema of the glottis are also common.

Other lesions.—Adenitis, Orchitis, Oophoritis and sometimes the internal organs e.g. lung may also be affected.

In the reaction of the lung, it closely simulates tuberculosis. We have seen several cases showing only acid-fast bacilli in the sputum under microscope, which were difficult to differentiate from tubercle bacilli.

Sometimes, the eye also is affected and shows signs and symptoms of conjunctivitis, Iritis, Keratitis, iridocyclitis and corneal ulcer etc.

Treatment.—*Lepra fever.*—When a patient is having high fever with or without reacting nodules etc. the first thing is to keep him to bed and cover him up with blankets and allow him to

sweat profusely and a dose of an antipyretic as given below should be given on the first day.

R Aspirin	...	gr. 5.
Phenacitin	...	gr. 5.
Pul. Dovers	...	gr. 5.
Calomel	...	gr. 3.
Caffeine citrate	...	gr. 2.
ft. pulv. one. t. d. s.		

The next morning a dose of saline purgative like Magnesium sulphate 1 oz. in 2 oz. aqua or Mist Alba 3 oz. stratum given. Alkalies like soda bicarbonate gr. 30 in 1 oz. of aqua or Mist Soda salicylas 1 oz. can be given. Some metallic drug preparations, e.g. Solygnol B, Mercurochrome and Potassium Antimony Tartrate are supposed to have some effect on re-action. In this Hospital we have tried Solygnol B intramuscularly in doses of 0.01, 0.25, 0.05, and 0.1 grams in 5 c. c. Aqua distillata. and also Mercurochrome 1% solution 2 c.c. to 10 c.c. was given once a week in prolonged lepra re-action, but except for breaking down of reactionary nodules, suppuration and ulceration of the same, no beneficial effects followed.

Generally, we give Potassium Antimony Tartrate intravenously 0.02 to 0.04 grams, dissolved in 2 c.c. of normal saline or Aqua distillata every alternate day and we find the temperature coming down in most cases and also the reactionary nodules subsiding, but in some cases where there is no temperature but nodulation, the P. A. T. is not satisfactory, for, such cases have recurrence of temperature. Such cases will benefit by alkalies e.g. soda salicylas and soda bicarbonate.

Aniline dyes e.g. fluoresceine 2% solution in 2% solution of soda bicarb equal quantities can be given intravenously 2 c.c. to 12 c.c. every alternate day, but in our experience it did not give beneficial results. Here we have tried some prolonged skin re-action cases with Diathermy treatment and found the results gratifying.

Nerve reaction, joint pains and bone pain:—For pain and tenderness with or without thickening of nerves, drugs like Ephedrine $\frac{1}{2}$ grain in one dram of normal saline or aqua repeated in 20 minutes given either orally or subcutaneously, may give only temporary relief in some cases; so also Liquor adrenalin hydrochloride 1 in 1000 solution $\frac{1}{2}$ c.c. subcutaneously. Locally, application of antiphlogistine, belladonna and ichthyol may also give temporary relief.

When the pain is persistent, the following drugs are of much use:

℞ Aspirin	...	gr. 5.
Phenacetin	...	gr. 5.
Caffeine citrate	...	gr. 2.
ft pulv. one. t. d. s.		

or

℞ Sodii Salicylas	...	gr. 10.
Soda Bicarb	...	gr. 10.
Tinct. Gelsemium	...	℥ 5.
Aqua	...	℥ 1.

1 oz, t. d. s. or 4th hourly.

For sleeplessness due to nerve pain or joint pain etc. give the following:—

℞ Sodii Bromide	...	gr. 15.
Chloral Hydras	...	gr. 15.
Tinct. Opii	...	℥ 15.
Aqua	...	℥ 1.

Sig at bed time.

In a thickened and painful nerve, operation should be performed; the nerve being exposed and stripped from the nerve but in our experience many such cases developed contractions of fingers and toes at a later period. It is therefore indicated only when every form of medical treatment has failed. In our hands, Diathermy treatment was of little or no benefit in acute and sub-acute nerve reactions but it may benefit chronic nerve reaction.

We had better results by injecting 2 to 4 c.c. of either hydnocarpus oil with 4% creosote or its esters with 4% creosote on alternate days along the course of the nerve. 2 to 4 such injections will very often relieve the pain and tenderness and in some cases thickening of the nerve was also reduced.

In abscess of the nerve, the abscess should be opened, pus drained, necrosed tissue removed and where necessary, the nerve decapsulated.

In joint pains, one should elicit the history in order to find whether the pains are gonorrhoeal or rheumatoid and treat on appropriate lines. We have treated apparent lepra reaction joint pains without effect and on later investigation it was found to be gonorrhoeal infection, treatment for which resulted in the disappearance of symptoms.

In bone pain, as in nerve pain and joint pain, local applications like antiphlogistine, belladonna and ichthyol may be used. Orally, alkaline drugs and antipyretics can be administered.

(B) COMPLICATIONS OF THE NOSE.—The following nasal complications of the lesions are frequently seen:—

(1) Pain and Erythema.

(2) Shattered ulcers of septum Ethmoid, base of Sphenoid and Vomer bones.

(3) Encrustation.

(4) Excision, ulceration and destruction of bases.

Early signs are:—Bleeding, crust formation, smell and finding of bacilli.

In addition to the above, most patients complain of dry rhinitis or scab formation and for such cases we use the following nose drops each evening:—

℞ Camphor	...	℥ 1.
Creosote	...	℥ 1.
Hyd. oil	...	℥ 1.
Sweat oil (Gingely oil)	...	℥ 9.

Method of administration:—The mixture is taken in 10c.c. or 5 c.c. glass syringe without needle; patient is seated with face upwards and has injected about $\frac{1}{2}$ c.c. of the above drops in each nostril and has the mixture retained in the nostrils for a few minutes.

One can treat such cases by first irrigating with 1 in 80 solution of Borax and afterwards using any one of the following nose drops:—

℞ Tinct. Iodine	...	1 part
Glycerine	...	2 parts

or

℞ Camphor	...	1 dram
Creosote	...	1 dram
Hyd. oil	...	1 oz
Olive oil	...	2 oz

In superficial lesions and crusting granulations, spray the nose with 5% chromic acid, or 1 in 3000 brilliant green or 50% lactic acid twice a week. For swelling and oedema of the mucous membrane irritate the nose by irrigation with concentrated solutions of the following:—

℞ Sodium chloride	...	200 parts
Borax	...	200 parts
Mag sulph	...	200 parts.

or 1 in 8 solution of Borax every day or every alternate day.

Certain Important Factors in the Treatment of Leprosy

BY

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LEPROSY is a disease, whose treatment is a very difficult one. So far, no specific is known. Moreover, there is no hard and fast rule, as to how to treat a case of Leprosy, as the disease presents itself in various forms, quite different from one another. Every individual patient of Leprosy, therefore, is to be treated on his individual merits. The doctor, who deals with these cases, has to go on watching the progress of the case and has to regulate the doses according to his experience. It is, therefore, quite essential that the doctors-in-charge of the Leprosy Centres should not be transferred or changed. In the treatment, a strict co-operation between the doctor and the patient is required; otherwise no good results are possible. Most of the patients think that the injection of the Ol. Hydnocarpus in one form or other is the only thing required for their treatment. But, though this treatment is of immense value, there are certain other important factors about which the patient has to see for himself, especially those who are out-door patients. Without observing these, the patients do not derive as much benefit as they should. In another article I propose to deal with my personal observations regarding the effects of the oil Hydnocarpus and its preparations in the treatment of Leprosy. In this article, I am dealing only with those important factors, which, in my opinion, are as essential as the treatment with the Hydnocarpus oil. If these factors are ignored, much benefit cannot be expected and hence the doctors-in-charge of the Leprosy clinics should impress these points on the minds of the patients, on the very day they first appear for treatment, as otherwise the treatment will do no good and the patients will be disappointed after a long and painful treatment (being the injection treatment) and thus the clinic will lose its reputation and the anti-leprosy campaign will be undone. Now-a-days every one wants things ready made. No one likes to exert himself even for his own necessities and the patients want that everything should be done by the doctor for them and they should not exert in the least. On the other hand, the doctor's job is a very difficult one. He has to educate his patients

by often giving lectures and demonstrations to them and as, unluckily, the patients are generally of the poorer and uneducated classes, the doctor has to do his work or fulfil his mission with great patience and tact. Undoubtedly, the difficulties of the doctor are immense and he has to fight against great many odds besides his being always in danger and hence only those doctors should come forward for this work, who have love for humanity and are eager to serve this most hated, neglected and most suffering section of the humanity. Their goal in life should be to sacrifice their lives and worldly pleasures in the service of others. To serve others should be the best pleasure of their life and they should think this as their duty, which they have to do by the order of the Heavenly Father above and so their best *pooja* or prayer should be—Oh! our Heavenly Father, give us strength enough so that we may sacrifice our life in the service of others! They should realize that the lepers and we are the sons of the same Heavenly Father and so it is most cruel on our part if we hate our own brethren. Besides this love, the doctor should be a hard-working and a strong-willed gentleman, as being a poor man's work he is sure to get discouragements at every step. Hence the above sort of doctors or workers, who are ready to sacrifice everything for the sake of these fellow-brethren, whom there is none to love in this world excepting the doctor, should come forward for this work. Undoubtedly he will have the blessings of his patients, who will ever regard and love him as their God on earth. What I mean to say is that there should be a real love and co-operation between the doctor and the patients. Both should help each other and both should move together like the various parts of a machinery, as without this, success cannot be attained. Besides this co-operation, the other factors are:—

(1) *Cleanliness*.—Leprosy is a disease of dirt and if the patients do not keep their body and clothes clean, the treatment will not do them much good. The doctor should, therefore see that the patients keep themselves quite clean and tidy in all their habits.

(2) *Exercise*.—Leprosy patients should always lead an active life. They should be trained to do regular and systematic exercises daily in the open air. Gardening is also a very good and healthy exercise. Every part of the body must be moved—even the small muscles of the hand and feet, which usually atrophy and cause the curvatures of the fingers and toes. I have seen that regular exercise of the fingers and toes improve them greatly,

where they have begun to curve. Moreover active patients doing hard work daily, as daily labourers, farmers, soldiers, etc. can stand much higher doses of the medicines with no reaction than those who lead a sedentary life and hence improve in much shorter periods than the latter. Usually anyone suspecting this disease, begins to hide himself fearing the hatred of his fellow brothers, also of the police where compulsory segregation is enforced, and thus in due course he becomes an advanced case and then besides becoming a danger to the community, he makes his life miserable. Hence activity is essential for patients suffering from this disease. These exercises are taught to the patients in my outdoor department.

(3) *Diet*.—The diet of the patient must be simple and nutritious. By experience we have seen that the Punjab diet on the whole is the best diet. The Punjab diet consists of *chappatis* or *rotis* of wheat flour, dals of various sorts, fresh vegetables, fruits, milk, *dahi*, *lasshi*, ghee and butter. The disease is chiefly found in places where rice is the chief diet of the people; where it is so, let the patients take one meal of rice and the other of *chappatis*; better the rice is avoided altogether. The treatment even with the best and most reliable medicines cannot do the least good if the patients do not take proper diet. What can medicines do to one with a hungry stomach? So till there is poverty and the people cannot feed themselves well or at least be able to fill their bellies twice a day, in spite of our best medical aid, Leprosy can never be rooted out of the world. Leprosy has been practically stamped out of Europe, not because of Medical aid as these are only the recent researches, but because of the improvements in health, diet, general hygienic and sanitary measures; this again was the outcome of industrialization and hence the relative disappearance of poverty from Europe. In those countries, where there is poverty, I am sure, from my long experience in this disease, that Leprosy can never be stamped out in spite of the best efforts of the respective governments till the root cause of the Leprosy is removed or till the extreme poverty is removed and the general condition of the people is improved.

Wines should be totally avoided; smoking, if not totally given up, may be allowed in moderation only.

(4) The patients should be taught to lead the life in hygienic and sanitary conditions. Dr. Muir has rightly pointed out that an anti-Leprosy campaign includes all the health problems. Till the above things are strictly observed or in general, the patient's

power of resistance is kept high, the patients cannot derive much benefit by *Hydnocarpus* injection alone.

In my out-door Department, the total number of patients on the roll are more than 1950. The average bi-weekly attendance is about 700 but by patience and tact, I have been able to change the entire out-look of the patients. Undoubtedly to achieve this, I have to work very hard, as the poor uneducated people and especially the hill tribes residing in the Himalayas are exceptionally dirty in their habits. It seems as though cleanliness is absolutely unknown to them. Even the middle classes are very dirty, though their outside look may be clean but their inside, the skin and the clothes next to their skin are generally extremely dirty. Sometimes they connect their dirty habits with one or other religious superstition as the religious preachers who are themselves dirty cannot expect to teach the Gospel of cleanliness to others. It is due to this and rice diet etc. that Leprosy is found in the hills, even in the hills of the Punjab, while in the plains it is practically unknown so far. So, a good deal of tact and patience is required to deal with such sort of people. I personally inspect on each turn the bodies, clothes, and teeth of the out-door patients and it is after a hard labour of several years that I have been able to mend their habits a good deal. In this way, Leprosy workers deal not only with the Leprosy problem but also spread the holy Gospel of Cleanliness through their patients.

Some Malaria Problems

BY

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THE object of this article is merely to discuss, to some extent, some of the problems and the main difficulties that baffle medical thinkers and those that are called to put into practical use what science offers towards bringing relief to malarial sufferers. The intense and alarmingly widespread suffering of the island of Ceylon, from endemic and epidemic malaria in recent years, has made the study of the problems connected with this disease more urgent than ever before. We shall, in this article, attempt to focus attention on such points of the general malarial question along which further advance of our present knowledge is indicated.

The Ceylon Epidemic of 1934.—As the reader is no doubt aware that towards the end of 1934, there broke out in Ceylon an epidemic of malaria of such magnitude that could hardly be rivalled by any previous outbreak in any country. Nearly a hundred thousand people were destroyed by it within the space of seven or eight months. It was previously quite an unheard of and even an unthinkable phenomenon that a disease like malaria could kill such a large number in that way. It baffles one when one reflects on what perhaps has really been at the bottom of this colossal pestilence. It laid waste nearly one-quarter of the extent, and a third of the population of Ceylon. For fuller details of this epidemic, *vide Journal of South Indian Medicine*, vol. III, No. 4 and 5. Although a careful and detailed study of the epidemic was made and it has brought out some new facts, there still remains a good deal that is difficult of understanding as to how an epidemic of such magnitude does occur and how it could be prevented in the future.

Malaria—The largest Public Health Problem of India and Ceylon.—Malaria is the largest public health problem of India and Ceylon. This has been demonstrably supported by the vital statistics of these countries. Those who have travelled in these countries have seen the damage that has been wrought among the people by malaria. Those who have travelled by ships that call at

Indian ports carrying emigrant Indians have seen the pathetic sight of returning emigrants from rubber, tea, and cocoanut plantations of Ceylon, Malaya and other places: only a few with some jewels and personal apparel on their bodies, and chattels along with them, can be found while, the majority look mere wrecks, physically and morally. Malaria and hook-worm are the predominant factors in that destructive process.

In most countries people wish for, and some, work actively towards the check and, if possible, a total eradication of malaria. 'Panama' is often mentioned as an outstanding example of success in this matter. It is well to remember that Panama was a comparatively small area, a land that had to be tackled, and the resources expended on it were enormous, and the strictness, with which the sanitary regulations were enforced, almost amounted to martial law conditions. All countries, especially the poor ones of the East cannot achieve what Panama was able to, but still they realised that something more could be done than what has already been done till now, in the several malarial parts.

Malaria in ancient and recent times.—Many ancient lands were apparently afflicted by malaria. The ruins at Pollannaruwa and Anuradhapura in Ceylon, mark the devastation of vast tracts of the country that were once prosperous and populous, and apparently malarial fevers reduced these extensive areas of Ceylon into depopulated jungle tracts. It is estimated that today nearly two-thirds of Ceylon lies undeveloped, but yet the Sinhalese people are opposed to the immigration of the people from India into Ceylon.

Not only in the tropics, but in more temperate lands, fever of an intermittent nature was known among the ancients and even associated with certain seasons, rainfalls, stagnant waters and marshes; the use of mosquito nets to sleep under, which warded off fever from the marshes, was also known. Obviously, the ancients connected fevers with mosquitoes. The Roman fever had been a bane of ancient days, and one sees even to this day remnants of the ruin of those magnificent viaducts on the Roman Campagna for miles and miles, which probably were part of the efforts of the engineering skill of ancient Rome that was directed against the spread of fever from the Pontine marshes adjoining the city. As the planter of tea and rubber battles to-day against malaria in the process of opening out jungle-land so also did the ancient Greek colonists and the Roman Eagles face the enemy of Ague as perhaps their most formidable obstacle to colonization and expansion.

Malaria decreasing in Europe.—Even in England, France and Holland, malaria is nothing like what it was a century or two ago. It is attributed to draining of marsh lands, better housing and sanitary improvements generally. In other words, Europe has been steadily increasing in prosperity. Although after the Great War, a great number of soldiers returning from the Balkan, Egyptian, Mesopotamian, Indian and African campaigns, came home to Europe with malarial germs in their system, and although *Anopheles maculipennis*, the great malaria carrier of Europe, has not left Europe (in spite of the War), still astonishingly little spread of the disease was noticed in any of these countries in spite of thousands of infected soldiers returning from abroad. The climate is against it, since a sustained warm temperature of 58 to 60 degrees Fahrenheit is needed for the development of the B. T. Malaria parasites in the mosquito to make it infective. Here then is a very prominent fact to note, especially by such as still think that an anti-malaria campaign means almost entirely an anti-mosquito war. You cannot kill the mosquitoes of the world, no more than you can think of catching all the fishes of the sea; for we hear it said often that there are as many fishes in the sea as ever came out of it. Terrible epidemics of malaria were known in Holland a century ago. But today malaria is insignificant. The change is due to general sanitary and social improvements mainly. Better houses have lessened the chances of infection. Medical help was improved, and quinine was sold cheaply and used extensively. All these factors are deplorably absent in a scheme of malaria control that we commonly see in the East, while anti-mosquito measures at enormous cost figure prominently. Quinine remains expensive, not so widely used as it should be, not so easily available in the tropics, although cinchona can grow only there! But we shall return to the subject of quinine later.

In this connection, it is interesting to read these words written by the League of Nations' Malaria Commissioners:—

"In some countries of Eastern Europe with limited financial means, nothing has retarded the effective control of malaria so much as has the belief that because mosquitoes carry malaria their elimination should be the object of chief concern and expenditure. We have seen some extreme instances in which so much of the time of the available medical men was taken up by anti-larval measures in the field that little or no treatment of the people could be carried out."

The Commissioners also state that in many parts of Europe

they are to-day ridding themselves of malaria by means other than those of antilarval measures and without the actual occurrence of reduction of the anophelines.

The Cycle of Golgi.—In accord with the biological law of 'alternation of generation' commonly met with in the life history of certain Protozoan animals, the malarial parasite similarly has been shown by Golgi and by Ross, in the course of their brilliant researches, to exhibit the two phases of the asexual and the sexual multiplication—the Cycles that have been worthily named after them. The completion of the life cycle of the parasite cannot, therefore, be accomplished unless man and the anopheline mosquito come together. It is now commonly known that the oval and crescent-shaped bodies, known as the gametocytes, which remained for long periods in the peripheral blood of one who has had malaria, are the units that start the generation of the parasite in the mosquito phase of the cycle. The gametocytes are then the only forms that can carry on the completion of the cycle in the mosquito. Now, these gametocytes do not appear in the blood of the patient with the fever bout. They take some time to make their appearance in the blood in peripheral circulation. Seven to ten days after the fever has commenced, these gametocytes begin to appear. They take a further seven to ten days to mature and become fully developed and so able to start their career in the stomach of the mosquito. Also it has been noticed that these gametocytes do not develop at all in patients treated with quinine from the first fever bout. So then, it becomes apparent that just as much as the mosquito infects man, man has to infect the mosquito. The fever is never passed on from man to man, (unless the blood containing the malarial spores are injected artificially into the blood of another as has happened accidentally in drug addicts, or else deliberately in experimental work, or in inducing malaria as a therapeutic measure).

Before and after.—As we have seen before, from very early times in the dawn of human intelligence, through all the struggles of man to colonize on newly cleared lands of earth, right up to our times, fevers, jungles, marshes and mosquitoes were all associated intimately in man's mind. But not till the twenty years between 1880 to 1900, that the secret was more completely uncovered through the brilliant *Laveran-Golgi-Ross* researches into the life cycle of the malarial parasite. Until then the prevention of malaria in endemic areas was possible after long and weary years of struggle. But even so, wholesale drainage of the land and continuous culture of the soil have eliminated malaria in many

parts of the world, including many districts of India and Ceylon. The malarial discoveries since then have placed in our hands more effective and definite weapons, and the problem now becomes for us as to how rapidly and expeditiously could we get rid of malaria from our midst by taking advantage of these discoveries.

Can we place a break in the cycle?—A break placed at any important point in the life cycle of the malaria parasite, if effectively carried out, must necessarily prevent its reproduction and multiplication. Three separate methods have been experimentally tested. They are:—

- (1) To exterminate the *Anopheles* mosquitoes.
- (2) To prevent mosquitoes from biting malarial patients.
- (3) To sterilize the blood of man carrying the malarial parasites so effectually as to prevent mosquitoes from being infected.

Extermination of the Mosquito.—As mentioned before, this method has received the greatest amount of attention. If it could be undertaken effectively and at reasonable cost, such a method can be relied on always. The cost of prevention of breeding of the carrier mosquitoes near towns and human dwellings has been proved prohibitive in several undertakings. Mianmir is a well-known instance. At the beginning of this century, the Government of India started a campaign against malaria through mosquito reduction in a small area of Mianmir near Lahore. It is a military station surrounded by irrigated wheat fields. These foci of stagnant water caused mosquito production. After spending several lakhs of rupees every year, and after persisting in this campaign for nearly ten years, they abandoned it as they found the measure adopted by them ineffective and useless in reducing malaria.

Mosquito Nets and Proofing.—Again, the prevention of mosquitoes from biting human beings consists of providing mosquito proof houses and mosquito curtains for beds. Such may be attempted in the case of the State hospitals, but even among the well-to-do classes in certain places in the tropics, as in Ceylon for instance, the use of mosquito curtains at nights is not a practicable suggestion. Many persons, even some doctors, (I was shocked to notice), are in the habit of having the bed room windows tightly closed at night. I have heard from the lips of a doctor that he closed the windows in order to keep out mosquitoes. If such is the case with the doctor, what of the laymen? This method of prevention of mosquito biting cannot therefore be trusted for extensive use in the

Tropics. Besides, nets are expensive. Other methods would need to be thought of and studied for easier application for poor Tropical conditions, such perhaps, as the popularisation of crude juices from strong smelling oily leaves of domestically cultivable plants as a means of warding off mosquitoes from the skin on which such juices could be comfortably smeared. Such methods are employed against ticks, some fumes of burnt leaves and flowers are used against snakes and mosquitoes etc. Of course, the best teacher of a race in such matters is very often to be found in the experience and in certain common-sense domestic habits of the people themselves; as for instance, the smearing of oils on the surface of the body and exposure to the sun's rays thereafter, the smearing of the mud-floor of village hamlets with freshly prepared solution of cow's dung—a practice possibly based on its disinfecting and therefore hygienically sound principle, considering the easy availability of the product domestically. I mention it merely as an example to select from indigenous habits, although not directly applicable perhaps in malaria prevention.

The Mosquito Factor.—In Ceylon, eighteen different species of *Anopheline* mosquito is known to exist. Of these, only one species—*A. culicifacies*, is actively concerned in the transmission of malaria. That there is the possibility of some other species also occasionally acting as vectors is mentioned. This species is essentially a 'dry zone' dweller and is rare above elevations of 2 or 3 thousand feet and seem to show a predilection for sun-lit pools in river beds, but it also breeds freely and frequently in several other places, more especially in wells (where the sun never enters) and in drains, pits, and pools after rains. Although this species is regarded as the routine transmitter of malaria in the Island, endemically as well as during the great epidemic of 1934, still much reasonable doubt by competent authorities is expressed if an exclusively mosquito theory can adequately account for the causation of so widespread an epidemic as this recent one, without inclusion of other factors. Although this variety of the mosquito is known to occur also throughout India, their breeding is kept down, as some suppose, by modified agri—and piscicultural methods there; while, in Ceylon, their breeding is connected with the state of the drying rivers—a natural cause, not man-made. A good deal still remains to be understood as regards the habits of these *anophelines* and the precise factor in their selection of the breeding waters. This species, as also several others of the family, are known to be indifferent feeders on the blood of man, of cattle and other animals. It is fancied by some

observers that the disposition of cattle around human dwellings might possibly determine the mosquito's choice of its host. In Holland, for instance, the absence of infected mosquitoes from the houses in the warmer summer months, is attributed to cattle being thirty-three times as attractive to them as man; but in the colder months of the year, the insects stay in the houses, instead of going out to lay their eggs and not returning as they usually do in the warmer months of the year. A high infection of the *A. maculatus* was noticeable in animal houses and less in dwellings, and larger numbers of these insects were found in the stables than in the houses. We are reminded however that the Anopheline habits are different in temperate climes such as Holland, and such peculiarity does not obtain in the more malarious tropical areas. It would seem that further study is needed in this matter. States an observer from Italy, "that the breeding of cattle represents a more important aim than the breeding of *Gambusia* to devour the larvae, namely the obtaining of anopheles without malaria" (*B. M. J.*, Aug. 13/27).

The League Commissioners, in their travels through Europe, give the instance of a place where, in a pig farm, a line of stables acted as a trap for the anophelines and effectively screened the villagers' huts from a marshy area, where the anophelines bred freely. The two anopheline carriers in Macedonian War area have been reported as having been found to feed largely on buffaloes. Some workers on the habits of malarial mosquito carriers even indicate that mosquitoes can be induced—some with ease, while others with difficulty—to feed on human blood. We are told that as a rule only the female anopheles sucks human blood, while the males are plant-juice feeders. The female sucks blood, which, apparently it does with a view to obtain nourishment for its eggs. Possibly this urge is somewhat of the nature of the 'longing' for some kinds of food as acid fruits, ash, earth etc. that some women get while pregnant. The female mosquito has also been known to feed on vegetable juices. It is said that she feeds on blood once a day, in nature.

The Parasite Factor:—In all malarial epidemics that have been investigated, the parasite of malignant tertian malaria (*P. falciparum*) has been found to play a predominant part. In the Punjab and Sind, the parasite of benign tertian malaria predominated during the initial stages of the epidemic, but it was soon replaced by *P. falciparum*, which was almost exclusively present at the end of the epidemic. In the case of the great epidemic of 1934 in Ceylon, the observations seem to tell a somewhat puzzling

story. The Pathologist of the Colombo General Hospital found *P. falciparum* in 67.2% of the 2574 films, *P. vivax* in 32.7%, and no infections with *P. malariae*. Somewhat discordant are the figures from laboratories outside Colombo, which show, *P. vivax* 60.2%, *P. falciparum* 38.5%, *P. malariae* 1.1%. Some doubt is cast on the reliability of some of the latter findings, while it is somewhat vaguely assumed that a larger proportion of grave forms of malaria were admitted to the Colombo General Hospital than elsewhere. It is however remarked that the benign tertian parasite probably played a predominant part in the recent epidemic in Ceylon, but when one reflects on the appalling mortality from a malarial outbreak one hardly thinks the parasite could be regarded as particularly happily named. Although the quartan parasite has not apparently played a great part in the epidemic, still it is significant to notice that in certain times and places before the epidemic (1922-23) this quartan variety was found, from an examination of a large number of blood-films from all over the Island, to have been more prevalent (*i.e.*, 33.7%) than it is in most parts of India.

In this connection, it is of interest to read the remarks on primary infections and relapses made by some observers, "that it made one wonder whether almost all cases of subtertian malaria seen in the tropics are not freshly acquired mosquito-borne infections, whereas the majority of benign tertian infections seen, are relapses of old standing infections." (Knowles and Senior-White, 1933). One also wonders why the quartan parasites that have figured fairly high in endemic malaria some years before the epidemic in Ceylon, should have been almost non-existent from the bloods examined during the recent epidemic. How is the phenomenon to be understood that one species predominates at the initial while another species almost completely dominates the blood picture towards the close of an epidemic? Can benign parasites create malignant results? It has been observed that the same species of the parasite may exhibit varying degrees of virulence in geographically different areas.

Not alone the seed but the soil also has to be considered. Abnormal meteorological conditions may, and as have quite obviously affected the recent Ceylon epidemic, determine an increase in the virulence of a particular strain of the parasite. Similarly a given population might possess different resisting capacities in different years when the epidemic visits them. This was the case in Ceylon after drought and famine conditions have devitalised the population as a whole. The greater resistance of the

Australian is likewise attributed to better economic conditions and better nutrition of the race than of the aboriginal tribes.

Gill (1928) regards the seasonal periodicity of malaria as being dependent on a two-fold cause: some quality inherent in the specific parasite and some favourable environmental condition that occurs at irregular intervals. It has been suggested that it may be some form of an urge which impels the female mosquito to seek human blood and to utilise any breeding place available near to where food is found. In regard to this "some inherent quality", one is strongly reminded by analogy of the appearance at night only and disappearing during day time of the filarial embryos from the peripheral circulation at a time most suited to the night feeding habits of the mosquito, perhaps also as the result of an urge.

Importance of Quinine.—In Italy and in Greece, the State supplies quinine to the poor. The sale of quinine at cost price is done in some countries that aim at malaria reduction. When the epidemic raged in Ceylon recently, at one stage, it was very difficult to obtain quinine in the open market owing to its shortage (the writer had to hunt for it one day and had to pay Rs. 5 for an ounce of Howard's sulphate of quinine).

There can be no two opinions that the supply of quinine at cheap price in malarial countries as India and Ceylon is a matter for serious thought by all. It is said that the Dutch people who have large cinchona plantations in Java control the present price of quinine. Why cannot cinchona be more widely cultivated in Ceylon and India? It is said that in Ceylon, in the year 1887, there were 30 thousand acres of land under cultivation of cinchona, with 40 million trees, and 16 million pounds of bark were exported; and this had the effect of reducing the market value of quinine in Europe from 12 sh. to 1 sh. per ounce. There can be no doubt that if cinchona be cultivated in India and Ceylon, attempts could be made to popularise its derivatives through cheapness, and by causing them to be employed more largely as household remedies. The Red Indian taught the secret of the bark to Countess Cinchon. The malarial villagers of India and Ceylon could likewise be trained to befriend this plant. Therein lies, it seems to fitably travel, at least in so far as the economically backward, tropical, malaria-stricken peoples are concerned. Every single village, where the plant can grow, should have its own cinchona garden, from where crude parts of the plant could be taken for home-remedies freshly brewed; and also perhaps the malarial

villagers might be encouraged to chew the same along with the betel-chew.

Other methods have also got to be pursued with the aim of making finer products of quinine, cheaper. This is a matter for the manufacturing enterpriser, and for the Government and its 'quinine policy.'

Behaviour of Indigenous Malaria in Areas of Low Endemicity

BY

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Introduction

A TYPICAL case of malaria is perhaps the easiest thing for diagnosis by a medical man. An atypical case on the other hand presents the greatest amount of difficulty in diagnosis. Like hysteria, it may be said of malaria that there is no disease on earth which cannot be simulated by it. In the case of the former, the physician has a valuable guide in the age and sex of the patient, whereas, there is nothing like them in the latter case. Parasitic finding, the sheet anchor of diagnosis in malaria, is of value only in positive cases. A negative finding, though of value in cases of children who have not been given any anti-malarial remedy within a pretty long time, has nothing to speak of in its favour in the case of the grown-ups and in individuals who have been treated with any anti-malarial drug recently. Parasites are seldom found in old persons suffering from undoubted malaria. Again, mysterious are the ways in which an anti-malarial drug causes disappearance of the parasites from peripheral blood. The smallness of the quantity of the drug that causes it and the rapidity with which it is effected, make it obvious that it must be something other than destruction of the parasites by the drug. From a case in the clinic, a Hindu, male, aged 25 years, a slide was taken which showed plenty of parasites—*plasmodium malariae*. He was immediately given an intra-muscular injection of quinine bi-hydrochlor 10 grains in 2 c.c. Just half an hour after the injection another slide was taken, which did not show a single parasite even after careful search.

Such are the fallacies of parasitic finding. Yet for the sake of accuracy, I shall consider the behaviour only of those cases in which malaria parasite was found sometime or other during the course of observation.

Symptomatologic differences of different epidemics are well known. Epidemics at different places and at different times show variations in the course of the disease and in complications which cannot be explained thoroughly. Whether they depend on the peculiarities of particular strain of parasites or on the peculiarities of the sufferers or on the geological or climatic condition of the locality, has not been worked out satisfactorily. Like the prevalence of an epidemic in the locality in case of small pox, the occurrence in an endemic area and the season of occurrence go a long way towards diagnosis of malaria. But in an area of very low or unknown endemicity, the physician is confronted with much difficulty. The difficulty is all the more increased on account of the fact that cases occurring in such an area often differ in a variety of ways from cases that occur in an endemic area or are imported from an endemic area.

Endemicity of Calcutta.—Calcutta, the capital of Bengal Presidency, has for a long time the reputation of being least infected with malaria, though surrounded on all sides by a dense malarious zone. Considering the huge population of the city, the number of indigenous cases that occur here are negligible. The Health Officer's report for the year 1934 shows that death rate from malaria in the city was 59 per thousand population, whereas the average death rate from the disease in the province was 13 times as much. The actual difference, when only indigenous cases are considered, is perhaps many hundred times greater; because, (1) A vast majority of the cases of death in Calcutta are imported—rural people suffering from malaria having come to the city either for treatment or to earn a living and having died there. (2) The towns-people in their cunningness often pass other diseases, which they do not want to disclose *e. g.* tuberculosis, as malaria, whereas in rural areas the people are more simple minded and on account of their constant acquaintance with malaria they think lightly of the disease and in case of death, try to find and report other causes (3) The whole of rural Bengal is not equally malarious. A major portion of the districts, comprising Eastern Bengal, is practically free from malaria; so the death rate in the endemic area must be very much higher.

So far about death rate: the difference in the amount of suffering is far greater. There are villages in Bengal, where

almost every inhabitant suffers from malaria, whereas in Calcutta the sufferers from malaria form only a negligible fraction. Covell (1932) after careful investigation of the incidence of the disease in the town has declared that the amount of endemic malaria existing in Calcutta must be very slight.

Investigation.—During the 12 years, 1924-1935, I examined the blood of 3584 cases clinically diagnosed as malaria and at the time residing in Calcutta. Of these, 2147 or 59.9% cases showed malaria parasites in their blood. Banerjee (1934) mentions that in a series of 4500 cases of malaria in the Medical College Hospital, Calcutta, malaria parasites were found in approximately 53%. Among the parasite-positive cases of my series, 93 gave the history of either never being out of the town or having been so long ago and having had no fever in the intervening period, they could be presumed to have got the infection in the city. With a view to study the endemic question more closely, I took a more detailed history of the previous movement of the patients that attended the clinic and a charitable dispensary under my control during 1936 and were clinically diagnosed as malaria. There were 307 cases in all of which 188 had malaria parasites in their blood (*i. e.* 61.2 %).

While studying the question of endemicity, several differences in character and behaviour were observed between cases which acquired their infection in Calcutta and those which had their infection from a highly or moderately endemic area outside the city. These form the subject of this paper.

The 188 parasite-positive malaria cases, studied during 1936, were divided into the following seven groups:—

Group 1. Those who were normally resident of a known endemic area and had come to Calcutta on a short visit either for treatment or for any other purpose—29 cases.

Group 2. Those who had been to an endemic area within 2 months—53 cases.

Group 3. Those who had been to an endemic area more than 2 months ago but were having attacks of fever during the intervening period *i. e.* between the time of coming from an endemic area and coming under observation—48 cases.

Group 4. Those who had been to an endemic area more than two months, perhaps years, ago but had no fever during the intervening period—23 cases.

Group 5. Those who had never been out of the city boundaries—22 cases.

Group 6. Those who had been out of the city to an area, the endemicity of which is not known—10 cases.

Group 7. Those who could not give any history or gave one which was not reliable—3 cases.

The last two groups are not taken into consideration on account of uncertainty; cases in group 5 had obviously got their infection in the city. In cases of group 4 there is the possibility that the infection was obtained outside the city, and remained latent in the individual only to produce manifest fever during observation. But the probabilities are against it. It occurs only in rare instances where the infection remains latent for a long time so that it may safely be concluded that all the cases of this group—at least nearly all of them—had their infection in the city like cases of group 5. The cases in the first 3 groups might on the other hand have got the infection in the city but the probabilities are that they got the infection from without and they are therefore regarded as such. Thus we have 45 cases (groups 4 and 5) who got infected in the city where endemicity is very low and 130 cases (groups 1, 2 and 3) who had their infection outside the city from an area, whose endemicity was much higher. On close observation and subsequent following up, these two series showed some differences on general lines.

Character of Fever.—The character of fever is often typical. Low fever which comes and goes often without any knowledge or disturbance of the patient was not found in the series under consideration. Such types of fever were very commonly found in cases of group 1. Bhattacharya (1936) reports cases with "slow" fever from a highly endemic village in Bengal. The only atypical fever in this series was a typhoid-like continuous fever with ladder-like beginning. These cases were very misleading, specially having occurred in a city where indigenous malaria is rare and typhoid fever common. The only difference, these cases had, from typhoid fever, so far as the character of the fever was concerned, was observed in some cases in the form of a sudden drop, perhaps one morning usually in the middle of the 2nd week, without any apparent reason. If still not treated in antimalaria line, the subsequent temperature took one of two courses: it either began again to run a continuous course for a few days more to drop again to subnormal another fine morning, perhaps a week after, or it may

from that date take up a typical course for malarial fever, *viz.* coming with rigor and leaving with sweat every day or every other day.

Response to Treatment.—Response to quinine and other antimalarial remedies was very prompt, rapid and thorough in these cases. Quinine-fast cases which are often found in more endemic areas and in some instances of group (1) was never found in this series.

Relapses.—Relapse is the rule in malaria, specially when, as is unfortunately often the case, the treatment is inadequate. But the cases under consideration, seldom showed relapse though it must be admitted, the treatment was often inadequate, attributable mainly to the unpleasant taste of the remedies and outdoor patients not always obeying instructions. This behaviour throws a new light on the question of relapse in malaria. Relapses are of course common in malaria but what are almost always regarded as relapses in a highly or even moderately endemic area are in many instances cases of reinfection rather than relapses.

Spleen.—Enlargement of spleen is perhaps the most constant feature of malaria. In most endemic areas, the spleen index is higher than other indices namely fever index and parasitic index. But in the present series of 45 parasite-positive and fever-positive cases of indigenous malaria, spleen was found palpable only in 3 cases (6.7%). In my previous series of 93 parasite-positive indigenous malaria cases in Calcutta during 1924-35, spleen was palpated in 78 cases and found palpable in 6 cases (7.7%) only. These low incidences of enlarged spleen contrast markedly with the high incidence of 102 (*i.e.* 78.5%) in 130 parasite-positive imported cases. Of them again, the cases of group (1) had spleen index of 97.3% and group (3), 89.2%. This wide difference in spleen index between the local and the imported cases demands careful consideration. Another important fact about spleen is the time when it becomes palpable. In the imported cases it is either palpable from the beginning or becomes so very early in the disease whereas in the indigenous cases it takes some time—in many instances more than a week, to become distinctly palpable. In those cases which were treated early, spleen was never palpable. In endemic areas, it is probable that the splenic enlargement begins early with small doses of infection which may or may not be sufficient to cause fever. Or it may be that such small doses of infection produce a state of hypersensitiveness which results in allergic enlargement of spleen when a subsequent dose of infection comes.

Conclusions.—Indigenous cases of malaria in an area of low endemicity present certain characters by which they differ from cases in areas of higher endemicity.

Atypical cases of fever less common, low fever never found.

Response to quinine is good. It never failed in any case of my series.

Relapses are seldom found.

Spleen takes longer time to enlarge. In cases treated early, splenic enlargement is not found.

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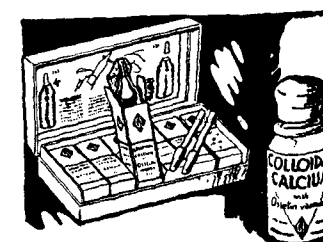
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Malaria Epidemic in Travancore

BY

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IN recent years nothing has excited the human appeal in this country more than the great malaria epidemic which is the most severe outbreak that Travancore has ever witnessed, though it stands only second to the Ceylon epidemic of 1934. As is well known, there are several areas in our country, lying all along the foot-hill of the western ghats, which are subject to the ravages of malaria, resulting in depopulation of fertile tracts and undermining the health of the people. Major Mulligan, in one of his reports, referring to the inhabitants of malaria-infested villages, aptly describes them as 'living corpses' and says that he was less moved by the spectacle of the Quetta disaster, than by the sight of the malaria-stricken inhabitants of our country. Sudden disasters like earthquakes, floods or volcanic eruptions, are bad enough in all conscience, or a visitation of plague, cholera or small-pox, but the slow killing and undermining of the people through the effects of malaria are not less serious; and yet the slow death of thousands of people due to a disease, which is preventable, seems hardly to make any impression either on the people or on the Government, because it lacks the element of dramatic suddenness in it.

The recent epidemic however came as a thunderbolt to a people who were already impoverished by the failure of the crops, brought about by the same drought which caused the malaria. It started early in 1935, in the originally endemic areas and very soon invaded fresh interior areas, with alarming rapidity, the taluks of Neyyattinkara, Vilavancode and Nedumangad, bearing the brunt of the explosion. It was indeed an unexpected and unprecedented emergency which attracted more than usual attention on account of its unusual dimensions and exceptional features. From August 1934 onwards, rainfall in Travancore, was generally deficient owing to the partial failure of the North-East monsoon and no district could show anything even near the average rainfall. The drought continued unabated with the consequent failure of crops, and famine conditions came into existence.

The abnormalities of the rainfall gave rise to the expectation of an increased malaria incidence in the endemic areas, but the overwhelming epidemic, which broke out with startling suddenness, was not however anticipated. By February 1935, the

Hæmatology of Malaria*

BY

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BLOOD of 3584 cases, clinically diagnosed as malaria, was examined during 12 years, 1924 to 1935. Whenever time and opportunities were available, as much detailed examination as possible was done. The following were the items of examination:—

1. Total R.B.C. Count.
2. Total W.B.C. Count.
3. Differential leucocyte count. (Schilling method)
4. Arneth count.
5. Halometric reading.
6. Reticulocyte count.
7. Thrombocyte count.
8. Hæmoglobin estimation.
9. Sedimentation test.
10. Volume index.
11. Icterus index.
12. Aldehyde test.
13. Urea stibamine test.
14. Fragility test of R.B.C.
15. Coagulation time estimation.
16. Calcium estimation.

It was not possible to do all the items of examination in all the cases of this big series. Some of the tests were applied to only a few of the cases while others were applied to a majority of them. Calcium estimation and coagulation time determination were begun in the latter part of the period and were done only on a very small number of cases. These items are therefore omitted from this report on account of their limited statistical value. The number of cases on which each of the other items were applied are noted against individual items along with the findings.

Of the total number of cases only 2147 or about 59.9% showed malaria parasites in their blood. Banerjee (1934) mentions that in a series of 4500 cases of malaria in the Medical College

* Read at the Reunion of the Medical College of Bengal, held in December 1936.

Hospitals, Calcutta, malaria parasites were found in approximately 53% and reports that out of his own series of 62 cases, 58 % were parasite-positive.

In this paper are recorded and discussed the hæmatological findings in those cases only which were parasitologically positive. The findings are compared with the normal findings by various authors. Local data for comparison are (1) for males, those of Drs. Basu and Chatterjee, derived from 50 normal persons from amongst students, members of the staff and menials of the Carmichael Medical College, persons who were performing their ordinary avocations without any subjective or objective complaints and (2) for females, those of Dr. J. Dhar from 86 healthy non-pregnant Bengalee women between ages of 16 and 35, who were students (of different colleges), probationary nurses or healthy female relatives of indoor patients of Chittaranjan Seva Sadan, Calcutta.

As it is not possible to report the findings of the individual cases of such a large series separately, only the mean, the highest and the lowest findings are reported. The average or standard deviations are also not given on account of the enormous mathematical calculation involved therein.

The methods used were standard and recognised ones that are used in common laboratory practice and are therefore not described in detail. Only the bare outlines are given in most cases. Greater details are given where any special method has been used.

(1) *Total R. B. C. Count*—Ordinary Thoma-Zeiss counting chamber with parallel trenches (Burker type) was used and dilution made 1 in 100 with Toisson's fluid.

No. of examinations made	...	1432
Mean count per c.mm.	...	2,584,371
Highest count per c.mm.	...	6,120,000
Lowest count per c.mm.	...	1,160,000

This shows very marked anæmia when compared with the normal figure of 4,782,000 per c.mm. for average healthy Bengalee male or even with the very low figure of 3.73 millions per c.mm given by Dhar for average healthy Bengalee female.

(2) *Total W. B. C. Count*.—Thoma-Zeiss counting chamber was again used for total W.B.C. count. Dilution was made 1 in 10 with 3% acetic acid solution tinged with methylene blue. It was found that 0.5% solution of the acid which was used formerly

was not sufficiently capable of disintegrating the R.B.C. completely, especially after some days' keeping.

Number of examinations made	... 2175
Mean count per c.mm.	... 4,224
Maximum count per c.mm.	... 14,100
Minimum count per c.mm.	... 3,500

The normal count of W.B.C. is very variable. It may double itself in the same individual in the same day. Even counts at the same sitting have been seen varying from one another by 5, 10 or even 20 per cent. Physiological variation may be due to conditions of food intake and exercise. Variation in cases of a particular disease may be attributed to the disease itself when it is found in a majority of cases and the mean of many patients varies from the normal mean. Basu and Chatterjee put 7,420 as the average normal count and from 5500 to 11900 as the normal limit. Accepting this standard of normal count the total W.B.C. count of my series of malaria case was on an average 56% of normal. Taking into consideration the duration of illness, it was found that the total W.B.C. count, as also the total R.B.C. count, gradually diminished as the disease progressed.

(3) *Differential Leucocyte Count.*—Smears for differential leucocyte count and parasites were drawn on grease-free slides by Daniel's method. A thick film was made at one end of the slide sometimes by spreading a drop round and round with one corner of another slide and finishing at the centre of the drop and at other times simply by lifting the slide used for making a thin film just before the thin film was completed so that at one end of the thin film there remained a rather thick line of thick film.

The smears were stained by Giemsa's method or by the 'Panoptic method' described by Pappenheim, which is as follows: The smears are first stained with Jenner's stain for 3 minutes, then equal amount of neutral distilled water added, thoroughly mixed and kept for one minute more. Then the stain is thrown away and immediately replaced by dilute Giemsa's stain (one drop of stain to 1 c.c. of distilled water) which is allowed to act for 10 minutes. The slide is then washed in neutral distilled water and allowed to dry at room temperature.

Differential count of leucocytes was done in all cases. With the exception of some of the early cases, every count was done by Schilling's method in which different kinds of leucocytes, young and old, are expressed in percentage of the total number of

leucocytes. Only Schilling counts are taken into consideration in this report.

According to Schilling's method, the neutrophile leucocytes are divisible into the following four groups:—

- Myelocytes with round or nearly round nuclei which are the youngest forms obtainable in peripheral blood. These correspond to the "M" forms of Arneth.
- Young metamyelocytes with slight indentation of the nuclei also known as "Juvenile," roughly corresponding to "W" (Weening) type of Arneth.
- Older metamyelocytes with deep indentation but no true lobulation of nuclei i.e. with horse-shoe shaped or S-shaped nuclei commonly known as "band" forms or "stab" forms corresponding to "T" (Tief) or "S" (Schilling) types of Arneth.
- True polymorphs or "Segments"

Schilling count was done in 1752 of the positive cases. The individual items of the Schilling haemogram are considered separately. The findings were as follows:—

	Found in cases percent.	Mean percentage.	Maximum percentage	Minimum percentage
Myelocytes	3	0.02	5.5	0
Younger Metamyelocytes (Juvenile)	29	0.13	7.2	0
Older Metamyelocytes (stab)	94.5	12.1	18.7	0
Segments	100	43.1	63.5	23
Lymphocytes	100	29.6	50	11.5
Monocytes	100	7.6	22	1.5
Eosinophiles	100	9.9	27	3
Basophiles	42	0.7	4.0	0

Among the neutrophiles the total percentage of nonsegmented forms (i.e. the myelocytes and the younger and older metamyelocytes together) is of some importance. Schilling himself quotes 4 as normal for this. But Basu and Chatterjee have shown that

the normal figure for Bengali male is 10.5 and Dhar has shown that for normal Bengali female it is 11.23. The average reading of my series of malaria case was 12.55 *i.e.* above normal. The segments (segmented forms) were slightly below normal. The lymphocytes were fewer than normal (Basu and Chatterjee's normal of average was 37%). Monocytes were increased much above normal (3%—Basu and Chatterjee). Eosinophiles were slightly above normal figure of 7% (Basu and Chatterjee).

Stott (1915) from an observation on a large number of malaria patients gives the following average differential count and compares it with the average count of 25 healthy natives.

	Malaria Cases.	Healthy Natives.
Polymorphs	64	70
Large mononuclears	21	10
Small mononuclears	14	16
Eosinophiles	1	4

Large mononuclears in his scale obviously included the monocytes and the large lymphocytes.

(4) *Arneth Count*.—Arneth count was done in 383 cases by Cook and Ponder's modified method in which segmentation of nucleus was recognised as such only when the individual parts were connected by means of a chromative thread and nothing else. This is now recognised as the ideal method and it makes a lot of difference from Arneth's original method in which simple narrowing of nuclear material was regarded as segmentation. Maximum Arneth index was 88 in a case which had the following Arneth hæmogram:—

I	II	III	IV	V
34	46	16	4	0

Minimum index was 46 in a case with the following Arneth hæmogram:—

I	II	III	IV	V
4	17	50	19	10

Average of Arneth indices was 69. This is much higher than the average normal index which is 61. The Arneth hæmogram of malaria cases therefore shows decided shift to the left.

(5) *Halometric Reading*.—Halometric reading was taken in 561 cases by means of an Eve's halometer. The maximum reading of average diameters was 7.48 μ while the minimum reading was 6.6 μ . The mean of all the readings of average diameters was 7.17 μ . According to Price-Jones' (1933) latest figure the normal average diameter is 7.202 μ . Napier and Sharma (1933) declare 7.216 as the normal figure for Indian males. Basu and Chatterjees' figure which was 7.4 μ was rather high probably, as stated by them, on account of the fact that the readings were taken during the busiest part of the day. Dhar's normal figure for Indian women was very low being only 7.003 μ . If we take the mean of Basu and Chatterjees' (male) and Dhars' (female) figures as normal it comes to 7.2015 *i.e.* very nearly Price-Jones' figure and comparing with that, the average corpuscular diameter is diminished to a small extent in malaria.

(6) *Reticulocyte Count*.—For reticulocyte count supravital stain was used. Special fat-free slides complete with a film of stain (0.5% brilliant cresyl blue in alcohol) on one polished surface were kept ready and a small drop of blood direct from the finger tip was taken in the middle part of a polished coverslip and placed immediately on the slide and allowed to spread and remain for 15 minutes after which the coverslip was drawn across the slide and the stained blood smear allowed to dry.

The highest figure in my series of malaria cases was 13.4%, lowest 0.2%. Average count was 1.2%. While European authors obtained in normal individuals figures varying between 0.7% and 2.6% with a mean of 1.5%, Brahmachari's (1934) figure for normal healthy Indian adults was an average of 0.5% with range of variation between 0.17 and 1.04%. Basu and Chatterjee quoted normal figures 0.3% to 1.6% with a mean of 0.8% which is midway between the previously mentioned quotations. In my series of malaria cases the average count was 1.2%. This is distinctly higher than the normal Indian average. This is probably due, as Benhamon thinks, to the response of bone marrow to the destruction of blood caused by malaria parasites. In one of his cases of malaria with high degree of anæmia (R. B. C. 1,300,000 per c. mm.) the count rose up to 19% even without any treatment.

(7) *Thrombocyte Count*.—Thrombocyte count was done in 288 cases. A drop of filtered 14% solution of magnesium sulphate was placed on the cleaned finger tip and puncture made through it. The blood as it oozed out through the drop was mixed with it. A smear was made with the mixture fixed in methyl alcohol for 5

minutes and then washed thoroughly with neutral distilled water to remove any trace of magnesium sulphate. It was then stained in the usual manner with Giemsa's stain and both R. B. C. and platelets in several fields were counted until at least 1000 R. B. C. were met.

Maximum count was 538,000 per c.mm., minimum was 69,000 per c.mm. Average was 162,837 which was slightly less than normal average (222,100—Basu and Chatterjee).

(8) *Hæmoglobin Estimation*.—Hæmoglobin estimation was done by the very rough method of comparing with Tallquist's scale. Highest finding was 100%, lowest 20% and average was 43% which indicates fairly high degree of anaemia.

(9) *Sedimentation Test*.—Determination of rate of sedimentation of erythrocytes was done in 1621 cases. Venous blood was collected and mixed within the syringe with one fourth of its volume of 3.75% sodium citrate solution in distilled water. The contents of the syringe were next put inside a narrow test tube 13 c.m. x 4 m.m. (internal diameter) and graduated in millimeters upto 125 m.m. from the bottom. Just sufficient blood was allowed to run into the tube to bring its level to the top of the graduation i.e., just up to 125 m.m. Readings were taken every 15 minutes for 2 hours and then again after 24 hours. From these readings, only the highest rate was taken as the rate of that particular sample of blood and expressed in millimeter per hour. Highest rate recorded was 56 millimeters per hour and lowest 6 millimeters per hour. Average rate was 19 m.m. per hour. It was generally observed that cases of recent infection had comparatively low rate while in the old standing cases the rate was high. 6 m.m. per hour is believed to be the average normal rate and 0 to 12 m.m. per hour the normal range.

(10) *Volume Index*.—Volume index or the percentage ratio of the volume of packed corpuscles to the total volume of blood was determined in all cases in which sedimentation rate was determined. The tubes in which the corpuscles had settled for 24 hours were centrifugalised until no further diminution in volume of the packed corpuscles was noticed and the top of the corpuscular level at once gave the volume index because, as the blood was diluted with one fourth of its volume of the citrate solution, 125 volumes of the blood and citrate mixture contained exactly 100 volumes of original blood. Normal index is from 46 to 48. The maximum in my series of malaria cases was 47.5 and minimum 9. Average was 24.2 which indicated a fairly high degree of anaemia.

(11) *Icterus Index*.—Icterus index was determined in 733 cases. It was done by comparing the colour of the serum in a comparator with standard solutions of potassium dichromate in weak sulphuric acid solution. (2 drops of concentrated acid in 100 c.c. of distilled water). The solutions are graded in strengths corresponding to icterus indices of 1, 3, 5, 7, 10, 12, 15, 20, 30, 50 and 100. Normal range of icterus index is from 2 to 5. In my series of malaria cases the lowest index was 6 i.e. above normal limit, the highest was 35. Average was 10.4. This constant high rate was in all probability due to constant breaking down of red corpuscles and formation of bilirubin from hæmoglobin.

(12) *Aldehyde Test*.—Aldehyde test was done in 1236 cases and in doing so, Napier's technique was strictly followed. It was found negative in all the cases.

(13) *Urea Stibamine Test*.—Urea stibamine test was done in 1572 cases. In most cases it was done with diluted serum and in some cases with undiluted serum. It was negative in all cases with fresh infection in which spleen was not palpable, while in cases of longer standing and fairly big spleens it was almost always positive though the cases were all parasite-positive malaria cases, thus showing that it has got very little diagnostic value as a test for Kala-Azar.

(14) *Fragility Test of R. B. C.*—Fragility of R.B.Cs. was tested only in 65 cases. It was done by Sanford's method which included the range from 28% to 50% of sodium chloride. When the range of hæmolysis was seen to go beyond these limits the test was repeated with a fresh set of tubes with higher or lower range of strength of saline.

The case with most fragile corpuscles showed the range between .50 and .40. The case with least fragile corpuscles showed the range between .40 and .32. Average of maximum points was .443 and average of minimum points was .346, which were very near the normal limits.

Summary.—Haematology of only the parasite-positive malaria cases is reported. It deviates from normal in the following way. The total count of R.B.C. is markedly diminished. Total W.B.C. count is also diminished specially in long standing cases. Schilling hæmogram shows increase in younger neutrophils, monocytes and to a less extent in eosinophiles, and diminution in the "segments" and the lymphocytes. Arneth hæmogram shows decided shift to the left. Halometric readings reveal only slight

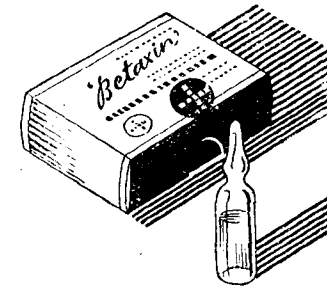
diminution of average corpuscular diameter. Reticulocyte percentage is increased. Thrombocyte count shows only slight lowering. Haemoglobin estimation indicates fairly marked anaemia. Erythrocyte sedimentation rate is much exaggerated. Volume index is markedly diminished. Icterus index is increased much beyond normal limits. Aldehyde test is negative in all cases. Urea stibamine test is negative in early cases, positive in cases with fairly enlarged spleens. Fragility of R. B. C. remains near normal.

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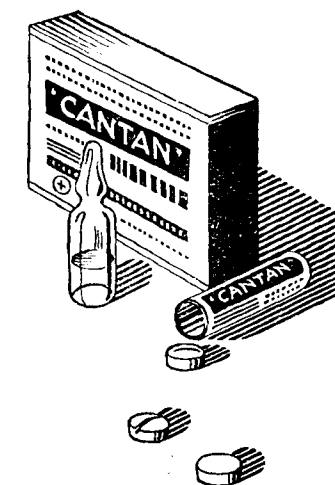
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Malarial Vagaries and their Treatment

BY

Dr. Satis Charan Mallik, M.B., D.T.M.,

Giridih.

Introduction

By Malarial Vagaries I mean the irregular manifestation of malaria, not definitely pointing to its typical clinical symptoms and thereby misleading the average medical practitioners. It is a matter of pity that a large number of people die of malaria every year in spite of the settled fact that malaria is caused by definite parasites, transmitted from infected man to a healthy one through mosquito bite and that quinine is the specific remedy. Why is this? To me it seems to be that :—

(1) Atypical manifestations of malaria occur in abundance which are hardly described in our ordinary text books.

(2) The number of qualified and competent medical hands is limited in mofussil towns, and villages where the disease predominates and the atypical cases are consequently overlooked and seldom reported.

(3) Abundance of illiterate quack practitioners, ignorant of necessary facts regarding clinical symptoms, diagnosis and treatment of the disease.

(4) Economic condition, want of health, education, apathy of lay public etc. etc.

It really pains me to note that an unfortunate child died of malaria with vague symptoms of malignant infection undiagnosed and without proper treatment even in a metropolitan city like Calcutta in the house of an eminent physician.

All these atypical cases, however, are due to the infection with *Plasmodium Falciparum*. This fact has been corroborated by laboratory findings in the majority of cases. The examination of peripheral blood was negative in some of the cases, but the diagnosis was confirmed by the therapeutic test. These cases with irregular symptoms should be grouped in the list of pernicious attacks. These pernicious symptoms vary according to the localisation of attack and therefore pointing to the system affected. These vagaries are due to the maturation of the malignant tertian parasites in the internal organs, unlike the other

varieties and to the mechanical plugging of the capillaries by parasites and pigments—melanin and haemozoin. The red corpuscles containing parasites become more sticky to the endothelial cells lining the capillary, hence the mechanical obstruction by plugging is more marked.

During my mofussil practice, extending over a period of fifteen years in a place which is the hot bed of malaria, I had frequent opportunities of seeing, studying and treating hundreds and hundreds of such cases. In this quarter, malaria is frequent almost throughout the year and it plays a havoc, from a fortnight after the rainfall to the onset of the winter *i.e.* from the month of August to November. The number of deaths is generally greater in the first and last parts of the so-called season of malaria.

I am not going to describe here in detail the usual clinical symptoms of ordinary malaria which is familiar to almost every one in a malarial district. I shall touch only the varieties of pernicious types I have come across during the period of my several years' practice. In this connection, I must admit that I could not do full justice to each and every case both from the point of therapeutic and scientific value as the blood examination was not possible in all cases, where the mofussil practitioners are handicapped in more than one way and where they are apparently called for some other diseases in a place several miles away from their laboratories or dispensaries.

Clinical Varieties.—Doctors practising in mofussil districts, specially in the endemic and hyperendemic areas are quite conversant with the vagaries of malaria. The clinical types I have met with, are as follows:—

- Cerebral*—1. Convulsive.
2. Hyperpyrexial.
3. Comatose.
4. Maniacal.
5. Neuritic.
6. Head-ache.
7. Aphasic.
8. Meningitic.
9. Amnesiac.

- Intestinal*—1. Algide.
2. Dysenteric.
3. Colic with and without fever.
4. Pernicious vomiting.

- Pulmonary*—1. Broncho-pneumonic.
2. Influenzal.

- Cardiovascular*—1. Dyspnoecis with occasional angina-like pain.
2. Hæmolytic leading to hæmoglobinuria.

- Cutaneous*—1. Sudorific.

- Splenitic*—1. Acute Splenitic.

- Septic*—1. Simulating septicæmia during the puerperium

In the cerebral type there may be a good deal of organic changes in the form of inflammation of the covering (meninges) as well as the brain tissue associated with degeneration of nerve cells, disappearance of Nissl granules, thrombosis of vessels and pigments. In some such cases, Post Mortem findings may be nil.

The following clinical varieties have been described by Dr. S. P. Bhattacharjee, M.D., formerly the acting Professor of Tropical Medicine, School of Tropical Medicine and Hygiene, Calcutta, in his lectures, in describing the pernicious types.

I. Without localisation of symptoms in any system:—

- (1) Hyperpyrexial.
- (2) Toxaemic type—seen specially in Algide type without diarrhoea or dysentery.
- (3) Sweating type—leading to collapse.
- (4) Acute progressive anæmia with fatal result.
- (5) Bilious remittent—more common.

II. With localisation of symptoms in a system:—

A. NERVOUS SYSTEM:—

- (1) Cerebral.
- (2) Neuritis.
- (3) Neuralgias of various types.
- (4) Paralysis of one or both sides.
- (5) Localised facial paralysis.
- (6) Symptoms of Meningitis.
- (7) May be epileptic form of convulsion.
- (8) Affection of the special senses—
loss of hearing, sight, taste, sense of smell
- (9) Aphasia.
- (10) Amnesia.

B. ALIMENTARY SYSTEM:—

- (1) Choleric.
- (2) Dysenteric.

C. JAUNDICED TYPE.

D. RESPIRATORY SYSTEM:—

- (1) Broncho-Pneumonia.
- (2) Bronchitis.
- (3) Lobar Pneumonia.

E. PANCREATIC PERNICIOUSNESS—SIMULATING ACUTE HAEMORRHAGIC PANCREATITIS—RARE.

F. SUPRARENAL PERNICIOUS.

With lumbar pain, low blood pressure, debility fever etc.

G. CARDIO VASCULAR SYSTEM.

- (1) Acute fainting attacks with syncope and cardiac failure.

H. URINARY SYSTEM—RARELY AFFECTED.

I. OTHER SYSTEMS:—

- (1) Parotitis.
- (2) Mastitis.
- (3) Purpura.
- (4) Localised gangrene just like Reynaud's disease.

Diagnosis.—1. *Examination of blood.*—The blood should be examined in each and every case when possible. In all these cases Malignant Tertian rings are generally encountered in the peripheral blood, as the maturation takes place in the internal organs and we may not consequently get any parasite in the peripheral blood always. Blood should be examined thrice at least before a diagnosis is made. It should be taken during the height of temperature and preferably after an injection of adrenalin, when the result of the first examination is negative.

2. *Temperature of variable degree.*—Excepting hyper-pyrexial type, the temperature is generally moderate. Axillary temperatures should not be relied on. Oral and rectal temperatures, when possible should always be taken. In a limited number of cases the axillary temperature was not above normal and in one case alone, the temperature rose up after the first injection of quinine.

3. *Spleen and liver.*—There is the enlargement of the spleen, but it is not always so. Liver is enlarged in a majority of cases, specially in children and in malignant tertian ones.

4. *Icteric tinge of conjunctivae* and actual jaundice in many cases.

5. More anaemia in comparison with the period of illness.

6. Periodicity and intermittency of symptoms.

7. Exacerbation of pernicious symptoms with the rise of temperature.

8. Appearance of pernicious symptoms generally between forty two and seventy six hours after the onset of the disease.

9. There is generally a history of previous attack of fever and probably checked with quinine.

10. Lastly, in a doubtful case, an injection of Quinine intramuscularly will clear the diagnosis.

It should always be a golden rule to treat all acute cases, having irregular, pernicious manifestation with or without fever and without definite localised lesion in a particular system specially between the months of August and September, as Malaria and in most of these cases it has actually been found as such both by laboratory findings and therapeutic test.

Prognosis.—The prognosis on the whole is good. The percentage of cure is approximately cent per cent if Quinine can be pushed by injection within twenty four hours after the onset of the pernicious symptoms. It is sixty per cent within forty eight hours and uncertain after forty eight hours. The prognosis is only bad in those cerebral types where minute capillaries of brain are clogged with parasites etc. and the patient does not regain consciousness nor improve after the two injections of Quinine, one of which at least must be an intravenous one.

Treatment.—1. *General Management*—As usual, good rest in bed, in a well ventilated and lighted room, ice caps on head, hot bottles to lower extremities, evacuation of bowels, plenty of fluids, liquid nourishing diet, prevention of chill etc.

2. *Medicinal Treatment*—By drugs. The drugs of chief value are Quinine, Plasmoquine and Atebrin.

(1) *Quinine*—It is the specific remedy and decidedly is the drug of supreme value. From the outset the treatment of malaria with Quinine is an easy task but experience and judicious use of the drug are essential in curing such patients completely. By cure, I mean, the cure in its truest sense. That is, patient's trouble should be immediately checked; he should be relieved of all his symptoms; blood should be made sterile and relapse should be prevented, keeping an eye to avoid further fresh infections.

It should be noted that in all doubtful cases, blood should be taken for parasite before pushing Quinine.

Method of Administration.—*By mouth*—In all these pernicious cases, quinine by mouth should never be relied on alone. It should be used along with injection. Quinine hydrochlor and Q. bi-hydrochlor should preferably be used. Quinine Hydrobromide should be used for malarial headache and malaria with pregnancy. Quinine salicylates for Influenza-like cases of malarial origin.

Quinine should be given for a sufficient period according to the nature and virulence of infection. It can be prescribed in pill form freshly prepared but preferably in mixtures. Sugar-coated pills should never be used and relied on. Quinine can safely be given when the temperature is high but it should be given every time after a dose of alkaline mixture containing Sod. citras, Sod. Bicarb, Spt. ammon aromaticus and syrup Aurantii in water.

By Intramuscular injection—It acts as a magic in the majority of cases—10 to 15 grains of Quinine Bi-hydrochlor in 2 c.c. distilled water. The gluteal region should be selected for the site of injection on account of bigger area and the patient's attention during the time of injection is less drawn. Injections should be repeated daily till the malignant symptoms disappear and Quinine B.H. should be pushed by mouth as soon as the patient can swallow, along with the injection.

Regarding the intramuscular use of Quinine, many authorities decry the method for the formation of abscess, necrosis, tetanus etc. These are all due to faulty technique. They may develop if the skin and syringe, specially the needle is not properly sterilised, the needle is rusty, the skin wound is not aseptically closed and the needle is not pushed straight deep into the muscle. Each and every case will cause necrosis, if it is given into the fatty layer on account of its irritant action. Several hundreds of Intramuscular injections of Quinine I have given myself, none of them suppurated, nor showed any untoward effect warranting its use.

The Actual Technique.—1. *Preparation and choice of drug.*—In my dispensary, I do always weigh 10 gr. 15 gr. of Q. B. H. and dissolve the same in 2.25 c.c. of distilled water in a Test tube and boil the same till the solution is a clear, sticky, homogeneous one with its peculiar pale golden yellow colour. Outside the dispensary and on gentle patients, I use the ampoules prepared by Medical Supply Concern Limited, Calcutta. I prefer Quinine in Saccharose solution as the pain is decidedly less.

2. *Sterilisation of the Syringe etc.*—A good 2 c.c. syringe with sharp rustless needle be selected. It should be washed thrice with sterile distilled water. Absolute alcohol should then be drawn in and rejected. The syringe should then be made dry. The

outer part of the needle should further be made sterile with absolute Alcohol. Quinine in solution can now be safely drawn into the syringe.

3. *Preparation of the patient.*—The patient should always be in lying down condition and preferably on one side. I have seen several cases falling down after taking the injection of Quinine in sitting position. I, therefore, always avoid the sitting posture and generally give a dose of stimulant containing spt. ammon aromat 20 to 30 minims in one ounce of water. The upper and outer quadrant of the gluteal region should generally be selected. The skin should be made sterile with Absolute Alcohol and Tr. Iodine. The needle of the Syringe should again be wiped several times with Absolute Alcohol and immediately be pushed straight deep into the muscle. The content should then be pushed in gently and the needle taken out. The part properly squeezed and massaged and puncture of the skin is sealed with collodion or Tr. Benzoin Co. The deltoid region can safely be utilised if the solution is not more than one c.c.

With the above usual plain technique, which is almost known to all, none of my cases developed any untoward sign or symptoms as already stated. I owe an apology to my readers for stating such a common simple thing, as some of the authorities have condemned the most useful method very much, which has actually dreaded the ordinary Medical Practitioners.

It should further be added that I used to push Adrenalin in low patients. Two of my cases developed alarming symptoms, one was a Kabuli Mahammedan and the second was a thin Bengali gentleman. They developed violent twitchings of the whole body including head and neck and more like tetanic convulsion, immediately after pushing Adrenalin. I was actually frightened and you can well imagine my position. Pulse was not bad in both the cases though very weak. Plenty of cold water was poured over the head and the patient came to the proper condition after 3 to 5 minutes. This is probably due to the action of Adrenalin on the sympathetic nervous system which, in some way or other, was not in equilibrium in particular patients. Adrenalin should therefore be used with caution and I shall be glad to know of any such experience from my medical friends. In patients with low condition, I have safely used digitalin and strychnine and digitalin combined along with the injection of Q.B.H. Intra-muscular injection of Quinine will save and cure about 95 per cent of these cases and the period of suffering is decidedly less.

Intravenous Method.—Is not generally required. This route is highly advocated by authorities. It is only indicated in those cases where capillaries of brain are clogged with parasite, pigment etc. It is indispensable in these cases and intramuscular injections are no good. The following is the result of some of the intravenous Q. injections in my hands.

(1) A case of Choleric type. Intravenous injection was given—patient expired.

(2) One case of Hyperpyrexia with convulsion. Patient, adult; Intravenous Quinine was given: expired.

(3) One case with maniacal symptoms. Intravenous injection was given; cured.

(4) One case with gradual loss of memory finally leading to unconsciousness; parasites were found. Intravenous Quinine was given. Cured.

Before pushing quinine intravenously, I generally inject a dose of strychnine and digitalin. About a quarter of an hour after, I give intravenous injection of Q. B. H. I generally use $7\frac{1}{2}$ grns. in 10 c.c. with $\frac{1}{2}$ c.c. Pituitrin (P.D.) and push the piston very slowly just like the Neosalvarsan and N. A. B. injections in Syphilitic cases. I repeat the Quinine again after 8 hours but intra-muscularly and utilise the oral route as soon as the patient can swallow.

The combined intramuscular and oral method is the best, safest and surest in these types of cases excepting where Intravenous injections are absolutely indicated as stated.

Rectal Administration.—In one case I used Q. B. H. with saline rectally after an initial injection of quinine intramuscularly. It was a case of Choleric type with marked vomiting and the patient's general condition and pulse was very bad before the first dose of Intramuscular injection. I am not definite whether this route is a reliable one. However this route may be utilised in case of necessities (Case 6).

2. **Plasmoquine.**—It is an organic synthetic compound. It has been used with success in so-called Black Water fever, both orally and by injection. In children it has been used with Euquinine and Sodi Bicarb in malignant tertian cases, after a preliminary dose of quinine injection. It has got a few drawbacks. It produces methæmoglobinæmia and consequent cyanosis. It may also produce gastric irritation from abdominal pain to diarrhœa and dysentery. But all these toxic manifestations can be avoided if it is used in therapeutic doses; Sodi bicarb and Adrenalin are

used from the onset. Its use should be withheld with the onset of the toxic manifestations. It may be used alone or in combination with Atebrin and Quinine. It is of special value in Black-water fever and Quinine idiosyncrasy. The tablets should always be taken after meals. The adult dose of Plasmoquine, I generally use is $\frac{1}{4}$ gr. T. D. S. or B. D. It should be stopped for 3 to 5 days after using nine tablets altogether. It may then be repeated after the interval period. In children, I have used $\frac{1}{4}$ to $\frac{1}{8}$ tablet T. D. S. without untoward results. I never used them for more than three days.

3. **Atebrin.**—It is a dihydrochloride of 2 methoxy-6-chlor-9- α -diethyl-amino-8-pentyl amino-acriline. It is a dye stuff.

It has been advocated by many as it destroys the asexual malignant tertian parasites. In all these cases of pernicious attacks, it is unreliable. It is indeed a good adjuvant. Recently in a case of mine of mixed infection, where both immature forms of B. T. and M. T. parasites were found, I used Atebrin tablets thrice daily for three days without marked benefit. The temperature shot upto 105.6 daily one or two points less. The only improvement noticed was the definite remission. For fear of losing the patient and on account of their impatience, I had to give an intramuscular injection of Q.B.H. gr. 10 in saccharose solution (M.S.C.) and prescribed Q. B. H. in 5 gr. doses orally T. D. S. The temperature stopped rising from the very day the injection was given.

It has been said, however, in a course of Atebrin treatment extending over some time, a transitory harmless yellow discoloration of the skin may occur. This is said to be due to the deposition of the dye-stuff like remedy under the skin. It disappears soon after the cessation of the course and it is said to be harmless. The doses advocated are for adults 3 to 4 tablets daily for 4 to 7 days and for children, 1 to 2 tablets daily according to the age of the child. I generally use three tablets for 5 days in adults. I have got no experience of Atebrin injection.

To sum up, Quinine stands pre-eminent in the treatment of malarial vagaries and specially by the intra-muscular method.

4. Other symptomatic treatments—Alkaline mixture, purgative etc.

Case Notes.—1. A. H. F. Child, Rameswarpur, in Burdwan aged 7 years. I saw the patient on the 4th day of her illness. *History.*—patient cannot sit up on account of the present illness from the day before; had an attack of fever lasting for six or seven days about twenty days back. Nothing more can be elicited.

No history of accident etc. on examination. Temperature 102.4. P/R—128/38; lungs congested; spleen palpable; liver enlarged; bowels—not clear; tongue thickly coated with all the signs and symptoms of neuritis of the lower extremities. The lesion was bilateral. Malaria was then in full swing. The lower portion of the bowel was cleared with glycerine and olive oil enemata. She was given a dose of stimulant mixture containing Spt. ammon aromat and Spt. V.G. aa m viii in water. Q. B. H. 5 gr. in Saccharose solution (M. S. C.) 1 c.c. was injected intramuscularly in gluteal region and the following mixture was prescribed.

R Quinine Bihydrochlor	...	gr. 2½
Acid Phos. dil.	...	m 5
Tr. Nuxvom	...	m 5
Ammon Chlor	...	gr. 2
Sodi Sulph	...	gr. 30
Syrup Rose	...	m 15
Aqua Chloroformi	...	Ad 3½

Mft. Mist. dose one. Send 8 such. One dose every 4 hour T. D. S.

R Oil Eucalyptus	...	3 2
Camphor	...	gr. 5
Olive Oil	...	3 2
Oil Sinapis	...	Ad 3½

Mft. Lint. to rub the affected parts as directed. I heard nothing about the case next day and I thought I had lost the patient. But the father of the child came to me a day after. I learnt that the patient was better, temp. etc. were less but the pain was persisting. I gave her one more dose of Q. B. H. by intra-muscular injection and the patient reacted very well to my surprise. Mixture was continued. This seems to be a case of malarial neuritis.

2. Master Dutta—son of A.K.D., Kalna, age 11 years; I saw the boy on the second day of his illness for fever and inability to speak and on the third day of the attack, Spleen and liver enlarged Temp. 101.2°; Eyes half closed; cannot speak; could swallow and was semi-conscious. Q.B.H. 7½ grns. was injected as above. There was remission. Fever again rose upto 101°. Slight improvement regarding consciousness; aphasia and drooping of eyelids persisting, general condition seemed to be better. After 4 injections he could speak well, but he could not open his eyes properly till

the 6th. injection was given. For complete recovery he was given 8 injections altogether. This was a proper case for Intravenous injection. I did not push the I.V. injection as the patient improved steadily after the intra-muscular injection though slowly. This is an aphasic type of pernicious malaria probably with the affection of the nerve supplying the upper eye-lid. I have seen and treated only one such case.

3. A marwari girl aged 14 years at Kalna, died of high Temp. 107.6° on the 4th day of her illness. I was called in consultation. She had enlarged liver, spleen—soft and palpable. Lungs showed the signs of Bronchopneumonia. Blood was not allowed to be taken. Q. B. H. gr. 10 was injected with 1½ gr. digitalin. The girl expired after 3 hours. The temperature was over 104 at the time of death. This is probably a case of pulmonary type.

4. B. K. B. age 30, Hindu, male, said to be a very bad and hopeless dysenteric case. Numberless stools with abdominal pain, almost unconscious from pain, extreme weakness. Temperature 103.4°. Spleen and liver enlarged. On enquiry it was gathered that the number of stools and other symptoms increased with the increase of temperature. There was practically no mucus in the stool but more blood; nor was there any tenesmus. I diagnosed it to be a case of malignant malaria and immediately pushed 1 ampoule of Q. B. H. 10 gr. (M. S. C.) intramuscularly ½ c. c. adrenalin and quinine mixture orally. Curiously enough the picture was thoroughly changed and the patient came to my dispensary next morning though he was very weak. He was given a second injection and was completely cured. This is a case of dysenteric type of pernicious malaria.

5. Another case, within a fortnight, almost a similar complaint, a relation, came from 10 miles off, not in a position to procure any Doctor. Diagnosis from history was made to be a case of dysenteric type of malaria, as the patient developed the symptoms on the third day and the number of stools increased with the rise of temperature. I prescribed the following mixture and the patient was alright.

R Quinine	...	gr. 5.
Acid Sulph Dil	...	m 10.
Liq. Hydrarg Perchlor	...	m 15.
Tr. Belladonna	...	m 2½.
Spt. Chloroform	...	m 10.
Syrup Rose	...	m 30.
Aqua Camphori	...	Ad 3 1.

Mft. Mist. dose one, send 8 such, one dose every 4 hour, 4 times daily.

I have seen three to four dysenteric types of malaria every year. They respond very well with the single intramuscular dose of Q. B. H. and one injection is sufficient to check the disease and cure is generally effected after another injection or several doses of mixture orally. It is interesting to note that I have seen dysenteric and Algide types of pernicious malaria to develop every year in particular area and two cases of each type in one house, and in different areas.

6. Mr. Pande—Called in to see him for cholera. He had all the typical signs and symptoms of cholera with thready almost imperceptible pulse with cold extremities, eyes sunken etc. But the axillary temperature was 102.2° and the number of stools increased with the increase of temperature. The patient was vomiting often. It also increased with the increase in height of the temperature. There was slight enlargement of the spleen. Q. B. H. gr. 10 was injected intramuscularly with adrenalin. Rectal saline with Q. B. H. double the oral dose and glucose were given every three hours. Patient improved. Vomiting checked with adrenalin: pieces of ice to suck. Every drink, alkaline mixture etc. was given with ice. Rectal saline with Q. B. H. was stopped from the next day. I am not definite whether Q. B. H. by rectal route was helpful in this case. Q. B. H. intramuscularly was given once daily for 4 days. Q. B. H. with citric acid in pill form plus alkaline mixture etc. was prescribed. Later Q. B. H. in effervescent mixture was given.

In all these algide and dysenteric types, I use adrenalin freely besides the specific drug. Plenty of fluid in the form of barley water, water of the green cocoanuts and rectal salines.

7. Mrs. P. M., Hindu Male, age 35, wife of Station Master, called at 9 o'clock in the night for cholera, having large number of choleric stools, vomiting, etc.. The Diarrhoea started with griping and increased with the height of temperature. She was better in the morning when there was no temperature. It should be noted with interest that cholera never starts with any griping. The temperature, however, was 100.2° ; spleen palpable, liver enlarged. There was a slight amount of Jaundice which was found next morning. My diagnosis was an algide type of pernicious malaria. I at once unhesitatingly pushed Q. B. H. gr. 10 intramuscularly and it acted like magic. Two more injections were required in this particular case besides other symptomatic treatment.

Different authorities have said algide types are rare and dysenteric types are frequent and vice-versa but I say from my

humble experience that they are equally frequent and their number is not a small one.

8. A young man, aged 21 years. He was having intestinal colic daily and regularly for some time without any improvement with ordinary treatment. I was called in consultation. The spleen was palpable and comparatively hard, liver enlarged, icteric, had history of fever. There was regular periodicity. However, blood was examined; M. T. rings found. Q. B. H. injected. Pain relieved, the temperature shot upto 101.8° . However, with few more Q. B. H. injections, he was cured. This is a rare case of intestinal colic of malarial origin.

9. Hindu male—age 27 years—came to my dispensary for acute painful condition of the spleen. There was no fever. Blood was examined, M. T. rings found. Q. B. H. injected; temperature rose upto 103.0° cured after 3 more injections. This is a case of acute splenitis of malarial origin.

10. Patient, Hindu, Male—age 22 years, apparently healthy, came for bad unbearable frontal headache. There was nothing wrong with stool, urine and eye. Spleen and liver enlarged. Temperature 101.2° Q.B.H. gr. $7\frac{1}{2}$ injected, fever and pain disappeared after one injection. One further injection was given and quinine mixture with acid Hydrobrom Dil and salt was continued.

11. One local Medical Practitioner, aged 35 years, had a temperature for 3 days, not exceeding 101.6° . I was called in to see him for extreme prostration and weakness and sweating. On examination, temperature 96.2° , pulse weak, rapid, heart weak, no enlargement of spleen, liver enlarged and there was profuse sweating which lasted for 8 hours. It could not be checked with Atropine, Belladonna, Spt. Ether Sulph, Makaradhwaja, Strychnine, Calcium etc. Adrenalin 1 c.c. under the tongue ultimately was given and the doctor began to improve. He was finally cured with an alternate $\frac{1}{2}$ c. c. doses of Adrenalin. (P. D.) and Q. B. H. gr. 4 pill, each 4 doses daily. This is probably a case of sudorific type of pernicious malaria.

12. Doctor S. K. M., aged 33 years—Black Water, high temperature, fever for 3 days, maximum temperature 104.6° . Fever started with rigor. Passed haemoglobinuric urine during the height of temperature. Jaundice, anaemia, weakness etc. Treatment—plasmoquine, atebirin, orally and alternately; calcium, plasmoquine, caffeine sodi benzoas by injection. Ext. cassia bearana liquid, calcium adrenalin etc. orally; cured. A case of haemolytic type of pernicious malaria.

13. R. N. D. age about 25 years—Temperature 106.2° unconscious, convulsion, parasites found in the peripheral blood. Q. B. H. interavenous was given—proved fatal.

14. Niece of S. K. C. H. F. Ch., age 9 years, Comatose, temperature 106° , Spleen—palpable, liver—definitely enlarged. History of previous attack of fever, urine scanty, no motion etc. Blood examination revealed M. T. rings, cured with quinine-intramuscular injection.

15. Grandson of J. S. H. M., Ch., age 8 years—Went to see the child for Pneumonia which was diagnosed and treated by a Homeopath as such. From the written report I found that the fever was for the last 5 days. He had a remission on the 2nd day. Temp. varying between 99.8° to 103.2° . P/R-110/50. On examination I found Temp. 102.6° P/R-105/25; there was no sign of Pneumonia, lungs quite clear. The picture looked more like a case of Uraemia, no spleen, liver enlarged. Comatose, with occasional low muttering delirium, urine very scanty, high colour, passing once or twice a day. Fluid restricted for Pneumonia. Urine was examined. Albumin trace, just accountable for the Temp. The case was diagnosed to be a case of Cerebral type of Malignant Malaria. Blood was examined. M. T. rings found. Patient was cured with difficulty after injection of quinine, glucose etc. It should be noted that another child died of similar trouble in the same house last year under the treatment of the same Homeopath. It was probably also a case of Malignant Malaria.

16. Gonra, a child of U. P. K., age 4 years—Temperature 106.2° with occasional convulsion, spleen and liver enlarged, constipated. Bowel moved with enema. Ice applied to the head, hot foot bath etc. Temp. came down to 105.4° Q. B. H. 4 gr. was injected intramuscularly. Temperature shot up to 106.4° ; convulsion increased. With sufficient application of ice to the head and on the nape of the neck and iced rectal saline, the temperature was controlled and it gradually came down. After another 2 injections the patient was cured. Besides the injection, usual alkaline mixture and Quinine by mouth were also regularly given.

N.B.—In the Hyperpyrexial type it is always safe to lower down the temperature mechanically with water and ice by two degrees at least and Quinine should then be pushed to prevent further reaction as in the previous case. Sufficient cold water should be poured over the head where ice is not available.

17. A baby, daughter of a cultivator, age 3 years. Temperature 102.0° with convulsion, Spleen only palpable, liver was

enlarged. Q. B. H. gr. 3 was injected daily for 6 days. The temperature was controlled but the convulsion persisted. Fractional doses of Luminal, Ammon Bromide and Quinine cured the patient.

18. A female—simulating Tetanus. There was a spasm and the lock jaw which was unlike the tetanus one. There was temperature and the Trismus was less with the lowering of the temperature. Spleen was enlarged. Blood was examined. M. T. rings found. Three injections of Q. B. H. gr. 10 one daily, cured the patient, besides Bromides, Mag. Sulph etc.

19. One Female—age 22, with high temperature and all the symptoms of mania. Blood was examined, parasites detected. Intravenous Q. B. H. was given followed by oral and intramuscular administration of the drug. One 1/100 gr. of Digitalin was injected first, about 20 minutes after $7\frac{1}{2}$ gr. of Q. B. H. in 10 c.c. with $\frac{1}{2}$ c.c. Pituitrin was pushed slowly. The patient made an uneventful recovery. Three intra-muscular injections were further required.

20. A. C. M., age 36 years, had moderate temperature for 2 days. Next day temperature was 96.8 . Had the following symptoms: dyspnoea, restlessness, angina-like pain on the left chest with occasional palpitation, sweats etc. One of his brothers died of the same complaint after an injection of Q. B. H. and the injection of the drug in the particular case was disallowed. However, his trouble was over and he was cured with adrenalin, quinine and plasmoquine orally. Parasites could not be detected in the first examination but the blood picture was suggestive of malaria. This was probably a case of Cardiac type of pernicious malaria.

21. A female child, aged 1 year and half, daughter of H.N.R.—Child apparently very healthy, Temperature 106° , nothing else could be detected excepting slight enlargement of the liver. Lungs were quite clear on the first day, ratio between pulse and respiration was normal. Temperature touched the normal next day after alkaline mixture and plasmoquine and euquinine orally. I was not called in again to see the baby as she was fever-free. On the night of the second day, temperature again shot up. I was called again. On the third day, temperature 103.4° , pulse respiration ratio increased being 1:2 and all the signs and symptoms of Broncho-Pneumonia were present. On the 4th day the child expired. It was probably a case of Pulmonary type of malignant malaria.

22. A Lady (Doctor's wife) aged 25 years.—I saw her on the third day with all the symptoms of influenza, pain all over the body, especially on the lumbar region, headache, temperature

103°. Spleen and liver were not enlarged, drowsy with partial loss of sense. Blood examination showed parasites. Injection was not allowed as her heart was too weak. I prescribed the following:—

R Sodi Citras	...	gr. 15
Sodi Bicarb	...	gr. 15
Spt. Ammon Aromat	...	m 15
Hexamine	...	gr. 7½
Mag. Sulph	...	gr. 30
Tr. Eucalyptus	...	m 10
Syrup Glucose	...	m 60
Aqua Cinamoni	...	ad. 1 3

Mft. Mist dose one, send 4 such every three hour for 4 doses only.

R Q. Salicylas	...	gr. 3
Caffeine Citras	...	gr. 3
Sodi Bicarb	...	gr. 10
Sodium Cinamate	...	gr. 2

Mft-Pulv. Dose one send 4 such, one dose 1/4 an hour after the mixture, 4 times daily.

The patient recovered but she took comparatively longer time to come round. It was probably a case of Influenzal type of malarial origin.

23. J. C. S., age 58, had a high temperature controlled with Q. B. H.; loss of memory. Blood could not be examined. He was quite alright before the fever. There is still some loss of memory. This is probably a case of amnesia of Malarial origin.

Conclusion.—Above, I have given synopses of some of the Atypical cases of Malaria. In the majority of them blood examination was positive and in the rest the therapeutic test cleared the diagnosis. Malignant Malaria remains in obscure form, produces vague symptoms and deceives the average medical practitioners. Their frequency of occurrence is greater than what is generally conceived of. Hyper-pyrexial and convulsive types are very common. Next in frequency are Algide and Dysenteric types. Then come the Hemolytic and Meningeal types. Other varieties are not so common.

I repeat again, with apology that in a malarial place in the so-called season of malaria, every acute case with or without fever showing vague pernicious symptoms and having no definite

localised lesion in any system should be considered as Malignant Malaria. Blood if possible should be properly examined and Q. B. H. should immediately be injected intra-muscularly. One must not be misled by the applauded therapeutic value of the new comers—plasmoquine and atebrin in these cases. Independently they are no good in malarial vagaries but undoubtedly are valuable adjuvants.

Latent and Masked Malaria

BY

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(A) Latent Malarial Infection

LATENT Malarial infection is indicated by a very slight disturbance of health every day or every other day at a particular time with clock-like regularity; or, only at a particular season every year, or rarely every fortnight, but giving rise to no very distressing symptoms or affecting the ordinary duties of the patient. Repeated examinations of the blood will show few malarial parasites perhaps, but administration of inadequate dose of quinine invariably brings on an onset of malaise, rigor and fever and the parasites are found in the blood in numbers.

(The writer has practised in a malarial district for over 25 years. In spite of vigorous health, he fell into the clutches of this infection in the sixth year of his practice and recovered after a hard struggle of 3 months. But from that time till lately, that is for over twenty years, he had the latent symptoms in him almost every day. He would have to wear a warm stocking and wrap himself up warmly often at 4 in the afternoon. Sometimes during the rainy days there appeared regular rigor and the temperature rose to 99° or 100°, but he would be alright the next morning and attended to his food and duties. Some of his colleagues used to partake of their usual rich dishes even during these exacerbations of fever and never repented for the same! On a change of climate or during heavy engagements, these symptoms were never felt.

Many of the I. M. S. officers have written similarly in

magazines. Even after retiring and living in England they have not escaped from such slight malaise and feverish feelings at a particular time every day. A friend of the writer working as a Judge in a native State, gets such feverish feeling every year, only after the rainy season and as long as he abstains from taking adequate dose of quinine, he goes on suffering every day).

Exceptionally, the symptoms take the shape of *periodical gastro-intestinal disturbances* attended with severe gastralgia or vomiting. And quinine injections give immediate relief. An L. M. P. colleague of mine had such attacks twice every year for 3 years and we found out the remedy during his third attack. After the sixth relapse, quino-plasmoquine was systematically administered to him and for the last 3 years he has not got any further attack.

Migraine and Headache;—I have the record of a Moslem lady of Panitar who used to suffer from a severe form of migraine every fall lasting for 3 months. Finding the severity of her symptoms occurring at a fixed hour every day, I prescribed Whitla's recipe,—Antipyrine, caffeine citras and quinine hydrobrom with quick and permanent relief. Next year she came to my clinic exactly during the rains and I cured her with 3 injections of quinine. Some periodic headaches I have similarly cured with anti-malarial remedies.

Neuralgias or neuritis, especially of the legs, coming regularly every evening, or on going to bed, usually find specific remedy in quinine.

Many "Brow-ague" and "Sun-headache" cases, in which periodicity is marked and the pain increases as the sun rises and abates with its setting, are strikingly relieved by the above Whitla's recipe, which contains 4 grains of quinine per dose.

Solis Cohen found the following latent forms of malarial infection in Panama:—

- (1) Sensation of cold in the lower limbs and back; these feelings are commonly complained of in Lower Bengal.
- (2) Oedema of the face and extremities.
- (3) Trigeminal or intercostal neuralgias.
- (4) Peripheral neuritis with paraplegia of the flexor variety.
- (5) Attacks of dizziness and
- (6) Pain in the epigastrium with or without periodic vomiting or enlargement of the spleen and liver; usually seen in

malarious Bengal. Recovery commonly follows the use of a cholagogue followed by small doses of quinine for a few weeks.

I have come across some cases who primarily hailed from Bengal and settled at Bihar and who, after a few years, began to suffer from daily attacks of dizziness and loss of vision at a particular time which baffled diagnosis but which were ultimately cured with quinine and arsenic.

Combined Infection.—It is a common knowledge that in a malarious country, typhoid fever, pneumonia and other diseases including even cholera, apoplexy etc. are commonly combined with plasmodium infection. That is to say most persons living in the endemic area harbour M. P. latently in their system, and when resistance fails, the body gets otherwise infected and weak, the M. Ps. join hands with the concomitant infection, and augment all troubles, especially the fever. (Ref. C.M.J. Jan. 1932—"Apoplexy complicated with Malaria". "Antiseptic" No. 33, "Cholera Asiatica;" "C. M. J. 32: Notes on Typhoid Fever).

Diagnosis of Latent Malaria rests mainly upon these two points.—

- (1) Periodicity in the attack; and
- (2) Examination of the blood usually fails to detect any parasites; but small doses of quinine administration let loose crescent forming M. Ps. and recovery taking place after atebirin and quino plasmoquine.

(B.) Masked infection of Malaria

Here the symptoms are misleading and atypical; symptoms of some other diseases completely mask the malarial origin of the case.

(The writer divides Masked Malaria into two forms: the simple and the pernicious. In most of such cases, symptoms point to some other diseases and the diagnosis rests upon keen observation, experience and blood examination. Solis-Cohen and others regard masked malaria as only those cases that are obscured by the symptom-complex of a concurrent ailment, usually another ailment.)

I. Simpler manifestations of masked malaria.—*Hiccough*, acute and chronic,—with periodicity and when uncomplicated, finds a remedy in quinine injections.

(K. C. of Kerangachi, aged 70 years, was suffering from severe hiccough for 3 weeks in 1915. I reached the place in

the evening and learnt that the violence was then abating and by 9 P.M. the patient would be comparatively quiet and remain well the whole night. But from 8 or 9 in the morning, the frequency as well as the severity of the hiccough would go on increasing and by 2 or 3 P.M., the old gentleman would be almost moribund. Nothing had the slightest effect in reducing his hiccough and the patient was rapidly sinking. The writer injected quinine at once and repeated the same next morning. The old man kept him waiting till the afternoon and feeling no return of the terrible demon allowed him to return home.)

Many *Myalgias*, *Neuralgias*, *Neuritis*, *Headache*, *Migraine*, etc in malarious districts have their origin in plasmodium infections.

Urticarias flaring up at definite periodical intervals are often amenable to quinine preceded by some cholagogue.

Some *Gastritis*, *Enteritis*, *Appendicitis* and even *Orchitis* have been found out to be malarial in origin. (*vide I. M. G. May 1921; Orchitis*)

Eye Lesions:—Dr. Manson has mentioned Dendritic ulceration of the cornea, Intracellular haemorrhage, and various affections of the Motor nerve of the eye caused by plasmodium. Dr. Pareira has written about Haemorrhagic neuritis; Dr. Omak, about Silver bands and Opacities; Dr. Kirwan, about Cataracts and others, of Optic neuritis. (*See I.M.G. December, 1928, Medical Review 1904*).

Pleuritis, *Peritonitis* and *Nephritis* have been mentioned by Manson as malarial in origin and as having been cured by him with quinine.

Renal and Biliary Colic have been known to be caused by plasmodiums,—*Vide Medical Review, 1907. Herpes (I. M. G. December 1932) and Dysphagia (I. M. G. January 1928)* had been mentioned.

The writer has seen some *Hysteric* manifestations proceeding from malarial infection. One case of *Insanity* was cured by him with the administration of quinine and arsenic. In this patient, there was a very definite periodic rise of temperature with simultaneous increase in violence on the part of the insane.

Fits in the course of Pregnancy was once traced by the writer to be regularly following slight rise of temperature which had escaped the notice of the attending doctors and quinine cured both the fever and the fits.

(There are many other masked manifestations of Malarial infection which can be profitably collected from the Medical Magazines. The writer requests some active members of the profession to undertake the task.)

II. Pernicious Manifestation of Masked Malaria.—(It will be noticed that in all the following grave signs and symptoms, the disease is seldom diagnosed rightly at the first visit of the doctor, that some other grave disease is first suspected and that the underlying nature of the cases only ascertained after ruling out differential signs and after blood examination.)

Pernicious attacks of malaria are usually classified into *Cerebral* and *Algid*. The *Cerebral* are again divided into *Hyperpyrexial*, *Comatose*, *Convulsive*, *Paretic* and so forth. The *Algid*, into *Syncopal*, *Choleric*, *Haemorrhagic*, etc.

Hyperpyrexia:—(Some families are noted for high rise of temperature especially amongst their children. My friend Mr. Huq's children invariably got 106° and 107° temp. during the early winter months almost every year. Only once, I had some anxious hours about a small girl and when the nature of the affection was known, we managed to put in some quinine intramuscularly at the beginning of the rise of temperature and had the satisfaction of checking the disease at the onset. The character of the fever had been a continuous rise without congestive signs or delirium or pain in the chest or abdomen. No cough, no other complaints, but simple and continuous rise of temperature were the characteristics. And so long as quinine was not applied, the fever left them not, but only showed slight morning remissions one or two degrees only. Homeopathy, Kaviraji, Unani,—all had to lower their banners before quinine, which checked the fever invariably at the shortest periods.

Sometimes the temperature remains within bounds for 2 or 3 days and then suddenly shoots up to 107° or even higher. Unless active quininisation is undertaken from the first day, these cases generally prove fatal once the temperature goes above 106°: Mr. A. S., Inspector of Police's only son had a remittent type of fever for 3 or 4 days and before we could realize the nature of the disease by blood examination, the temperature began to rise and shot up to 109° and the boy expired. Blood revealed numerous plasmodium falciparum.)

Convulsions.—(In 1933, I reported two cases of Apoplexy which were diagnosed by me as malarial in origin and treated

them with injections of quinine with success. Both were middle aged peasants above 45 and both had hemiplegic paralysis. The characteristic signs which pointed towards plasmodium were: normal blood pressure; periodicity in the convulsive seizures beginning with the onset of fever and declining with the lessening of the temperature; even the paretic limbs showed movement during defervescence; also, the patients showed slight reawakening of sense early in the morning, when the temperature reached 97° . The cases were seen at remote villages and as urgent steps had to be taken, quinine was injected immediately and then blood was taken for examination. Both the cases showed estivo-autumnal parasites).

Type of convulsion may take either of tetanic or epileptic nature. Such convulsions are common in children. But when these occur in elderly people without a previous history of rigor or fever, the case misleads even experienced men. And the symptoms look so urgent, the patient in an unconscious state, with perhaps stertorous breathing, and eclamptic fits every 10 to 15 minutes with paralysis of half side of the body, immediate steps are required to be taken and some definite diagnosis expected by the attending physician and the family.

Meningitis.—Symptoms simulating cerebro-spinal meningitis, —cases that are designated as *Cerebral Malaria*, are seen every year at odd times mostly among robust, full grown, young men and women. Many Mofussil doctors believe that true Cerebro-spinal meningitis never occurs in the villages of Bengal and therefore they invariably inject quinine at the onset of acute symptoms before calling any consultant, possibly to prove their up-to-date-ness. But I have drawn pus and blood from lumbar punctures in many cases of meningitis and assert that true meningitis cerebro-lis is found even in the villages. It may be that malarial meningitis predominates in the malarious districts and I believe that intramuscular quinine does no harm even in real meningitis cases.

Close inspection in these cases will give a clue to the real nature of the affection. Kernig's sign is almost always absent in the malarial cases; periodicity regarding the fits and fever are present; history of malarial attacks every year at the time is sometimes elicited; and blood examination may show the plasmodiums.

Coma.—High fever, delirium and coma may come in quite suddenly or gradually but the onset is so abrupt and unlike the

usual slow distinctive malarial nature that the diagnosis becomes difficult even in an infective zone. In cases of children, the diagnosis of "Worms" and in the elderly people "poisoning" have been often encountered. The writer saw two brothers attacked in exactly similar manner within 10 days of each other, coma setting in, on the third day of the fever. Both recovered from injections of atabrin followed by its oral administration with quino-plasmo-quine. (The family has a terrible dread against quinine injection.) In some cases at the comatose stage, temperature drops down to subnormal; but the rectal temperature will be found much higher. Blood examination for M.P. is a great help in these cases.

Paralysis with aphasia following slight rise of temperature without unconsciousness has been encountered by the writer once only where the diagnosis was established by systematic blood examination on the ground of a suspicion of syphilitic taint. Numerous M.P.s. were detected and quinine promptly removed the paralysis.

Dementia, Delusional Insanity and Psychical disturbances have been traced to be malarial in origin by Manson and others. These various cerebral manifestations are explained by embolism of cerebral capillaries from clusters of M.P. actually demonstrated during post-mortem examinations.

Algid forms of Malaria.—Algid forms of the pernicious type are so-called on account of the entire absence of the first two stages of true malaria, the rigor and heat, and the presence of only the cold stage, characterized by collapse and syncope. Hence the true origin of the cases are obscured and the main symptoms point to cholera, acute dysentery, gastric crisis and similar other affections.

(The writer has seen in some families, malarial fever is invariably followed by a grave sweating stage, when the temperature goes down to 95° , pulse to 60, the whole body perspires terribly, resulting in cold extremities and extreme weakness. Administration of quinine is often held responsible for this condition; but when similar symptoms appear during the next attack or in other members of the family without the use of quinine, then the real cause is admitted to be M.P. Once the true nature is known, alarm gives place to assurance.

Hyperpyrexia does not necessarily react to a subnormal temperature, although every doctor expects such a sequence. Hyperpyrexial deaths usually take place when the body is still warm.)

Typical algid malarial fever ushers in with slight rigor which is followed by signs of collapse, nausea, one or two vomits and a quick heart. The condition is usually ascribed to some grave indiscretion of diet and symptomatic treatment is undertaken. Possibly this gives temporary relief. But when in the next morning, the same symptoms return with greater virulence, then the consultant comes and discusses about the previous history, palpates the spleen, looks for periodicity, takes the blood for examination and perhaps puts in quinine into the muscle in anticipation. (I have not read anywhere about this type of Algid Malaria.)

Manson mentions a *Gastric* form which is accompanied by severe epigastric distress, tender retracted abdomen and incessant vomiting. The vomited matter may contain blood. (Possibly it is the graver type of the form mentioned above.)

Choleric Form.—(In 1923, I saw a case in a well-to-do farmer's house at Hasnabad with the following history: the present patient's elder brother had died of cholera only six days before, after suffering for 2½ days. His disease was ushered in at about 1 p.m. after meals with shivering, vomiting and purging. The colour of the stool was at first biliary, then brownish and afterwards watery. Cramps and coldness of the body supervened at midnight. In the next morning he was almost free from any symptoms and took ground-rice-water. But soon a relapse of vomiting and purging took place and the doctor administered Salines. Cramps disappeared, some reaction took place but he succumbed in the morning. My patient had been attacked the previous day at about 10 a.m. with shivering, vomiting and purging which continued up till midnight with vigour and frequency. Homeopathic remedies had been applied and he felt better in the morning. The sad experience of the previous case had compelled the father to call me and when I called at about 9 a.m., the patient seemed to be none the worse for the previous day's suffering. But within an hour he looked pale, shivered and passed a copious watery stool. He then began to vomit and within an hour was pulseless and cold. I administered one pint of hypertonic saline and then followed this with quinine and 50 c.c. sugar solution. Reaction took place soon. In the afternoon another glucose was given and quinine injected intramuscularly. Next morning he received both glucose and quinine and recovered without any trouble. The family agreed to take quinine for prophylaxis. There had been no

case in the village prior to these 2 cases, neither there broke out any other case afterwards. I forgot to mention that the rectal temperature of the case had been 102° when he was shivering before me.)

Dysenteric and *Haemorrhagic* forms are very deceptive in character and are seldom diagnosed in the mofussil. Most village doctors are in the habit of prescribing quinine along with emetine or with antidysenteric serum. The treatment is especially difficult in children. Haemorrhagic cases, especially when bleeding takes place within some organ or in the brain or spinal cord are seldom rightly diagnosed.

Malarial Cachexia is seldom met with now-a-days. Old doctors of the previous century will remember the cachetic look of chronic malarial cases, with big spleen perhaps covering the whole of the thin abdominal cavity, cancrum oris threatening, with signs of blisters and burns all over the spleen. With the advent of village doctors armed with quinine and a hypodermic syringe, such cases have become very rare. In its place we see acute Black Water fever cases, typical *Acute Haemolytic Malaria*. It is a curious fact that in olden days, we did not meet with the type of Blackwater cases which began to crop only when the villagefolk had lost the dread about quinine administration and were freely using Post Office quinine packets. We had lots of Cachexias, but those were chronic cases and unlike the present day Haemoglobinuric Fever. Hence the writer believes that besides *P. falciparum*, there are other conditions which bring about an acute attack of Haemolysis,—namely irregular half-hearted use of quinine and some other unknown factors.

"Transmission of Malaria in Blood Transfusion from a donor, who had served as a soldier in India for six years, but who never had any attack of malarial fever. But after his return to England he had a short series of shivering attacks each lasting 2 or 3 days; malarial parasites could not be found in his blood," This is a case of *Latent Malaria*. (*C. M. J.* Nov. 1936).

This number also contains "Two Atypical manifestations of Malaria" which are easily mistaken for T. B., or T. S. or Hepatitis.

The following other rare Latent and Masked cases I have collected from the Medical Journals:—

(1) "Cerebeller Incoordination".—*I. M. G.*, April, 1922,

- (2) "Afebrile Cerebral Malaria and Spastic Paraplegia"—pp. 724 and 140 of 1927, *I. M. G.*
- (3) "Pleurisy and Peritonitis with Ascitis"—MANSON.
- (4) "Renal and Biliary Colic, Nephritis, Cholecystitis."—*Medical Review*, 1907.
- (5) "Herpes"—*I. M. G.*, Dec. 1932, and "Dysphagia"—*I. M. G.*, 1928, p. 21.

Malaria and its Prevention*

BY

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BEFORE I begin the subject of my discourse, I shall deal with, as a sort of introduction, an allied subject, kala-azar and show how it has been eradicated and what part the medical profession of Bengal and Assam played in it.

Long before the discovery of Leishman—Donovan bodies, a disease known as Aleppo or Delhi boil was discovered in which double nucleated bodies were found in clusters inside cells and these were then described as Wright's bodies. Towards the end of the last century, Leishman found in Netley Hospital, similar double nucleated bodies in clusters inside the endothelial cells in spleen puncture smears taken from a soldier hailing from Dum Dum, who got admitted into the Hospital for treatment of fever and enlarged spleen, the nature of which was not known then, it being called malarial cachexia. Six months later, Donovan found similar bodies on spleen puncture of cases of fever of unknown nature but the cause of which he first associated with these bodies. Bentley, working in Assam, found similar bodies in cases which were then definitely known as the deadly kala-azar of Assam, which bodies he saw was the cause of the disease. No one knew at that time what they were. These bodies were first cultured artificially in citrate solution in the Bacteriological Laboratory of the Medical College, Calcutta, and proved to belong to the big class of Herpetomonas. A few months later, Dr. R. Row in Bombay, cultured the parasite of Delhi Boil, and very soon, Nichols working in South of France, found the close relationship

* A Lecture delivered at Shillong before the Medical Association on the 3rd November, 1936.

of these with a disease known as infantile Leishmaniasis, occurring in children which was cultivated in solid N. N. N. medium. Later on the three parasites were named respectively Leishmaniasis Tropical, Leishmaniasis Donovanii and Leishmaniasis Infantum.

A worker in Brazil, named Viviani, found Tartar Emetic effective against a disease allied to Delhi Boil but occurring in nasal mucous membrane. Soon Caraini in Italy found it effective in Leishmaniasis Infantum.

Rogers in Calcutta and Muir in Kalna (Burdwan) soon started using the drug with marked effect in real kala-azar cases. The Central Co-operative Anti-Malaria Society Limited, of which I have the honour to be the Honorary Secretary, started treatment centres, which later on were known as kala-azar centres, in Baraset subdivision (24 Perganas) and elsewhere, where thousands of kala-azar cases were found out after the starting of the centres. These centres had the peculiarity of their own differing from the other treatment centres, in that they were not located in special buildings known as Charitable dispensaries, but very often in the verandahs of dak-bungalows or sheds of Railway Stations and very often underneath shades of trees. The Central Anti-Malaria Society sent out sterilised (autoclaved) 4% solutions of scale preparations of Sodium Antimony Tartrate in phials, each of which containing 4 or 5 doses and each phial costing not more than one anna including the cost of sterilization. Basketfuls of these antimony phials were sent out free by the Central Society to the various centres. The number of patients at the centres went on increasing from 10 or 15 to as much as 700. Qualified medical men working as volunteers at these centres not being able to cope with the huge rush of patients at all the centres, notices were posted at the Medical College and schools inviting senior medical students to join hands, the Central Society paying travelling expenses for all the medical volunteers. A large number of medical men and senior medical students who worked at these centres got an idea for the first time as to what is known as field work in preventive medicine; the experience they gained there, was utilised later on when they had to do similar work while in service and Assam owes its salvation to many of these doctors such as Capt. S. N. Dutta, of the Public Health Department whom I can recognise as one of our late volunteers here.

Very soon we discovered that a surprise was in store for us while working at these centres. When we started these centres they were meant for treatment alone with a philanthropic motive and not for prevention of the disease for which we depended on the

research work that was being carried on assiduously in laboratories to find out the vector and the mammalian reservoir, the sandfly, the hamsters and the like. The surprise consisted in this that in each of the centres, after a rapid increase in the number of patients, there was a diminution of cases, there being a sudden drop of cases after 6 months and that abruptly and quickly. As no accidents occurred in any of our centres, the cause of unpopularity of the centres could not be put forth to explain this. Soon we guessed, which ultimately was proved to be true, that antimony salt was acting as a sterilisans magna in Ehrlich's sense, but much more effectively than he got by his Salvarsan in Syphilis. Infection among the villagers diminished rapidly till village after village was perfectly free from kala-azar, which we were able to verify by personal visits to places accessible from the centres, and thus we outstripped the laboratory workers.

As similar centres have since been started all over Bengal by Local Bodies and Government as well as by our workers, where similar results were observed, the laboratory workers who were then engaged in finding out the vector, were left stranded, as they could not find any case for their research work which had to be stopped in disgust. Now, in this work, the medical profession of Bengal and Assam take a justifiable pride.

Just think of the condition prevailing in Nowgong in Assam in those days about which Mr. K. C. De., C.I.E., I.C.S., told me one day. He was going on an elephant through Nowgong for some inspection work, when a storm overtook the party and they had to take shelter in one of the villages where they were horror stricken to see the skeletons of human beings in those houses who died of kala-azar long ago, not a single living man being found in the village. This work of the medical profession has not been appreciated by the community. Those who had to do this work are starving now.

Now, leaving the subject of kala-azar, can any one do similar work in the case of malaria also, which is much less a deadly disease and about which everything is known. Trial at "sterilisans magna" with intravenous injections of gram doses of quinine every day for two weeks made in Macedonia and elsewhere during the War did not give the desired result.

The recently much talked about preparations of plasmochin and atabrin, though remarkable for their effects could not do it, for none of them could stop the relapses brought about by the asexual cycle; so fresh crops of sexual forms are being formed

from the asexual ones; and the vector being there, the epidemic will go on.

The field experiment made on a large scale in Memari Thana (in Burdwan) by the Public Health Department, Bengal, has given results much inferior to what anti-larval measures do, there being only reduction and no eradication of malaria and that at an enormous expense.

I, as Secretary of the Central Co-operative Anti-Malaria Society Ltd., which has been in touch with thousands of anti-malaria societies in the villages of Bengal which are carrying on antimalaria work in their respective areas, can vouch for the truth of the assertion that malaria incidence has been brought down to zero in many hundreds of the villages and that it has been done by anti-larval measures combined with Quinine.

Before I talk about the efficiency and choice of anti-larval measures and about what we are doing in Bengal, a survey of what other people are doing in other parts of the world, will not be out of place here.

In Europe, (southern portion), the vector *A. maculopennis* breed in streams with strong currents in contrast to U. S. A. where *A. quadrumaculatus* developing in stagnant pools have given difficulty. Similar is the experience in hilly tracts of Bengal viz., Jalpiaguri Duare and also in Assam where *A. minimus* breeding in streams with strong currents have added to the troubles. So, here, if these hill streams become covered over with thick overgrowth as found by malariologists here, no larva develops. As soon as these overgrowths are removed, the incidence of malaria increases due to indiscriminate breeding of *A. minimus* larvae in these cleared streams. In the plains of Bengal, mosquitoes never breed in current streams, so the measures that have been advocated in Assam will not do. Advocacy of the measures adopted here will result in the growth of dense jungles, stopping the struggling water courses as in Bengal. We depend mostly on the biological control larvivorous fish which abound in all rivers, and not by the use of chemicals. Fish will get no access to the larvae if the water of the rivers is covered by dense aquatic vegetation.

There are several species of fish in the tanks and rivers e.g. *Panchax panchax*, *Haplochilus panchax*, *Amblypharingodon mola*, *Barbus ticto* etc. of Bengal which have been brought to the notice of the authorities by us in Bengal. It is gratifying to note that the present Director of Public Health, Bengal, has circularised the local bodies to use fish as much as possible, for anti-larval measures and no less gratifying is the fact that the Malaria Survey

of India under Col. Shinton is bringing out a revised edition of their brochure on "Larvivoracious Fish of India," which is not yet out.

I cannot help bringing to your notice, the progress in the knowledge of malaria, which has been made in recent years by Malariologists all over the world. You know Malaria parasite has not been cultivated on a scale like the kala-azar parasite, but in a way they have done it. Bass's method is not like N. N. N. media culture. The induction of malaria fever in paralytic cases being found beneficial, particular strains of malaria parasites have been kept alive by allowing the mosquitoes bred in the laboratory to bite an infected man and then passing it to the paralytic case and then they get another group of mosquitoes to bite them and so on. In this way strains have been kept alive indefinitely. For this an insectary has to be devised for breeding the vector—in the case of Europe it is *A. maculopennis*. Now, for inducing the malaria fever in paralytic cases, it is necessary that it should be always under control and should always be successful. In doing out this research, research workers have found particular strains of *P. vivax* named Maccoy strain which differs from other strains of *P. vivax*. It has the above qualification. It can be kept indefinitely. Another strain of *P. vivax* has been isolated, which has a more marked malignant effect than the ordinary plasmodium *vivax*. Soon they have found out certain facts about immunology of malaria which was not known before. They found the Negroes are not infected by *P. vivax*; so, in the case of Negro patients *P. falciparum* had to be used; if used in white paralytics, they being not immune to any one of these parasites become dangerously ill.

Another thing found in culturing *A. maculopennis* in insectaria is that if they are habituated to bite cattle, the number of teeth in the maxilla increases which makes them incapable of biting man. This is what is called zoophilism and so the more the cattle are stall fed, the more zoophilic are these mosquitoes. This was first noticed by Angello Celli viz. that there can be extensive anophelism without malaria.

I leave aside the intense propaganda work which the Central Anti Malaria Society is carrying on to help the Irrigation Department to remove the marginal embankments specially along the tidal creeks to prevent the free flow of tides and thus prevent the increase of man-made malaria which is going on apace throughout the whole of the coastal region of Bengal as much as possible by which the upland waters of sweet water rivers can find easy access to the

sea and prevent malaria and devastating floods now occurring everywhere.

Leaving aside the non-tidal portions we will have to say something about the tidal portions, on which depend the prosperity and well being of a good portion of Bengal, depending as it is entirely on the maintenance of the proper regime of the rivers, as all non-tidal rivers finding exit into the sea, through these tidal creeks. It is necessary to state that to prevent flooding of the homestead as well as cultivated lands of the people which is likely to take place during the rainy season in the non-tidal regions, marginal bunds have been put in with consequent disorganisation of the river, by this, the silt carried by river is deposited in the bed of the river and not in the surrounding lands, and so the rivers get silted greatly. In the tidal regions the swollen condition of the water courses taking place twice during 24 hours of the day, which become increased during equinoxial spring tides, the people in certain coastal regions, but not everywhere, resort to putting in extensive marginal bunds. This has the effect of silting up much more quickly than the rivers of non-tidal regions, of the tidal creeks. It does not require an imagination of a very high order to make out, that not only the coastal areas will be waterlogged, but all the sweet water rivers, will empty later on into a huge marsh, on the border line at the junction of the non-tidal and tidal rivers, as happened in Italy in the case of the river PO at its outlet into the Mediterranean and the British Guiana where huge pumps have to be put up to pump out the water coming down from uplands through rivers into the sea—thus lessening a bit but not doing away with extensive anopheles breeding places, as has been observed by Hackett recently in Italy, where he found larva of anopheles growing so much that can be collected in bucketfuls in this region. This catastrophe has been prophesied by Addams Williams, in his book "Rivers of Gangetic Delta," in 1919, and this is happening on an extensive scale everywhere in Midnapore, Howrah, 24 Perganas and Eastern portion of Khulna and Baggergunge. The danger foreseen by Mr. Addams Williams due to wrong system in force, quarter of a century ago but which he could not remedy, due to extensive vested interest, is however being strenuously tried to be averted by the present officers of the Irrigation Department, though ineffectually, on account of the higher authorities not having realised the true position of affairs. So the Central Society has joined hands with this department, in mitigating as much as it lies in the power of this small organisation, this misery, by making known the true state of

affairs to the people whose vested interest is standing in the way of restoration of rivers regime to give up their rights. We advise them not to keep any homestead or cultivated lands in the spills of water courses, and to utilise them if possible for fisheries. So the bunds can be done away with.

Now, I shall finish my discourse with the advice which I am going to give, one month hence to the workers of the antimalaria societies, not necessarily medical men, who are laboriously working through evil and bad report, unmindful of the carping criticism of those who are better placed in cities, who care a bit for their ancestral dwelling houses in villages by a quotation from Carlyle's "Past and Present" and which I give to you also. "My friends, all speech and rumour are short lived, foolish, untrue. Genuine work alone which workest faithfully that is eternal and as the Almighty Founder and World builder Himself; stand by that; and let the Fame and the rest of it go prating.

" Heard are the voices
Heard are the sages
The world and the ages
Choose well, your choice is
Brief and yet endless
Here eyes do reward you
In eternity's stillness
Here is all fullness
To reward you
Work and despair not."—*Goethe*.

Oral Lesions Observed During the Epidemic of Malaria in Ceylon
in 1934-1935. By Dr. W. Balendra.



Stomatitis. (In Malaria.)



Profuse haemorrhage. (Case proving fatal.)

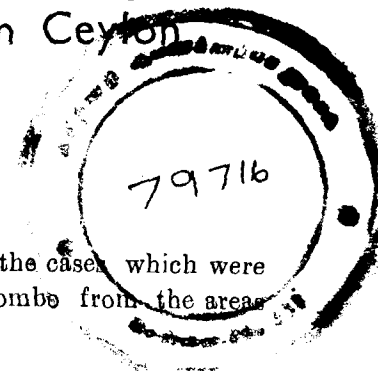
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Oral Lesions Observed During the Epidemic of Malaria in Ceylon in 1934-1935.

BY

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Colombo.



THIS short article attempts to describe the cases which were admitted to the Dental Institute, Colombo from the areas which were afflicted with malaria.

These may be divided into:—

- (1) Stomatitis—as is observed in any febrile condition.
- (2) Gingivitis and developing later into a chronic condition and presenting all symptoms of paradentosis.
- (3) Gingival Haemorrhage—varying from slight trickling of blood to profuse haemorrhage (one case proving fatal).

The inflammation of the mouth was frequently observed in patients having a high temperature due to malaria. This being common in any febrile condition, it is outside the scope of this article.

The inflammation of the gums, without an extensive area of inflammation of the mouth, was observed in many patients whose gingivae were normal before the onset of the febrile attack. The gums were swollen and red and painful to touch. The patients described that they felt the teeth to be loose in the sockets. Injection of quinine mixtures accentuated this condition—the quinine solution being definitely an irritant to the gingival tissues when it was taken for long periods.

Cases have been noticed of the teeth becoming loose and young patients complained that all their teeth became loose within a period of 12 months after the onset of the fever. In several cases complete extractions of teeth were performed in patients between 30 and 35 years of age within 2 years of the malaria infection.

Gingival haemorrhage in varying degrees of severity were observed in patients from ages varying from 5 to 60. In children the gingival haemorrhage was profuse during convalescence after a rigor.

The most severe case occurred in a man of 61 years of age. He was a chronic malaria subject. The parasites were found in the blood film. The fever subsided after an acute attack but a profuse

haemorrhage commenced from the gums. The haemorrhage continued for 3 weeks. Local packing with alum, tannic acid, Tr. Feri Petchlor was tried. Haemoplastin was given frequently but in spite of all attempts to stop the haemorrhage, he literally bled to death from the gums.

This short article is written to draw the attention of the medical practitioners that dental treatment is necessary in cases of malaria if the general health of the patient is to be maintained at the highest possible level, and I suggest that quinine mixtures should be taken by a small funnel into the gullet if the mixture is taken for long periods.

Treatment of Black Water Fever

BY

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THE question of the practical and effective treatment of Black Water Fever has become a great puzzle to the average medical practitioners, specially amongst the mofussil ones where intelligent attendants and nurses are not available and the co-operation of patients and their relations is very restricted. The incidence of the disease was almost a rarity in this part of the country, and was limited to Dooars, Assam and Bengal in India. However, with the increase in the malignant tertian infection, B. W. F. cases are almost prevalent in every part of the country, and the question of effective treatment has become an important problem.

Without making much academical discussion, I like to sketch the line of treatment alone which has been found very effective in my hands, and to point out that the percentage of cure under this line of treatment is amazingly high.

To understand the line of treatment, a brief description of changes is essential. It is a pernicious type of malaria, and should no longer be considered as a separate disease. This is evident from the following facts:—

- (1) Prophylaxis of malaria is prophylaxis of Black Water fever.
- (2) Black Water fever is only prevalent where malignant tertian infection is at work.

- (3) *Plasmodia falcipara* have been found in most of the cases.

ROGERS and MEGAW.—“Malaria parasites are present in 75 to 90 per cent of the cases in which the blood has been examined before the onset, and though they disappear rapidly in most cases during the attack, they usually reappear within a week to a fortnight after the acute stage of the disease is over. The parasites are usually malignant tertian, but cases have occurred in persons infected with benign tertian, and there have been a few cases in which only quartan parasites were found”. These are cases of mixed infection.

KNOWLES states that malarial parasites were found in blood films from patients in 73 per cent of cases examined on the day before the onset of Black Water fever; in 47.5 per cent on the day of onset of the fever; and in 23 per cent on the day after onset of the fever. They are soon destroyed in the general process of Haemolysis.

- (4) The clinical symptoms are almost similar to the infection with malignant tertian parasites plus the result of haemolysis.
- (5) The process of haemolysis is same but in Black Water fever the haemolysis is greater and the process is more rapid.
- (6) The changes associated with homoclytic shock are almost similar in both the diseases.
- (7) The treatment is also the same and the same drugs are used for both the diseases plus the treatment of haemolysis in so-called Black Water fever.

It should be properly termed as hæmolytic type of malignant malaria, as hæmolysis is the main feature and hæmoglobinuria is the result of the same. Megaw and Rogers in their recent edition of Tropical Medicine has termed it hæmoglobinuric malaria. It is however, nothing but a virulent infection with malignant tertian parasites to susceptible persons where hæmolysis is favoured with some unknown condition which is still to be worked out.

The infection is a heavy one, hæmolysis is great and generally straining the renal vessels, red blood corpuscles are great

destroyed by parasites themselves. The Kupfers cells of the reticulo-endothelial system, specially of the liver cannot cope with hæmolysis which is usually severe, bile pigments are absorbed, early jaundice ensues and Urobilin with hæmoglobin appear in urine. The Hæmopoëtic system cannot make up the loss and the patient in consequence becomes weak, anæmic and jaundiced, spleen and liver are also enlarged.

The products of Hæmolysis are excreted through kidney which becomes congested and enlarged, tubules are almost blocked with hæmoglobin infarcts, urine becomes black, acid and scanty, anæmia may follow and death may result from Uræmia etc.

Untoward symptoms and results are hyperpyrexia, gradually increasing jaundice and anæmia, persistent vomiting and hicough, anuria and uræmia, marked prostration, collapse and death.

During the process of hæmolysis, the patient gets all the symptoms of protein shock viz—rigor, high fever etc, and it should be noted that all cases in my group numbering three and twenty only were comparatively healthier ones in their families and their ages varied between three and thirty six years, majority being young adults and were of fairer complexion. The ratio between female and male is 1:4 approximately.

The Treatment Proper.—1. Absolute rest in bed to maintain the heart and to avoid further strain, as the patient becomes rapidly weak and anaemic.

2. The patient should be warmly covered to prevent any chill from outside and given warmth application to loins particularly. I have seen cases to shiver during hæmolysis which is actually encouraged by rigors.

3. Plenty of fluid should be given orally or by any route. Milk of green cocoanut where available is an excellent drink and seems to have some special action. It is an excellent diuretic and sedative to the gastric mucosa. It is said to be anti-hæmolytic on account of its calcium content. The fruit should be fresh and the milk must not be rancid. It should better be tested by litmus paper before administration.

Sufficient quantity of barley water. The drink should preferably be iced. Rectal saline should be given every three hours from the onset with Sodi Bicarb, glucose and adrenalin. Later on, if required Saline should be given subcutaneously. Intravenous Saline has also been recommended but personally I am not in favour of the same in the acute stage, as the reaction after the Saline is not at all desirable on account of the rigor and

high temperature which may encourage further hæmolysis. If the rectal route is utilised from the beginning, I am confident, subcutaneous or intravenous salines will never be required. The urine will remain free and the question of suppression will rarely arise. Salines should be used warm, and their temperatures should be controlled according to the temperature of the patient as in Cholera, but on no account, should they be given cold.

4. The blood coagulability, alkanity and its Ph. are reduced, and the urine is markedly acid. The free use of Alkali is, therefore, essential. Sodi Bicarb and glucose drink is also helpful.

5. Heart should be maintained by rest, liquid, nourishing diet and stimulants. The following drugs are of chief value. Adrenalin, Spritus Ammon Aromaticus, Spiritus Aetheris. Glucose, Makaradhawaj perferably with Calcium Lactate and Caffeine, and Caffeine Sodi Benzoas. The last named drug is given by injection, and it will also help in diuresis.

6. *Plasmoquine*:—It is given orally and by injection if required, that is if the temperature is persistently high and the number of hæmoglobinuric urine is more than three. It should be mentioned that its use has not been advocated by some on account of the consequent Cyanosis, which untoward symptom is actually avoided if Sodium Bicarbonate and Adrenalin are used from the onset along with the use of the drug. Rogers and Megaw in their treatment of Black Water Fever state:—"Pasmaquine was regarded at first as being safer than Quinine in this respect but recent reports indicate that reverse is the case". But all my cases though the number is limited, not only improved but were cured under the treatment of Plasmoquine. One case alone developed undesirable dysenteric symptoms: Plasmoquine was stopped, the patient did not improve but ultimately was cured with injection of Emetine.

Plasmoquine is alkylamino-6-Methoxyquinolin salt and a synthetic organic compound. It is given with the idea that Black Water fever is an infection with malignant Tertian parasites and its special merit is that it acts on the gametocytes more powerfully and rapidly. The crescents are actually destroyed within three to five days, in vibrio. It also acts effectively on asexual Benign Tertian and Quartan parasites. In some cases Atebrin and Quinine in fractional doses have been used as adjuvants for their action on Schizonts, when temperature persists for more than five days. In the majority of cases temperature comes down, and the condition of urine improves within three to five days. It should be noted that Quinine if at all used, should be given in

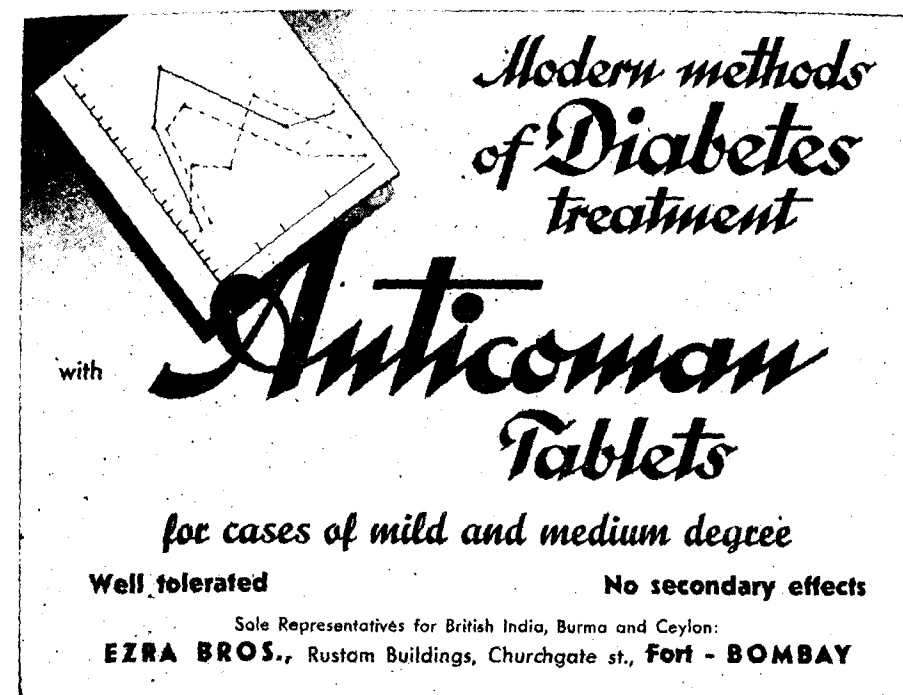
Methylene blue.—It has also been recommended, as used in Malaria.

Other methods of treatment by Quinine.—1. *Sensitization method*.—That is to sensitize the patient by using fractional doses of Quinine. After an initial dose of Alkali, Quinine should be given in fractional doses commencing with 1/100 gr. The dose should be doubled every 2 hours and the alkaline mixture to be repeated every four hours. The dose thus can be increased up to 15 gr. a day in 5 gr. doses thrice daily. But it should be stopped if there is any relapse of Black Water. It can again be repeated beginning with 1/100 gr. and using the same preparation and this is very important. The percentage of cure under this line of treatment cannot definitely be given as I have seen only two cases treated thus. The result is not unsatisfactory but the method is a tedious one. Patient refuses to take the bitter powder often. In cases of vomiting, hiccough etc. it cannot be used. Constant supervision, intelligent and reliable nurse are essential.

2. *Massive Dose of Quinine Injection*.—I have seen more than a dozen cases treated with intramuscular injection of Quinine bihydrochlor gr. (10) B.D. or t.d.s. Patients have been cured under this line of treatment and the percentage of cure is about sixty. The convalescence period is unusually prolonged. Patients generally die of heart failure and collapse. The treatment is painful, risky and costly. This is not a desirable and safe method of treatment in general practice.

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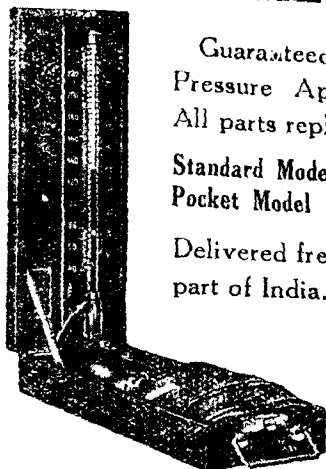
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SECTION VIII. KALA-AZAR

The Clinical Diagnosis of Kala-azar

BY

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I

KALA-AZAR is a protozoan disease which occurs endemically in many tropical and sub-tropical countries, particularly India, Indo-China, China, and the Sudan. An infantile variety of the disease is met with on the shores of the Mediterranean Sea.

In India the disease is practically confined to the eastern half of the Peninsula. It has a high incidence in Bengal, Assam, the eastern parts of the United Provinces, Bihar, and certain districts of the Madras Presidency. It is unknown in the Punjab, the Central Provinces, the Bombay Presidency, and the Malabar Coast.

The disease is very prevalent in the city of Madras. Hospital statistics show that from 2.5 to 5 per cent of the patients admitted into the medical wards of the city hospitals are victims of this disease. It has a higher incidence in certain parts of the city than in others. It is particularly common in Washermanpet, North George Town, Royapuram, Choolai, and Chintadripet. The disease is endemic also in certain places lying outside the city: e.g., Villivaukam, Perambur, Vyasarpady, and Tiruvatyoor, on the northern outskirts; Saidapet, Guindy, and St. Thomas' Mount to the South-West.

As regards the sex incidence of kala-azar, both sexes are equally liable to the disease. In hospital practice, however, more male cases are seen. This is to be accounted for as due to more men than women getting admitted into hospitals.

The disease may occur at any age. The greatest number of cases in my series occurred between the ages of 15 and 25. The disease is by no means rare in childhood. There were 22 cases in my series (of 213 cases) in persons below the age of 10. I have not seen the disease in infancy. The youngest of my patients was 3 years old. The disease is seldom encountered in persons above 50 years of age. The oldest of my patients was aged only 43 years.

Kala-azar affects persons of all classes. Circumstances of race, colour, and occupation have no bearing on the liability to the

disease except their influence in preventing or promoting exposure to infection. In the endemic areas, the European population is as liable to contract the malady as the Indian population. Further, both the rich and the poor suffer from the disease. But, in hospital practice, we see mostly, for obvious reasons, patients belonging to the poorer classes. It is interesting to note that many cases of the disease occur among Anglo-Indians.

Kala-azar is caused by a protozoan parasite, *Leishmania donovani*. In affected persons, the organism is present in the spleen, the liver, the bone-marrow, and the peripheral blood—situations where cells of the reticulo-endothelial system are found. It is oval in shape, and measures in the long axis from 2 to 3 microns and in the short axis from 1.5 to 2 microns. It contains a nucleus, which lies against the periphery, and a kinetoplast, placed almost at right angles to the nucleus. In films stained with Leishman's stain (or any of the Romanowsky stains), the nucleus can be seen as a red granular mass and the kinetoplast as a deeply stained compact rod.

It can be grown *in vitro* on such media as the Novy-McNeal-Nicolle (N.N.N.) medium and Roy's medium. In cultures it grows into a flagellate. The development into a flagellate is a phase in its life-history which occurs outside the human host. This phase is said to occur in the sandfly, *Phlebotomus argentipes*, which is regarded as its insect vector (or intermediary host.) The endemicity of Kala-azar in particular areas has been ascribed to the presence of the sand-fly in those areas.

II

Clinically, Kala-azar is characterised by long continued irregular pyrexia, enlargement of the spleen and liver, marked leucopenia with relative mononucleosis, anaemia, pigmentation, and progressive weakness and emaciation. But in its early stages, the disease presents few distinctive features by which it can be recognised, the characteristic clinical picture becoming evident only when the malady is well advanced. It is, however, extremely important to be familiar with the early symptomatology of the disease so that it may not be overlooked in the early stages.

The onset of kala-azar may be insidious or abrupt. The insidious mode of onset was noted in 70 per cent of the cases; in the remaining 30 per cent the onset was abrupt.

(a) *Cases with insidious onset.*—The illness begins with slight fever, headache, and general malaise. The temperature is seldom raised beyond 100° F. on the first and second day of the

illness, and the patient does not feel ill enough to stop away from work. The bowels may be regular, or there may be slight constipation. Profuse sweating is often noted, especially at night. In three or four days, the temperature shows rises to 102° or 103° F. In some cases, however, the rises in temperature are never beyond 101° F. Remissions of 2° or even 3° are by no means infrequent. The pyrexia may sometimes be of the continuous type. Constitutional symptoms are conspicuous, in the majority of instances, by their absence. The tongue is clean, and the abdomen soft and flaccid. Sometimes, however, there may be gurgling in the right iliac fossa. The skin is cool to the touch, and occasionally moist. The pulse shows no special features. Some of these cases are diagnosed as enteric fever, others as influenza, and a few are labelled P.U.O. (pyrexia of uncertain origin).

(b) *Cases with abrupt onset.*—The illness begins often with chilliness, and sometimes with actual rigor. The temperature rises rapidly, and in one or two hours, it may reach 103° or 104° F. Headache and malaise are usual accompaniments. Thirst is often complained of. Anorexia is present in some cases. Defervescence sets in in a few hours, and the temperature falls down rapidly. As the temperature comes down, the patient perspires freely. The temperature may reach the normal in some instances, but often it does not do so. The remission, is, however, so great that the patient is led to believe that the fever has left him. There may be another rapid accession of temperature, with or without shivering, in a few hours of the remission. Even triple rises have been noted in the twenty-four hours. In some cases, the pyrexia is of the intermittent type; but it is scarcely ever regularly intermittent, the febrile attacks occurring as a rule, only at irregular intervals. Patients often describe their illness as "ague," and even physicians of experience diagnose it as malaria. It cannot be denied that the clinical picture in many of these cases resembles, superficially at least, that of malaria, especially of the subtertian variety.

2. *The clinical course of early kala-azar* is by no means uniform in every case. At least three types may be recognised. We may divide all early cases of kala-azar into three groups on the basis of the different types of clinical evolution.

(a) In one group, comprising approximately 30 per cent. of the cases, the clinical course resembles very closely that of mild enteric fever. The pyrexia is more or less continuous in type, slight tumidity of the abdomen and bowel disturbances may

develop towards the end of the first or the beginning of the second week, and the temperature attains the normal in three or four weeks. In the majority of these cases, slight rises in temperature occur daily, either in the evening or at some other period of the twenty-four hours, after "convalescence" has set in. Rarely, a short period of apyrexia (of not more than a week, or at most a fortnight) may follow. This is succeeded by irregular pyrexia of a remittent type which continues till the patient is given the proper treatment for kala-azar.

(b) A second group is constituted by about 60 per cent. of the cases. In these, the pyrexia is irregularly remittent from the early days of the disease. The fever of the onset, which may have been continuous, remittent, or intermittent, is replaced in a few days by irregular rises and remissions in temperature. Apyrexial periods are seldom noted. Even if they occur, they are of short duration. Many cases show double rises in the twenty-four hours, and in a few even triple rises occur. Night sweats are noted in many cases of the disease. Wasting becomes evident in two or three weeks. The appetite is surprisingly good and the tongue remains quite clean. Diarrhoea many occur in some cases, but usually the bowels are regular or constipated. Some of those cases are diagnosed as acute miliary tuberculosis. If bronchitis is present, the diagnosis may read acute pulmonary tuberculosis. Malta fever is sometimes thought of, especially when the pyrexia is of the low remittent type.

In a small proportion of cases belonging to this group, the clinical picture is highly suggestive of malaria. The rises in temperature may reach 103° or 104° F., they are ushered in by chills or rigors, and they may occur with surprising regularity. The remissions are accompanied by profuse sweating. Further, the spleen may become enlarged in a fortnight of the beginning of the illness. A bitter taste in the mouth and loss of appetite also may be noted.

(c) In the third group, which comprises about 10 per cent of the cases, the initial period of pyrexia is short, usually not more than 10 days in duration. This is followed by a prolonged period of apyrexia, which may be two, three, or even four months in length. The patient says at the time he comes under observation that he had some months previously a short attack of fever from which he recovered completely, that this fever was associated with chill, but it was irregular, and that he has been keeping quite fit and going about attending to his work till quite recently when he

noticed a mass or swelling in his abdomen. It is possible that in many of these cases pyrexia of a low type has always been present but the patient has been unaware of it.

3. *The characteristic clinical features of kala-azar are usually slow in developing.* In the great majority of cases they can be noted only after the disease has run a course of three or four months. In a small proportion, however, they become evident earlier. I have seen four cases in which the clinical picture was fully developed by the end of the second month. It should be noted also that some of the clinical features develop much earlier than others.

(a) *Fever.*—The various types of fever occurring in the early stages of the disease have been described earlier. It is, indeed, a prominent symptom at all stages of the malady. In the later stages, it is irregularly remittent in character, and in the majority of cases, double rises in temperature occur during the twenty-four hours. These double rises can be best made out on recording the temperature at two-hourly intervals. Sometimes they may be noted in the four-hourly records also.

(b) *Wasting.*—Most patients show some degree of wasting even in the early days of the illness. As the disease progresses the wasting becomes increased. In some cases, however, the general nutrition is well preserved till late, when with the super-vention of cachexia, wasting becomes a prominent feature. Generally the appetite remains throughout good.

(d) *Enlargement of the spleen.*—In the majority of cases, the spleen shows demonstrable enlargement only by the end of the second month of the illness. In a few instances, the organ becomes palpable during the second or third week. The enlargement continues with the progress of the disease, and in many cases by the end of the fourth or fifth month, the organ attains such a size that its full lower pole is almost in the pelvis. The rate of growth varies from case to case; in some, it may be rapid, and in others, slow. I have seen cases in which, at the end of the fourth month of illness the spleen was almost at the brim of the pelvis, and also cases in which at the end of the ninth month, the organ had reached only the level of the umbilicus. The splenic mass is hard to the feel, and usually it is not tender. Sometimes however, tenderness is elicited on palpating the organ. This is especially the case in patients in whom the temperature shows high rises.

(d) *Enlargement of the liver.*—Enlargement of the liver is often noted towards the end of the second month of the malady. It may sometimes be delayed till three months have elapsed, and at times it may occur at a much earlier period. By the end of the sixth month, the liver is usually enlarged to such an extent that its anterior edge is palpable three or four inches below the costal margin.

(e) *Pigmentation.*—Pigmentation is observable when the illness has lasted about four months. It can be made out clearly on the palms, the soles, and the palate. In individuals of light complexion, it can be noted on the general cutaneous surface. It is of a slaty blue colour, and occurs in patches of varying size.

4. *The blood changes in early kala-azar* are a low grade leucopenia and slight mononucleosis. The leucopenia becomes noticeable by the end of the second or third week and the blood count done at the beginning of the third week might show that the blood contains only 4,500 or 5,000 leucocytes per c.mm., and a differential leucocyte count might disclose that the large mononuclears are increased to 9 or 10 per cent. By the end of the second month, the number of leucocytes would have fallen to 4,000 or 3,500 per c.mm., and the mononuclears would have shown a relative increase to 14 per cent or more. As the disease progresses, the leucopenia and the mononucleosis become more and more marked. I have seen cases in which the leucocyte count had fallen as low as 1,400 per c.mm., and the mononuclears had shown a relative increase to 26 per cent.

Other changes that have been noted are: a mild anæmia of the secondary type, a relative increase in the globulin fraction, and a fall in the calcium content. In the later stages of the disease, the red cells may have fallen to 3,500,000 or even less, per c.mm., and the hæmoglobin to 50 or 60 per cent of the normal.

The clinical features of *advanced cases* of kala-azar are sufficiently well-known that it is scarcely necessary to describe them separately. Moreover, to most of them attention has been drawn in the preceding paragraphs. It may be mentioned, however, that in a large number of cases ascites and even generalised œdema may develop. Further, pulmonary symptoms are by no means uncommon. Bronchitis, congestion of the pulmonary bases, and even hydrothorax may be met with. Bowel disturbances are quite common in the late stages, more especially diarrhœa. In some advanced cases, bleeding from the mucous surfaces has been noted.

III

The diagnosis of kala-azar can be seldom made with certainty on clinical grounds. Laboratory investigations are necessary in most instances to enable the diagnosis to be arrived at. In very early cases, positive evidence cannot be obtained even from the laboratory. In the more advanced cases, the laboratory findings are often suggestive of the disease, and occasionally of such a nature as to leave no room for doubt. It may be remarked that in most cases careful observation at the bed-side with detailed study of the blood will clear up the diagnosis.

1. In the early stages, the absence of distinctive features in the clinical picture makes the diagnosis extremely difficult and uncertain, and often impossible. When the disease has lasted a few weeks, and the pyrexia has become irregularly, remittent in type, in cases coming from endemic areas and under close observation, the true nature of the malady may be suspected, especially if double rises in temperature occur during the twenty-four hours on several consecutive days. The blood picture may be suggestive of the disease if the total white cell count and the differential leucocyte count are repeatedly done at intervals of three or four days. A gradual diminution in the total number of leucocytes and a progressive, but relative, increase of the monocytes may be noted even in early kala-azar. It should, however, be remembered that these changes in the blood picture occur only in cases that have been two weeks or more in duration. The correctness of the diagnosis is to be surmised by noting the response to treatment with preparations of antimony, which, if satisfactory, will argue in favour of the disease being really kala-azar.

It was pointed out, in discussing the clinical picture, that early kala-azar is mistaken for enteric fever, malaria, acute military tuberculosis, early pulmonary tuberculosis, and Malta fever. The differentiation of the disease from these conditions is usually effected by a process of exclusion.

Cases simulating enteric fever.—The diagnosis of these cases is extremely difficult. In the absence of repeated and thorough laboratory investigations, they can seldom be recognized as instances of kala-azar. A continuous fever, lasting more than a week or ten days, and attended by no definite symptoms and signs, is usually regarded by most clinicians as suggestive of infection with the enteric group of organisms. Indeed, enteric fever is the commonest continued fever met with in tropical and

sub-tropical climates. During the *first week* of the illness, in spite of the inability to grow typhoid or paratyphoid bacilli from the blood, the pulse rate being proportionate to the rise in temperature, and the abdominal symptoms being equivocal, a diagnosis of enteric fever is made. During the *second week*, notwithstanding a negative response to Widal's agglutination test for the enteric group of organisms, the absence of toxic symptoms usually observable in cases of enteric fever, and the spleen showing no enlargement, there is the greatest reluctance to discard the diagnosis originally made, even though the fever may no longer be of the continuous type. The slight leucopenia and the mononucleosis noted at this period make the difficulty in diagnosis greater: for, the same changes in the leucocytic picture are found in enteric fever during the second week of the disease. During the *third week*, the persistent absence of toxic symptoms, the negative response on repeating the Widal reaction, the absence of splenic enlargement, the irregularly remittent pyrexia, the absence of bowel disturbances, and the surprisingly good appetite and clean tongue may raise the suspicion that the disease is not enteric fever, but probably early kala-azar. The suspicion will be strengthened if double rises in temperature during the twenty-four hours occur on several successive days. It is, however, during the fourth week or later, when the expected convalescence does not set in, that kala-azar is usually thought of and confirmatory evidence sought for.

In the differentiation of early kala-azar from enteric fever, it is necessary, therefore, to keep the patient under close clinical observation, to record the temperature fourth-hourly (if not oftener), to have the Widal test repeated every week, and to make leucocyte counts repeatedly every three or four days. An irregularly remittent pyrexia, absence of splenic enlargement, good appetite, absence of toxic symptoms, persistently negative Widal reaction, and increasing leucopenia and mononucleosis are against a diagnosis of enteric fever and in favour of one of kala-azar (especially if the patient has come from an area where kala-azar is endemic). In cases coming from endemic areas, the physician should beware to accept as positive a Widal reaction unless it is obtained in serum dilutions of 1 in 100 or higher. If the Widal reaction is positive in serum dilutions of 1 in 25 or 1 in 50, the test should be repeated a week later, and if a positive reaction is not obtained then in a higher dilution, the diagnosis of enteric fever should be discarded. A positive Widal reaction obtained in serum dilutions of 1 in 100 and 1 in 200 is definitely in favour of a diagnosis of enteric fever;

so also is a reaction which is positive in successively higher dilutions on successive repetitions of the test.

Cases simulating malaria.—The diagnosis of these cases is seldom possible on clinical grounds. A sudden onset with rigor and rapid elevation of temperature, equally rapid defervescence with profuse perspiration, and repetition of the febrile attack daily or every alternate day, suggest a diagnosis of malaria. Even if the pyrexia is irregularly remittent in type and rigors are of infrequent occurrence, malaria cannot be ruled out, for, in many cases of malignant tertian (and some cases of benign tertian) malaria, the pyrexia is of the remittent type and rigors are infrequent. There is one point which may help in excluding malaria, namely, the absence of splenic enlargement. It is only rarely the spleen will show enlargement in kala-azar before the end of the second month of the illness; in malaria, enlargement of the organ will usually be evident in a couple of weeks.

Examination of the blood film for malarial parasites is of the utmost importance in establishing or ruling out a diagnosis of malaria, and in every case of fever suspected to be malarial in origin, the blood should be examined repeatedly for malarial parasites before anti-malarial treatment is given. If the patient has been receiving anti-malarial treatment, and the fever has shown no sign of abatement, the treatment should be stopped and blood examination carried out on several successive days. In cases of kala-azar such examination will yield negative results; in malaria, careful and prolonged search will, as a rule, reveal the causative organism.

The blood picture may often help in the diagnosis of these cases. In malaria, the red cell count will show marked decrease by the end of ten days or a fortnight. In kala-azar, the number of red cells is scarcely affected in the early stages. In malaria, the red cells usually show abnormal staining reactions, such as polychromasia, which are never seen in early kala-azar. The total white cell count does not show any deficit in early malaria, whereas in kala-azar a decrease is noted by the end of the second week. But in both diseases a relative increase of mononuclears is found.

Inability to detect the malarial parasite in the blood does not necessarily mean that the disease is not malaria. But it is unusual not to find the parasite in a case of malaria if careful and prolonged search of the blood films is made on several days. When, however, blood examination has been negative, but clinical evidence points to a diagnosis of malaria, anti-malarial therapy should be advised

and the result watched. In malaria, the response to treatment will be satisfactory; but in kala-azar the fever will be uninfluenced by the treatment adopted.

The absence of satisfactory response to proper anti-malarial treatment, taken in conjunction with the irregularly remittent pyrexia, the inability to find malarial parasites in the blood, and the changes in the blood picture, should pave the way for the correct diagnosis to be made. But it often happens that in many of these early cases kala-azar is not even suspected. They are looked upon as instances of malaria, resistant to treatment. I cannot, however, too strongly urge the need for revision of a diagnosis of malaria in cases in which anti-malarial treatment continued for a week, has evoked no satisfactory response, if the original diagnosis was made without finding the malarial parasite in the blood.

Malta fever is fortunately unknown in Madras, and it need seldom be considered in the diagnosis of the cases of early kala-azar in this area. The points in favour of this disease are: fever of insidious onset and undulant type; pains in the joints; glandular swellings; profuse sweatings; and a positive agglutination reaction on testing the blood serum with dead cultures of *Micrococcus melitensis*.

The typhoidal form of acute miliary tuberculosis is not usually thought of in the diagnosis of early kala-azar. If the suspicion arises in any case that this is the cause of the illness, considerable difficulty will be encountered in excluding it. A positive diazo reaction is obtainable in the urine of cases of this disease: in kala-azar this reaction is not obtained. Further, the progressive leucopenia and the relative mononucleosis noted in kala-azar even in the early stages are wanting in acute miliary tuberculosis. In fact, in this disease neutrophilia with lymphocytic decrease is the usual rule.

Early pulmonary tuberculosis can be excluded by a careful and thorough physical examination of the lungs, by repeated examination of the sputum for tubercle bacilli, and by radiological examination of the chest. Radiological signs are especially important in the diagnosis of pulmonary tuberculosis, and should be sought for when the disease is suspected and tubercle bacilli are wanting in the sputum.

2. Many cases of kala-azar come under observation in such an advanced state that the diagnosis can be easily surmised from the clinical picture. A history of low fever continuing irregularly for several months, dusky appearance of the face, weakness and

emaciation, marked enlargement of the spleen and liver, and patchy or universal pigmentation are so suggestive of the disease that in an endemic area the most reasonable diagnosis which can be made on this clinical picture is one of kala-azar. Examination of the blood will make the evidence more complete by revealing a marked leucopenia and relative mononucleosis. In these cases, further investigations are scarcely necessary to indicate the true nature of the malady. But, in order to avoid mistakes in diagnosis, it is advisable to obtain absolute evidence of kala-azar, which is possible only by demonstration of the causative organism.

More cases are seen at a stage when the clinical picture is not so clearly defined. The spleen and liver are but moderately enlarged; the pigmentation is not marked; the history may be of occasional bouts of irregular fever for three or four months. Kala-azar may be suspected; but other conditions giving rise to enlargement of the spleen, such as chronic malaria, splenic anaemia, and myeloid leukaemia may also be thought of. It is really hazardous, in these cases, to make a diagnosis of kala-azar without careful clinical and laboratory investigation. For, occasionally other diseases may be mistaken for kala-azar. THAMBIAN mentions a case of myeloid leukaemia coming from the George Town area of Madras City where kala-azar is endemic, in which even after examination of the blood film a diagnosis of kala-azar was made. In this case, besides the enlargement of the spleen and liver, double rises in temperature occurred on some days, and the blood film was negative for myeloid cells at the time the diagnosis was made. I saw a case of the same disease in which the clinical picture was suggestive of kala-azar, the aldehyde test was strongly positive, and irregular pyrexia was observed at the time the patient came under observation. Examination of the stained blood film, however, showed the typical picture of myeloid leukaemia. I had also another patient with enlarged spleen and liver, slight oedema all over the body, anaemia, and patchy pigmentation, and giving a history of irregularly intermittent fever of eight months' duration. Kala-azar was suspected on superficial examination; but examination of the cardio-vascular system revealed dilatation of the heart, and systolic and pre-systolic murmurs at the mitral area. It was really a case of chronic myocardial failure secondary to mitral disease complicated by chronic malaria. Mention may be made of yet another case I had under my care in which a diagnosis of kala-azar was made clinically, and confirmed by blood counts and aldehyde and stiburea

tests. A spleen puncture was done to make sure of the diagnosis. On examination of the material obtained by the puncture, no Leishman-Donovan bodies were detected, but numerous crescent-shaped gametocytes of *Plasmodium falciparum* found. Both the spleen and the liver were enlarged, pigmentation was a noticeable feature, and the patient had come from a part of the city where kala-azar is known to be endemic. These mistakes in diagnosis occurred in hospital practice which affords adequate facilities for thorough investigation of the cases.

In the clinical differentiation of advanced kala-azar, chronic malaria offers the greatest difficulty. Chronic malaria is a much more common cause of splenic enlargement than any other disease met with in this country. Malaria occurs endemically in the areas where kala-azar also is endemic. Enlargement of the liver, though not as often noted as in kala-azar, occurs in chronic malaria often enough. Pigmentation is by no means uncommon. The clinical history as told by the patient is not distinctive. And lastly, the blood picture shows leucopenia and mononucleosis. The points of distinction are:—

(1) *Freedom from fever.*—Most patients with chronic malaria do not have fever when they come under observation. In kala-azar, fever is a conspicuous feature at all stages of the disease.

(2) *Anæmia.*—This is well marked in chronic malaria; in kala-azar, it does occur, but is never of a severe type.

(3) *Impaired appetite.*—Impairment of appetite is often noted in cases of chronic malaria; in kala-azar, the appetite is good.

(4) *Spleen.*—The enlarged spleen is very hard to the feel in chronic malaria; in kala-azar, the same hardness of feel is not obtained.

(5) *Relation between splenic and hepatic enlargements.*—In chronic malaria, the hepatic enlargement is little marked in relation to the splenic enlargement, whereas in kala-azar the enlargement of the liver keeps pace with the enlargement of the spleen.

(6) *Pigmentation* is rarely a marked feature of chronic malaria in contrast with the well-marked slaty blue or dark pigmentation noticeable in cases of kala-azar.

(7) *The blood changes* in chronic malaria are characteristically those noted in cases of secondary anæmia. There is marked

reduction in the number of the red cells; polychromasia is a noteworthy feature; the colour index is less than 1; leucopenia occurs, but not to any marked degree as in kala-azar; macrophage cells containing malarial pigment may be seen on careful examination; often, on prolonged search, the malarial parasite can be demonstrated in the blood film. While these points of difference are helpful, nothing short of the actual demonstration of the causative parasite can be regarded as proof of a case being one of malaria or of kala-azar.

The diagnosis of kala-azar from the condition spoken of as *splenic anæmia* or *Banti's disease* is often fraught with considerable difficulty. For, in splenic anæmia, fever of a low irregular type may occasionally be present, and in addition to the characteristic splenic enlargement, anæmia and neutrophilic leucopenia, the liver may also show enlargement. A tendency to gastric and œsophageal hæmorrhage is a feature of the disease. The diagnosis can be made positive only by demonstrating the Leishman-Donovan body in the peripheral blood, the spleen, the liver, or the bone-marrow. However, as splenic anæmia is a much rarer condition than kala-azar, the difficulty is usually the differentiation of this disease from kala-azar. This may occasionally be impossible till the typical cirrhosis of the liver develops in the third stage of the disease. The points which help in its differentiation are: absence of pigmentation; gross enlargement of the spleen with slight enlargement of the liver; relative lymphocytosis; and hyperbilirubinæmia. The inability to detect the Leishman-Donovan body in the splenic pulp or the liver substance on two or three successive occasions should be looked upon as proof that a diagnosis of kala-azar is untenable.

The Diagnosis and Treatment of Kala-Azar

BY

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IN all cases of long, continued, irregular fever with splenic enlargement, Kala-azar should be suspected and negatived by its tests.

Leishman Donovan bodies are very rarely seen in peripheral blood. They are found certainly in smears made of splenic punctures. As splenic puncture involves the risk of fatal hæmorrhage, this test is not ordinarily possible except in well-equipped hospitals.

The Leishman Donovan body is a round or oval body 2.5 to 3.5 μ in diameter and contains a large rounded or oval chromatin body, lying towards one side and a smaller-rod shaped chromatin body lying at tangent to it.

2. The patient is anaemic. There is great diminution in the number of leucocytes. Poly morphonuclears are much diminished with relative increase of lymphocytes and large mononuclear cells: coarsely granular Eosinophiles disappear entirely.

3. Aldehyde test.

The Aldehyde Test.—1 c.c. of clear serum is placed in a small test tube and a drop of 30% formalin is added.

Positive result.

- | | |
|---|-------------------------------------|
| (a) The serum becomes solid, and completely opaque within 20 minutes. | +++ (strongly positive). |
| (b) The serum becomes solid and completely opaque within two hours. | [positive].
= - + + (moderately) |
| (c) The serum becomes solid and completely opaque within 24 hours. | = ± (Doubtful). |
| (d) The serum remains clear. | = - - Negative result. |

The Antimony Test.—The separated serum is placed in a fine glass tube and 4% solution of urea stibamine in distilled water is added slowly by means of a pipette. The solution should not be added too slowly as a certain amount of mixture at the junction of the fluids is desirable.

1. *Strongly Positive Sera.* (+ + +).

The precipitate here forms immediately and is thick and flocculent. The precipitate forms into a thick mass, generally at the junction of the serum and antimony solution and is difficult to break by shaking the tube. The precipitate is insoluble and will not dissolve if the tube is kept for 24 hours or longer.

2. *Moderately positive sera* (+ +).

In this class are included those sera in which a definite flocculent precipitate forms, but the precipitate is not so thick as in the first group. The precipitate has the same tendency to collect together into a mass at the junction of the two fluids, though the mass is not very thick. The precipitate sometimes settles at the bottom of the tube and is quite insoluble after 24 hours.

3. *Doubtful Sera* (±).

Under this category come the sera in which definite precipitate, or in some cases varying degrees of haziness, appears at the junction of the two fluids. The precipitate or haziness has not a flocculent character. If the tubes are allowed to stand for a few hours, sometimes the precipitate entirely dissolves. In other cases the precipitate still remains after 24 hours.

4. *Totally negative sera.*

In this group are included those sera in which no precipitate whatever is formed and there is not even the slightest haziness at the junction of the two fluids.

The Dilution Method.—The technique for this test is exactly the same as for the original test except that dilute serum is used. The serum is diluted with nine parts of distilled water. It is then placed in a small test-tube and the 4% urea stibamine is added.

- | | |
|--------------------------------------|------------------------------|
| A heavy flocculent precipitate | = + + + (Strongly positive). |
| A fine flocculent precipitate | = + Positive. |
| A homogeneous cloud without flocculi | ± doubtful. |
| No change in the mixture | = - negative. |

Pallacies.—The changes occurring in the serum of a Kala-azar patient which are indicated by a positive antimony test also occur to a less extent in the serum of patients suffering from certain other diseases such as Chronic Malaria and certain other conditions of splenic enlargement, phthisis and leprosy. The 'aldehyde reaction' appears to become definitely positive about the fifth month of the disease in most of the cases. As histories are notoriously unreliable, we take the size of the spleen as an indication of the duration of the disease. *When the spleen is four inches below the costal arch* the aldehyde reaction should be definitely positive. Therefore allowing a margin, we assume that all patients whose spleens are 6 inches or more below the costal margin and whose aldehyde reaction is not definitely positive are not cases of Kala-azar. Antimony test is positive in earlier months of Kala-azar. Dilution antimony test gives negative result in all non-Kala-azar cases.

Treatment.—About twenty-one years ago, Kala-azar was looked upon by the Profession as a fatal disease, and making a diagnosis of Kala-azar was equal to giving a death sentence. There was no specific treatment for the disease until Sir Leonard Rogers came to our rescue in discovering the specific treatment in the form of *intravenous tartar emetic*. For about eight or ten years the antimony tartrates were used exclusively for cases of Kala-azar. The drug is made up fresh in 2% solution and given by the intravenous route. The injection is given twice a week beginning with $\frac{1}{2}$ gr. dose and working into a maximum of 2 grs. Treatment should be continued for *at least two months after subsidence of fever and all symptoms*. A total quantity of 60 gr. or more should be given. Necessarily there were many drawbacks and practical difficulties in the above treatment. The treatment extended over a very long period during which a course of 30 injections on an average had to be given. The patients naturally fail to complete the course of treatment. There are certain *disagreeable symptoms* associated with the treatment such as coughing and vomiting.

After the year 1924, pentavalent compounds of Antimony come into general use and they have completely replaced the treatment by antimony salts. The toxicity of these compounds is very low; very much larger amounts of antimony can be given in each individual case; and the full course may be completed in a much shorter period. Adverse symptoms such as coughing and vomiting are absent. The first dose of pentavalent compounds of antimony such as urea stibamine, Neo-stibosan, is .1 gm.

intravenously; and subsequent doses .2 and .3 grms. should be given on alternate days till the course of treatment ends, which usually occupies four weeks. A total quantity of 4 grms. is usually required. These drugs are *dissolved in sterile cold distilled water, and given intravenously*.

Iron and Arsenic tonics are required to counteract anæmia. Good nutritious food is essential. Progress should be gauged by the diminution in size of the liver and spleen and the rapid increase of weight of the patient.

An intensive treatment of Kala-azar may also be done by giving a course of injections daily instead of once in two or three days.

In children where the veins are not prominent, Neo-stibosan can be given also intramuscularly instead of intravenously.

Case No. I

A case of Kala-azar with Parotitis as a Complication.

Name: Murugesan; *Age:* 22 years; *Occupation:* Toti; *Residence:* Kurumbur; *Date of admission:* 24th August 1936; *Complaint:* Fever with chill; *Duration:* Three months.

Condition on Admission.—Temperature 104° F. Tongue moist and coated. Spleen enlarged four finger breadths below costal margin. Liver enlarged two finger breadths below costal margin. There was bleeding from gums. General condition not satisfactory.

Blood.—Malarial parasite—negative; No leucocytosis. R. B. C. 2,075,000. W. B. C. 3084.

R. C. Polymorphos	...	50%
S. Lymphocytes	...	35%
L. Mononuclears	...	15%
Haemoglobin	...	50%

Temperature was keeping up from 102° to 105°F.

28-8-1936. An intramuscular injection of quinine acid hydrochlor, 7 gr. was given and it did not reduce the temperature. Temperature still keeping up.

1-9-1936. Kala-azar was suspected. Blood was taken for serum and aldehyde tests. Weight: 88 lbs.

2-9-1936. It was *positive strong* (both aldehyde and anti-mony test).

3-9-1936. 1 gr. of urea stibamine (Brahmachari), was given intravenously.

5-9-1936. Temperature gradually came down to 100°F. Second injection of urea stibamine, 15 gr. intravenous given. Temperature varied from 100° to 102°F.

9-9-1936. Third injection of urea stibamine 2 grms. given intravenously. Temperature came down to normal. Spleen reduced to three finger breadths below costal margin.

11-9-1936. He complained of pain below left ear. There was some swelling of parotid gland also. Temperature rose upto 101°F.

12-9-1936.—An incision made under local anaesthesia. Sinus forceps were thrust in. No pus came out. Poultice was applied.

13-9-1936.—Intravenous Iodine 1 c.c. given. Swelling on left cheek was increasing. Poultice applied. Spleen reduced in size to one finger breadth below costal margin—liver not palpable. Result of 3 urea stibamine injections.

14-9-1936.—Temperature came down to normal.

15-9-1936.—Swelling on the cheek was reducing still. Fourth intravenous injection of urea stibamine .2 grs. given.

22-9-1936. Temperature normal. General condition improving. Internally pepsin and hydrochloric acid were being given.

26-9-1936.—Swelling on the cheek almost subsided but there was some pain on pressure. Fifth intravenous injection of urea stibamine .2 grms. given.

29-9-1936.—Sixth injection of urea stibamine, .2 grs. given.

4-10-1936.—Seventh injection of urea stibamine .2 grs. given. Patient much improved in general health, has put on weight—weight increased by 8 lbs.

11-10-1936.—Blood again examined. Test for Kala-azar slightly positive.

12-10-1936.—Eighth injection of urea stibamine .2 grs.

16-10-1936.—Ninth injection of urea stibamine .2 grs.

19-10-1936.—General condition good. Injection could not be continued for want of drug. Weight 98 lbs.

25-10-1936.—Weight 104 lbs. (increased by 16 lbs).!

R. B. C.	...	2,175,007.
W. B. C.	...	3,594.
Polymorphos	...	66%.
S. Lymphocytes	...	27%.
L. Mononuclear	...	7%.
Haemoglobin	...	60%.

9-11-1936.—General condition improved much. Weight 112. lbs.

19-11-1936.—Tenth injection of urea stibamine .3 grms.

23-11-1936.—Eleventh injection of urea stibamine .3 grms.

27-11-1936.—Twelfth injection of urea stibamine .3 grms.

29-11-1936.—Blood was examined with the following result:

R. B. C.	...	5,437,505.
W. B. C.	...	3,437.
Haemoglobin	...	70%.

Tests for Kala-azar negative.

2-12-1936.—He was discharged cured.

His weight was 112 lbs. increased by 24 lbs. since admission. General condition was very satisfactory. Liver and spleen was not palpable. There was no fever. Quality of blood was much improved, though there was still some diminutions in W. B. C.

Case No II.

A case of Kala-azar, with sloughing of Tonsil.

A rare complication.

A male child aged 5 years, resident of Kayalpatnam, Tinnevely District, sought admission to the Dispensary at Kayalpatnam, as an out-patient for irregular fever, general debility and slight puffiness of the face.

Duration of the disease was said to be six months. On examination he was pale and anæmic. Hæmoglobin 40%. Spleen was enlarged *six finger breadths* below costal margin. Blood was negative for Malaria.

Serum test for Kala-azar was done. The tests of aldehyde and urea—stibamine were *strongly positive*. He was given a course of Neostibosan on alternate days intramuscularly for eight days. On 12-8-1935 he developed bacillary dysentery which

lasted a week. Subsequently he developed general dropsy which also subsided in a few days. There was no albumin in the urine or casts.

On 1-10-1935, the child complained of pain and swelling over the right angle of the lower jaw. On examination of throat there was swelling of right tonsil. He was having antiseptic gargles and throat paint. On 12-10-1935, it was noticed that the capsule of the tonsil had given way a little and a slough was protruding. The whole tonsil was beginning to slough in a few days. Mouth became very foul with inability to swallow any liquids. On 25-10-1935, there was profuse bleeding from the throat—about half a pint in quantity. The boy was not able to open the mouth and the bleeding part could not be examined. He given was anti-hæmostatic serum 1 c.c.

Bleeding stopped of its own accord.

25-10-1935—General condition was worse. Feeding was given by nose. Mouth very foul.

27-10-1935—Condition did not improve.

28-10-1935—Patient expired.

I am much indebted to Lieut. Col. T. S. Shastri, I.M.S., District Medical officer, Tinnevely District, for his valuable suggestions and permission to publish the above two cases.

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A Short Note on the Serological Tests and Treatment of Kala-Azar

BY

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Introduction

BEFORE serum tests were introduced, diagnosis of Kala-azar presented much difficulty and a correct and definite diagnosis was possible only in advanced cases. Treatment was more deplorable and there was a high rate of mortality before antimony treatment was introduced. Since aldehyde test has been introduced, the diagnosis has been comparatively easy, which has been further advanced by the introduction of Chopra's Urea-stibamine test and since aromatic organic antimonials have been introduced in the treatment of leishmaniasis, the mortality in Kala-azar has come down to a very low percentage.

Now only the very early cases present some difficulty as no serum test is found positive earlier than the third week from the onset of the disease. The different modes of onset of fever and its simulation with many tropical fevers may always lead to confusion and even in moderately advanced cases, where the serological test is not positive, accurate diagnosis is sometimes difficult to establish. Only in well-equipped hospitals, a spleen or a liver puncture may be possible or some blood may be taken and cultured in N. N. N. media, but in the field, no such contrivance is available and the physician is required to depend entirely on clinical signs, which are not very marked till the case is fairly advanced and on therapeutic tests and serological examinations. The scope of this paper will not allow us to go into the detailed clinical descriptions and the writer will endeavour to describe the serum tests possible in the field with short notes on different preparations at present in use and are efficacious against Kala-azar.

Serum Tests for Kala-azar.

Aldehyde Tests.—1. *Field test.* (Fox and Mackie). Prick the finger and collect a little blood in a Wright's capsule. Allow serum to separate. Place a drop of serum on slide and invert the slide over a small petri dish, which contains some formalin. In definitely positive cases the drop becomes completely solid and

opaque within a few minutes to half an hour. If only jellification occurs, without opacity, the test is not positive.

2. *Napier's Formalgel Test* (Napier, 1922).—Take 5 c.c. of blood from a vein and allow to clot in a test tube. When serum separates, place 1 c.c. of serum in a small test tube (1.5 cm. in diameter) and add to it one drop of formalin. Shake well and place. In positive cases the serum turns into a white jelly.

If jellification occurs within 20 minutes	+++
" " 2 hours	++
" " 24 "	+

If the serum is solid, not opaque but opalescent

If the serum is solid but slightly opalescent	... +	(Doubtful).
" " after 24 hours		Negative.

Antimony (Urea-Stibamine) Tests.—1. *Chopra's Test with undiluted serum* (Chopra, Gupta and David, 1927). Take some serum in a Dreyer's tube with a capillary pipette and add some 4 per cent solution of an organic antimony compound (Urea stibamine) slowly along the side of the tube. If the serum is positive, a thick flocculent precipitate forms immediately, otherwise there is no precipitate whatsoever or there may be only a slight precipitate at the junction of the two fluids.

This test can be performed in a fine glass tube about 0.5 cm. calibre sealed at one end, or a small test tube. Place the serum in the tube and place the tube in rack and add slowly with a capillary pipette some 4 per cent. solution of urea-stibamine. The solution should not be mixed too slowly as a certain amount of the mixture of the fluids is desirable in the junction. Reading should be taken after 10 minutes.

If the precipitate is immediate and thick and flocculent and forms into a mass at the junction, which is difficult to break down on shaking and insoluble after standing 24 hours	+++
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Precipitate definitely flocculent but not so marked as above. May collect at junction or settle down	... ++ or +
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A definite precipitate or haziness of various degrees at junction but not flocculent. May or may not disappear in a few hours	... ±
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No. precipitate or haziness	... -
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(2) *Chopra's Test with Diluted Serum* (Chopra, Gupta and Basu, 1927).—This test is performed as above (i) with undiluted serum, but here the serum used is diluted 1 in 10 with distilled water. Readings to be taken within 5—10 minutes, as late reading may not be accurate. A marked reaction shows a white flocculation and only the positive reaction should be taken as indicative of kala-azar. If spleen is more than 6 inches below costal arch, only a triple positive result should be regarded as indicative of kala-azar.

Readings:—

White and heavy flocculent precipitate	... +++
Fine flocculent precipitate	... ++ or +
Cloudy appearance without flocculation	... +
Without any of these	... -

(3) *Chopra's Finger-blood test* (Chopra and Mukerjee, 1930).—Collect a drop of blood by pricking a finger into a small test tube 3/8 inches in diameter and 2 inches long containing 0.25 c.c. of a 2 per cent. potassium oxalate solution. Shake the tube well to mix thoroughly and wait for 10 to 15 minutes to allow the corpuscles to settle down. Take off the supernatant fluid in a pipette and place in a small test tube. Run in some 4 per cent. solution of urea stibamine slowly down the side of the tube, which being heavy permeates through the diluted serum. Reading should be taken at 5 to 15 minutes. In kala-azar cases, a coarse precipitate appears slowly in the whole column.

(4) *Napier's Antimony Test* (Napier, 1927).—Place 2 c.c. of 0.25 per cent. urea Stibamine solution in a test tube to which add 2 drops of serum and agitate. Allow it to remain for 10 minutes and then take first reading and after two hours, second reading. In a case of kala-azar, a heavy or light flocculent precipitate forms and settles down, leaving a clear supernatant fluid.

In the three last tests described above, a very small amount of serum is required and the tests are useful for any practical purpose. Any other organic antimony compounds may be used in place of urea stibamine in all these tests and usually the antimony tests become positive after the second week of the disease.

Treatment.—Even in the early part of the twentieth century, no specific treatment was known and the mortality rate of the clinically diagnosed cases was as high as 80 per cent. Since 1913–14 trivalent antimony compounds have been used with success both in India and elsewhere. A 2 per cent. solution was

used beginning from 2 c.c. and increased by 1 c.c. upto 5 c.c. intravenously twice a week. Solution of Sodii antimonij tartrate or potas antimonij tartrate was used. Thirty to forty injections were required to complete recovery.

Later pentavalent compounds of antimony have been introduced and their introduction may be regarded as an important advance in the treatment of Leishmaniasis. Von Heyden in Germany first introduced a pentavalent aromatic compound "Stibacetin", which gave encouraging result and later Brahmachari of Calcutta prepared "Urea-Stibamine" under the auspices of the Indian Research Fund Association which proved therapeutically very effective. There are other homologous preparations in the market, which have a more or less similar composition and action. These are Stiburia, Urea-Stibol and Stiburamine. The advantages offered by aromatic compounds according to Napier (1927, a) are :—

(1) 'Pentavalent compounds are less toxic and can be administered in larger doses.

(2) 'Total amount of antimony can be administered in a smaller number of doses. Accordingly about 10 injections of pentavalent compounds are equal to 30-40 injections of tartar emetic : hence reduction of time from two months to three weeks.

(3) 'Resistant cases improve rapidly under larger doses of aromatic compounds.

(4) 'Disagreeable symptoms such as coughing, joint pains, severe reactions etc. associated with tartar emetic are much less frequent with pentavalent compounds.

(5) 'Death rate is markedly reduced with pentavalent compounds.'

Doses of and treatment with Urea-Stibamine:—Initial dose recommended by manufacturers is 0.05 gm. increased by 0.05 gm. at each injection upto 0.2 gm. to be injected intravenously, twice a week. The drug is issued in 0.025 gm. 0.1 gm. 0.15 gm. and 0.2 gm. ampoules and any of these doses may be repeated at the discretion of the Physician. Usually 8 to 12 injections are required but in resistant cases more injections may be necessary. The average amount of drug necessary for a cure is usually 2.6 gm. Brahmachari has successfully treated cases with daily injections of Urea-stibamine for 6 to 8 days. Earlier cases react better to treatment.

Urea-stibamine, urea stibol, stiburea, amino-stiburea and stiburamine are all similar in composition and have similar dosage.

Brahmachari claims that urea-stibamine is ammonium carbamino-stibanilate or urea p-stibanilate but Chopra (1936) has shown that it is not a compound but a loose combination of urea with para-amino-phenyl-stibinic acid and urea can be completely separated by repeated washing with alcohol. He has further shown that the different samples in the market vary in their antimony content from 20 to 43 per cent. and consequently very different form of result is obtained from different batches of the drug.

Neostibosan (Bayer 693 B).—Of late, this product is extensively being used and is said to be the best of the pentavalent compounds now on use. Few years ago, Von Heyden introduced a compound under the trade name of 'Stibosan' from which 'Neostibosan' (von Heyden 693 B) has been developed. This is a stable compound and is at least as effective, if not more, as urea stibamine.

Neostibosan forms an isotonic solution in 20 per cent. concentration and can be given by intramuscular route. Thus it can be used safely in cases of infantile and juvenile Kala-azar, where intravenous injection is always a difficulty. Owing to the small volume, the injections are well tolerated and can be given by a 2 c.c. hypodermic syringe. The drug can be given intravenously in 5 per cent solution.

The doses recommended are :—

Age	2—4 years	Initial dose	...	0.05 gm.
		followed by 4 doses of	...	0.1 gm.
	5—9	Initial dose	...	0.1 gm.
		followed by 4 doses of	...	0.2 gm.
	10—15	Initial dose	...	0.1 gm.
		followed by gradual		
		increase up-to	...	0.3 gm.
Adult		Initial dose	...	0.3 gm.
		followed by 7 such doses.		

Adult patients can be given in all 8 injections on consecutive days but many Physicians prefer to give the injections every second to third day. In India, a total dose of 2.4 gm. for an adult is found to be sufficient. The clinical symptoms, particularly fever, usually subside after the 5th or the 6th injection but in some cases it may outlast the whole course. There is entire absence of nausea and vomiting and the patients tolerate the drug very well.

Neostam or Stibamine Glucoside.—It has been prepared in the Wellcome Research Laboratory and offered by B.W. & Co., in the market. It has been used in a limited number of cases in the

Tropical School Hospital, Calcutta and is said to have given good result. A watery solution of 4 per cent. strength is used intravenously in the doses of 0.05 gm. to 0.3 gm. The writer has no personal experience with this preparation.

Relative value of Pentavalent Compounds.—There is a consensus of opinion that neostibosan is the most innocuous of all the antimonyl preparations, having altogether no untoward complications after injections. As regards speed of cure, the authorities in the Calcutta School of Tropical Medicine are of opinion that it produces the cure in the shortest period (Chopra, 1936). In our hands, the period of cessation of fever is almost the same as that with Urea-stibamine, if the injections are given with two days intervals. Rather some physicians think that fever is checked earlier with Urea-stibamine. Intramuscular injections are a little slower in checking the clinical symptoms than intravenous administration. Reactions are more common with Urea-stibamine, but they are usually absent with neostibosan. A case not improving with urea-stibamine may improve with neostibosan and vice versa.

Toxic Complications of Urea-stibamine Injections.—Nausea, vomiting and fits of coughing may follow immediately after an injection of urea-stibamine. In such cases the dose should be reduced and when increased, it should be increased very gradually. The vomiting usually starts within 20 minutes and it may be preceded by giddiness. The injection should not be given immediately after meals and the patient should be made to lie on bed for at least an hour after the injections. Fatal collapse and death are also on record (Chopra, 1936).

Recently the writer came across two cases of very severe reaction after urea-stibamine injections. One was a boy of 12 years who was given six injections without any untoward effect whatsoever. When the 7th injection of 0.1 gm. was given, the boy began to vomit and there was very severe shock, pulse became irregular, there was flushing of the face, choking and breathlessness. Half c. c. of adrenal chloride solution was injected and the condition passed away gradually.

The second case was a Hindu widow aged 30. She was given seven injections without trouble but when she was given the 8th injection (0.2 gm.), she had a similar attack. Her pulse became imperceptible at the wrist and the heart became very slow and irregular. One ampoule of adreno ephedrin failed to bring her round, and I was so frightened that a senior doctor had to be called in for consultation. Another injection of 0.5 c.c. of adrenal

chloride and 0.5 c.c. of pituitrin (P.D.) was given but still the condition persisted for more than one hour.

Such anaphylactic symptoms resembling 'nitritoid crisis' are perhaps due to change in the blood brought about by these compounds which are of a semi-colloidal nature. Usually these reactions are met with after the 6th or 7th injection, when the maximum dose is given. Just after the injections, the patient becomes restless, there is vomiting, face becomes puffy, there may be urticarial rash, there is dyspnoea, stertorous breathing. Pulse becomes weak, irregular and imperceptible, the patient is cyanosed and collapse soon supervenes. Unless prompt measures are instituted, the case may turn fatal. Such dangerous reactions are more common after urea-stibamine and are rare with neostibosan. Immediate injections of adrenalin and pituitrin are imperative in such cases.

In sensitive cases, it is better to abandon treatment for the time being recommencing with minute doses of some other compound, if necessary.

Contra Indications.—It should be used with great caution in weak, anaemic and emaciated subject. In cases associated with heart, kidney and lung diseases, the injections should be given. Urine should be examined at intervals.

Acknowledgement

I wish to place on record my deep sense of gratitude to my chief Dr. S. K. Biswas, Medical officer, D. H. Railway for his valuable instructions and his kind interest in this note.

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Factors for Consideration in the Treatment of Meningococcus Meningitis

BY

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BEFORE introducing the subject matter proper, it is necessary to dwell upon how Cerebrospinal Meningitis develops. Infection gradually takes place through the naso-pharynx. The infecting organism enters into the blood stream and causes a condition of Meningococcus Septicæmia and subsequently the organism settles in the meninges and gives rise to symptoms of Meningitis like stiff neck etc. Infection may take place in another way. Meningococcus may penetrate through the lymphatics along the olfactory nerve to the meninges. Direct extension through the bony-wall of the sphenoidal sinuses or by way of nasal sinuses through the lymphatics are also considered as channels of invasion. In the first mode of infection, the patient is seen with a condition of Septicæmia. The case may, then, be mistaken for malaria—but blood examination will show a high leucocytosis with a total count of, between 20 to 40 thousands, with a big rise in the percentage of Polymorphonuclear leucocytes (between 80 to 95 p. c.). This, in itself, will give an indication of Septicæmia and blood culture will reveal the real causative organism. A big quantity of blood, not less than 10 c.c. should be taken in a specially auto-claved all-glass syringe. 25 c.c. of ordinary broth with 0.2 p. c. Glucose will serve as the media for growth. After 24 hours, the growth has to be transferred to blood agar as well as ordinary agar, and watched for 48 hours for growth of any microbe. The same process is to be repeated after 48 hours. In the second type of cases, lumbar puncture is done and the Cerebrospinal fluid obtained is taken in the broth with strict sterile precautions and is partly centri-fugalisied and the deposit examined microscopically with Gram staining as well as Zeil-Neilson's Carbol-fuchsin staining. Gram-negative Biscuit-shaped Diplococci in the culture media will confirm the diagnosis of Meningococcus infection..... Examination in serum—Insulin to differentiate Pneumococcus and in serum broth to find out if Gram-positive chains (Streptococcus) or Clusters (Staphylococcus) be available, has to be done. Bacilli may be found out in some cases which may subsequently

be identified as Influenza Bacillus. Centrifuged deposits of C. S. F. may in some cases reveal the presence of Tubercle Bacillus.

Precipitin reaction.—Maegraith has recently described a precipitin reaction and claims that a diagnosis of Meningococcal meningitis may be made in a few minutes at the bedside within 24 hours after Meningococcus localises in the meninges. It will give negative or doubtful results in Septicæmic cases and only when meningeal invasion takes place, it gives a positive reaction. The reaction consists of a ring formation, from the interaction of Polyvalent group 1 anti-meningococcus infections and similar is the case with Group 2 Polyvalent serum.

Different types of meningococcus.—Research workers in the line have not yet been able to find out one antigenically broad Meningococcus, which covers the whole range of infective types. Originally, there were 4 types (after Gordon). But it has been recently found out that there is antigenic overlapping of types i and iii and of types ii and iv. It has therefore been proposed by the Medical Research Council to include types i and iii in group i and similarly types ii and iv in group ii, which includes all other strains of meningococcus that are otherwise typical but not agglutinable by group sera. Majority of Meningococcus strains isolated from C. S. F. belongs either to group i or to group ii. In some epidemics, prevalence of group i strains may be the rule, and in others group ii. At the height of an epidemic, again, strains belonging to group ii may predominate whereas at the fall of the same epidemic, group i may be more in evidence.

Sara E. Branham of United States Public Health Service obtained meningococcus-like organism (*Neisseria Flavescens*) which is pigmented i.e., yellow-coloured and does not ferment any of the sugars. This organism was, so far, known to have been only found in Chicago. But recently, we have obtained a few cases of Meningitis from C. S. F. of which yellow pigmented *Neisseria Flavescens* was isolated.

The author, while studying the nature of infective organisms in Meningitis, isolated in 40.6% of cases of a particular epidemic in Calcutta, a gram-negative diplococcus, which can grow in ordinary agar from the very beginning and does not ferment any of the Carbo-hydrates generally used in classifying the genus *Neisseria* viz: Dextrose, Levulose, Maltose and Saccharose. This has been described as 6 A group of Atypical Meningococcus.

Yet another peculiar red pigment-producing gram-negative tetrad Giant Cocci were isolated from two cases of which, one

was found in the middle of April, '36 and described in December, 1936 issue of the Antiseptic. The other, identically the same, was isolated from another case of Meningitis. This strain is still under study.

The important point is to bring to the notice of the physicians the fact that Atypical Meningococci are obtained in a large percentage of cases. Before decrying a particular serum, the physician will do well to send the C.S.F. for slide and cultural examination and after isolation of the causative organism, to find out if agglutinin is present in the particular serum against that particular strain. If not, he is not justified in saying that the serum treatment is no good but he is well justified perhaps in believing that the particular serum used will do no good to this particular patient. If the strain found from C.S.F. is found to be agglutinated by the serum used for a particular case, then and then only he can expect to get any result at all. For this reason, the author puts forward a plea for active collaboration between the technician and the clinician, if a definite answer is to be given as to why the mortality is very high in the case of Epidemic Cerebro-Spinal Meningitis.

Polyvalent Anti-meningococcus (Anti-bacterial) serum which contains antibodies against different kinds of typical and atypical infective strains has given moderately good results. Lumbar puncture, thereby allowing free drainage and intraspinal administration of serum is the method of choice. The author recommends, as he has done in earlier articles too, large single doses not less than 150 to 200 c.c. or even upto 300 c.c. according to age and repeated every 12 to 24 hours until definite improvement of signs and symptoms is noticed. Glucose intravenous is always to be given as a supplement.

As regards Ferry's Antitoxin, Banks found that in group 1 infections, it gives good results. Cisternal puncture with serum 2 inoculation has given good results with him. He recommends anti-toxin twice daily, alternately by the lumbar and cisternal routes.

In those cases where toxic manifestations start early, it is best to combine anti-bacterial and anti-toxic serum treatment. In that case, eye complications which may go even to Blindness, are sure to be avoided.

A new method of treatment, initiated by the author is under study now in an Infectious Hospital in Calcutta, with a certain success uptill now. Meningococcus Bacteriophage is being used intravenously in meningo Septicaemic cases in doses of $\frac{1}{2}$ c.c. on

the second day; 2 c.c. on the third day which is being increased to 5 c.c. in a few cases. In ordinary cases Lumbar puncture followed by intrathecal introduction of meningo-coccus bacteriophage in 5 c.c. doses is being done. Of course, a very small number of cases has been done uptill now and as such, it is unwise to give the result definitely. The cases are being closely followed and a change in doses will be resorted to, if thought desirable. A detailed report with case notes will appear shortly.

Hypnotics in Bubonic Plague

BY

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WHATEVER the chemical constituents of the endo-toxin of the *B. pestis* may be, it has a deleterious effect on the cardiac muscle no less than on the Central Nervous System. The disease has been thoroughly studied in its epidemical aspect but in the Chemo-therapeutic armoury, a gun has yet to be found out which will enable us to kill the bacilli in the initial stages of the disease before the blood-stream is saturated with the fulminating endo-toxin. In spite of the above disproportionate factor some remedies have gained reputation to act in a specific way during epidemics, only to make room for better ones within a short time. Thus a Hakim or a Veidu claims to have cured a large number of plague cases with the application of certain pastes to the inflamed buboes or some herbal mixture given orally, perhaps offered gratis, the formula of which cannot be disclosed by him without the permission of his worthy preceptor. This category of our noble profession promises to cure the patients for a paltry sum while on the other hand their Allopathic brethren in charge of a big hospital staffed by well trained assistants and stuffed with modern appliances can hardly utter a word of assurance while using costly anti-plague serum.

The plight of a general practitioner running a small dispensary in the rural areas is still worse. He is denied the advantages possessed by the two extremes of his profession. The laity looks upon him for the cure of disease with implicit faith and expects him to cure the patient at a cost not exceeding the price of the coffin for which he is fast heading. The view of the general

practitioner, however, has not undergone any material change so far as the treatment of bubonic plague is concerned. The same old methods of treatment still hold the palm of superiority although prophylactic inoculation plays an important part in the domain of preventive medicine.

It is needless to go into detail so far as the signs and symptoms of a bubonic case are concerned. It presents no difficulty during epidemics. It should not be, however, forgotten that nervous symptoms are developed as soon as the disease manifests itself. At the beginning of the epidemic the patient is found tossing on the bed, restless, and delirious and the practitioner is at a loss to know how to control the fever and the agonising pains of the bubo. The inflammatory process is so acute that even the adjacent structures may be affected. This is so especially in the neck. The inflammation is so great and extensive that the neighbouring structures, the wind pipe and the food passages are obstructed. The whole face is swollen so much so that a gloomy aspect renders the rural practitioner dumb-stricken. When the epidemic is practically tailing off, any line of treatment may give satisfactory results. Sometimes patients with buboes in their groin roam about in the streets sullen and dejected and recover with ordinary medicine and precaution. It is only in the beginning of the epidemic that the value of a certain medicine can be assessed.

The effect of anti-plague inoculation is in general twofold. It creates a sense of collective security which drives away panic from amongst the public. Secondly, it develops an early immunity and if the inoculated person be attacked, he has better chances to fight the infection. Clinically the toxic condition is less pronounced and the patient rarely develops a delirious condition. The plague is a self limited disease like Pneumonia or Typhoid. If a certain period elapses nature musters strong its vital forces in the form of immunity. The line of treatment, therefore, to be adopted in a bubonic case should be to make the cardiac muscle play its game and to detoxify the blood in the initial stages of the disease. This is the stage when the C.N.S. and the cardiac muscle require protection from the damaging effects of the endo-toxins of the *B. pestis*. Various preparations of Iodine and other drugs have been given intravenously but are not reported to have given entire satisfaction, or proved to be of any dependable value.

I have been using Septicemine (A.F.D.) for some years past. Its action has been found to be of some benefit. It has been used extensively in the Paris Hospitals in septicæmic conditions of the blood. If given intravenously it detoxifies the blood through the

cleavage of the idomethylate owing to the dynamic chemistry of the blood. Thus the toxic condition has a tendency to go downhill which is marked by an improvement in the clinical signs and symptoms after a couple of injections or so. Along with these injections digitalis and strophanthus in proper doses are to be given. Although so much has been said against the efficacy of these preparations, there is no go for us but to prescribe them. A drachm of the tincture of digitalis with a few drops of the tincture of Strophanthus should be ordered as a routine measure. It is quite essential that the patient sleeps well. It should be a cardinal rule that no plague patient passes a sleepless night. With this end in view, sleep-inducing drugs should be administered. Hypnotics have a wide range of usefulness in bubonic cases. Bromides 30 grains per diem and a hypnotic pill such as Veramon, Medinal or any other similar preparation with which the practitioner happens to be familiar should be prescribed before retiring. The administration of the pill should be properly timed so that the patient may go to sleep at his usual hour of retirement. This regime is of the utmost importance. The patient sleeps well. As the toxic condition clears off owing to the above injections, he is calmed down. It eases the attendants and averts frequent night visits by the practitioner. After a couple of days or so he may welcome his doctor with a gentle and grateful smile on his face in the morning. Morphia has its own advantages. Those that are using it may do so with some concomitant results. There is one school of practitioners who are inclined to make some fuss about the so-called depressant action of the hypnotics and will point an accusing finger at it. Those drugs that are distinctly depressant should, of course, be avoided. Fortunately we are at present armed with various preparations of a hypnotic nature which can be confidently prescribed without any harm.

The writer of this article has used sleep-inducing drugs liberally in the plague epidemics of 1932, and 1934 and has obtained good results in the early part of the epidemic when the mortality was high.

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Summer Fever in Children

BY

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IN a tropical climate like that of Bengal, children with weak constitution often develop a low type of fever during the summer months. Owing to its apparent relation to season and atmospheric temperature, it may be termed summer fever. This fever has also been described as Thermic Fever by Nandi (1930), but this may cause confusion because the latter nomenclature has been used by various authors to describe the hyperpyrexia of heat-stroke which is quite different from the disease under review. So the present author has preferred the term Summer Fever for this type of pyrexia.

Predisposing causes.—Premature infants are very liable to get this fever. Children born at full term but constitutionally weak, and of underweight due to malnutrition and other causes are also apt to have the disease. Most of the children who develop this type of fever are found to have been suffering from some form of gastro-intestinal disorder of which the present author has found constipation to be the commonest. Rickets and Congenital Syphilis are other predisposing causes. This type of fever may also occur in apparently healthy and well developed children after crops of boils during the summer or with summer diarrhoea. Unhygienic surrounding is said to predispose towards it, but the present observer has seen cases amongst children living in good hygienic conditions. It is more common in town-dwellers than in those who live in villages.

Mother's health.—In many cases the mother is found to be a subject of chronic ill-health due to chronic gastro-intestinal disorder, anaemia, repeated pregnancies, prolonged lactation, malnutrition, chronic-malaria and various other causes.

Seasonal incidence.—Most of the cases occur during the months of May and June when the atmospheric temperature is high and before the rains have set in. But cases may also occur in later months during the rains.

Onset.—The onset in most of the cases is insidious, and in many cases the mother cannot say precisely when the rise of temperature actually commenced. In some cases it starts like an acute infectious disease with a sharp rise of temperature several

degrees above normal. It is sometimes initiated by summer diarrhoea or furunculosis.

General condition.—In many cases there is not much emaciation though the child might have run the febrile temperature for several months. But in those cases where a high temperature or diarrhoea has continued for a long time there may be appreciable emaciation though never so much as is met with in many other acute infectious fevers. The child looks pale and is often peevish.

Age.—Most of the cases occur between the ages of six months and two years, but children of other ages are not exempt.

Temperature.—Temperature is generally irregular. In many cases with an insidious onset a low type of fever continues for several months. The temperature in such cases is generally found to be normal or subnormal in the morning and 100° F. or so in the afternoon, while occasionally there may not be any intermission at all. Sudden rises of temperature to several degrees above normal and lasting for several days without any apparent cause are not uncommon in the course of the disease. In some of the cases there is generally a morning rise of temperature instead of in the afternoon, though occasionally there may be a rise in the afternoon or evening. A number of cases have an acute onset with a sharp rise of temperature, and a remittent course for days or weeks, the temperature generally varying between 101° and 103° F. or so. In such cases after sometime the temperature gradually comes down and a slow, irregular, intermittent type of fever as described before continues. Nandi (1930) noticed that the rise of temperature in this type of fever varies with the diurnal variation of atmospheric temperature "being highest during the hottest part of the day or night and may have two or three exacerbations conforming with the temperature of the atmosphere", but this has not always been the case in the present author's experience. The accompanying temperature chart of one of the present author's cases shows the course of the temperature in a typical case of this fever. (*See Chart.*)

Course of the fever.—Some cases improve with the advent of rains. In others the fever continues till late autumn or the winter. In a few cases this irregular rise of temperature will go on for many months, irrespective of seasonal change of temperature, with occasional periods of well-being for some days, till there is definite improvement in the general condition of the child.

Gastro-intestinal tract.—The tongue is coated, but thick furring is not common unless acute gastro-intestinal trouble is also present. Loss of appetite is not common in the present observer's

experience. Constipation is present in a large number of cases. It is occasionally followed by diarrhoea.

Examination of stool.—It indicates nothing particular. In most of the cases culture shows organisms normally found in healthy stools. Two cases with diarrhoea showed organisms of the Salmonella group on culture of the stool.

Liver.—It is enlarged in some cases, in others, it is not palpable. The enlargement, when present, is never so great as in Infantile Cirrhosis of the Liver, nor is the organ so hard. The disease has not also the progressive character of the Infantile Cirrhosis of the Liver.

Spleen.—Not enlarged in any of the typical cases. In those cases where it is enlarged, it is associated with enlargement of the liver and the whole process resembles Infantile Cirrhosis of the Liver in its early stages.

Blood.—No characteristic change is found. There is slight secondary anaemia. Total leucocyte count varies between 6000 and 10,000 per c.m.m. Differential count shows a comparative increase of the lymphocytes. But these counts are normally seen in healthy children. So there is nothing characteristic in them. No malaria parasites are found.

Urine.—Quantity of urine passed is generally normal except during high fever when there is slight diminution due to fever. There is no difficulty in micturition. Chemical and microscopic examinations show no characteristic changes. Pus cells may be detected in many cases and there may be slight febrile albuminuria.

Urine Culture.—Most of the cases showed B. Coli on culture. Some of the cases especially those who were suffering from boils, tonsillitis etc. showed Streptococci or Staphylococci, most commonly the latter in the urine.

Pathogenesis.—Seasonal incidence, and also cure of the febrile temperature on the advent of the cold weather in many cases, lend support to the view that the pyrexia in these cases is related, at least to some extent, to the dysfunction of the central heat-regulating mechanism which cannot properly bring into operation all the balancing mechanisms which are necessary to keep the body-temperature normal under variable atmospheric conditions. But this is only likely in cases of premature infants. Blackfan and Yaglou (1933) have shown that the heat regulating mechanism in normal full-term infants is adequate to protect them from excessive heat by vasomotor shifts of the blood to the peripheral vessels, and by ample secretion of sweat. In premature infants however,

the physiologic response to, and the time of recovery from the effects of heat and cold are related directly to the foetal age and body weight. The smaller infants perspire but slightly or not at all, and the rectal temperature rises considerably on exposure to heat. On exposure to cold they likewise show an imperfect defensive reaction. The humidity best suited to stabilising the body temperature of premature infants according to these authors, appears to be about 65 per cent. with a temperature ranging from 75° to 100° F., depending to some extent on the general constitutional state of the infant and the body weight. A humidity of 30 per cent. induces instability of body temperature and other untoward effects.

Below is recorded a statement of temperature and humidity data as recorded in the Alipore (Calcutta) observatory and kindly supplied to me by the Meteorologist, Calcutta to whom my best thanks are due.

A statement of temperature and humidity data recorded at the Alipore Observatory:—

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
Mean humidity based on Observations 1901-1920	Highest % 87	% 86	% 87	% 87	% 87	% 89	% 91	% 92	% 92	% 92	% 89	% 86
	Lowest % 46	% 42	% 41	% 49	% 57	% 71	% 77	% 79	% 76	% 66	% 52	% 45
	Mean % 73	% 69	% 68	% 71	% 74	% 81	% 85	% 86	% 86	% 82	% 76	% 72
Mean humidity (1936)	60	66	55	63	82	86	86	84	85	81	70	
Mean Maximum Temperature (33 years mean)	°F 77.3	°F 82.0	°F 90.9	°F 95.6	°F 94.5	°F 91.5	°F 88.4	°F 87.6	°F 88.0	°F 87.2	°F 82.0	°F 77.0
Mean Maximum Temperature (1936)	73.1	83.7	95.9	98.9	92.8	89.1	89.1	89.3	88.9	87.7	84.9	
Actual highest maximum (1936)	83.2	94.3	100.3	117.2	99.6	94.3	92.7	93.1	92.3	92.6	89.1	
Actual lowest Maximum (1936)	76.4	72.6	91.2	89.4	82.2	80.9	81.5	83.6	82.2	79.7	80.2	

It will be observed from the above data that the maximum and minimum temperature recorded during the months under review do not differ much from the temperature range experimentally found to be best suited for the healthy growth and also stability of body temperature of even the pre-mature infants. Humidity recorded during these months has generally been high and has never been so low as to be injurious to the child. However, the

temperature and humidity data require to be further studied in relation to this disease.

In studying the pathogenesis of this disease one must also take into consideration several other factors which may have some influence on the causation of the disease. Thus most of the cases suffer from some sort of gastro-intestinal disorder. Some of the cases start with furunculosis or some other septic process. In most of the cases culture of the urine shows the presence of some pathogenic micro-organism, especially of the *B. Coli* group even on repeated cultures. In two cases of the present series culture of the stool showed organisms of the *Salmonella* group. Some of the cases are not cured at the advent of the colder months and the fever continues during the winter. About the benefit which is said to occur from change to a colder climate the present author thinks that the cure in these cases may be ascribed more to the change to a better and more salubrious climate than to a change of atmospheric temperature alone. The following case which is still under observation illustrates the point:—

A child 5 years old, resident of Calcutta, was attacked with this type of low fever since July last. He left Calcutta for a change to Jamshedpur in September and lived there for about one month. Immediately on his arrival there his condition improved. He had no rise of temperature since the fifth day of his arrival there and remained afebrile during the whole period of his stay there. Three weeks after his return to Calcutta he began to have the rise of temperature again which is still continuing even during the month of December. It may be pointed out here that the atmospheric temperature of Jamshedpur is similar to that of Calcutta but the climate is better.

From the facts discussed above it appears that this type of fever, at least in some of the cases, may be caused by sub-infection. Atmospheric condition seems to act in these cases by lowering the resistance of the protective gastro-intestinal mucosa and also the general vitality. Adami (1914) has shown that normally micro-organisms are being constantly carried away from the intestinal tract and are being constantly killed in the mesenteric nodes and the liver. If the vitality of the Gastro-intestinal mucous membrane be lowered, a greater number of micro-organisms will gain entrance and a febrile reaction will occur. This will continue till the resisting capacity of the gastro-intestinal epithelium or the general Somatic resistance or both improve sufficiently to cope with the

subinfection. This seems also to be the case when the process of subinfection starts from some other foci.

Outline of treatment adopted by the present author;—

1. Attempt is made to improve the general condition of the child.

(a) by giving nutritious and easily digestible foods at regular intervals;

(b) by rectifying the deficiency of vitamins, as the low vitamin content of the diet may help inter-current infections even without producing the specific signs of avitaminosis. Thus in deficiency of vitamin B there is said to be a derangement of the functions of the digestive tract and also devitalisation of the protective intestinal mucosa. Bactericidal power of the blood is also said to be diminished in cases of deficiency of vitamins A, B, and C, and along with it an increased susceptibility to infection.

(c) by general hygienic measures, as regular sponging or bath, sunlight and fresh air;

(d) by change to a better climate where possible.

2. Medicines:—Intestinal antiseptics, digestives and bacteriophage may be tried to improve the condition of the gastro-intestinal tract where it is at fault. Auto-vaccines prepared from the urine or other septic foci, commencing with minute doses and gradually increasing, may be attempted along with general hygienic measures. In the present author's experience, auto-vaccines are more effective in cases of older children than in infants. Hexamine may be tried for bacilluria.

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Aspects of Mental Disorder peculiar to Indian Conditions

BY

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I. Formulation.

The problem might well be split up into several sections.

(1) Whether the tropical conditions in India, function as the main aetiological factors, in the production of specific, exclusive, self-limited, mental disorders in the same sense, as the Cholera Vibrio causes Cholera.

(2) Whether due to tropical conditions, the course of a mental disorder is affected in any particular manner in its degree, intensity, duration, form, or content.

(3) Whether in the course of, or as a sequel to tropical diseases, (such as Malaria, Plague, Smallpox), mental abnormalities arise, and if so, what are the antecedent factors and are they diagnostic abnormalities? It is well known for example that in malaria typhoid, beri-beri etc., mental abnormalities are not infrequent. The question is, whether by merely observing the mental abnormality, can the physical disease be diagnosed? One recalls the famous case where Kraepelin is said to have diagnosed typhoid fever, from noting the abnormal behaviour of a patient, who did not show at that time any obvious physical signs or symptoms of typhoid.

(4) Whether due to the economic, and social conditions prevailing in India due in main to these tropical conditions, and in part to world stresses, and stresses incidental to contact with a foreign civilisation, aspects of mental disorder unknown in earlier periods are not thrown into prominence now?

The problem might further be complicated by the addition of some more clauses, but I think, these few are enough to show the importance of investigation on these lines; but from the nature of the problem, any unequivocal, conclusive answers are impossible now or at any time. It is essential to think not in set terms of Western text-books, but in terms of Indian conditions, and with the feeling that one is primarily an Indian. In investigating such problems, the choice lies between applying an arbitrary, hard and fast criterion of some diagnostic group, collecting a large number of such cases and seeing what becomes of them; and, on

the other hand, using a flexible diagnosis, taking a small number of such cases fully studied, and seeking to relate what one knows of their development prior to the illness and the phenomena of the illness itself to the later course of their lives. By the former method one obtains what Bleuler calls the "Richtungsprognose" or general drift of the illness; and by the latter one aims at discovering what details of form and development throw light on the individual prognosis. The former method has great advantages; it can be employed and controlled systematically, it *must* yield *some* results, and it permits of a statement of probabilities, at least for the group so defined. It is the method employed by Kraepelin. The other method is more in keeping with recent tendencies in psychiatry; it is concerned with the individual patient rather than with the fictitious disease or type and anything established by it could immediately be used in practice. But as a method, it is open to great objections; it is less controllable, its necessarily small material may be exceptional and, therefore, unsuited for clinical application; statistical treatment of it is difficult because of the smallness of the sample; it may be misused to confirm false impressions and prejudices, it requires much more time and its results may, after all, be nugatory. If, for example physiological or psychological phenomena *as yet undiscovered* are the most significant prognostically in these illnesses, immediate conclusions will not have much value. But no one can gainsay the scientific importance of such studies.

II. Preliminary Considerations

At this stage, some aspects of convergence and divergence of diseases of general medicine and of psychiatry might be summed up with profit. Every physiological or psychological process, normal or abnormal at any time is necessarily the result of inter-action of factors falling in four groups.

- (1) Tendencies inherent in the innate structure of the organism. (Heredity).
- (2) Tendencies associated with various ages, and physiological events and epochs. (eg: those of sex life).
- (3) Gross physical influences (injuries, inflammations, degenerations, new growths, deformities).

So long as the traceable causes of a mental disorder are limited to factors belonging to one, two, or all of the first three groups, so long is the disorder said to be 'functional' or 'psychogenic'.

- (4) A mental disorder is said to be 'Organic,' if, in addition to the above, or exclusively, the presence of a

physical pathogenic agent can be observed directly or can be deduced:

- (a) from changes of structure during life or death;
- (b) from anomalies of reaction such as fits or paralysis.

In psychiatry, however, one is not as a rule dealing with parasites, bacteria, or other exogenous agents which live and exert their influence in a definite order of time and place. Causes of mental illness have no set sequence or constancy. Those external to the patient are as variable and manifold as the pattern of daily life around us, and those intrinsic in him are seldom capable of forcing their way and becoming manifest as illness, no matter what befalls the patient. They are for the most part dependent on circumstance, and of their particular transmission and manifestation we know little: we infer hereditary and constitutional causes with good grounds, but of the details of heredity in any of the individual forms of mental illness we cannot be sure. Consequently we cannot find now any accuracy of prognosis, in the sense of predictable sequence in an individual, based on comparable experience of many others: the variations will be too many. We may expect such accuracy when we know more of the nature and mode of transmission, and of the influence of external circumstances upon time and form and degree of manifestation of each kind of mental illness. So it is usual to talk of mental disease, as reaction types, comparable to syndromes in general medicine, a group of signs and symptoms in a particular setting or constellation; (ataxia, paraplegia, aphasia, apraxia); the word 'disease' in general medicine on the contrary, is understood to mean, specifically a definite morbid process having a characteristic train of symptoms, affecting the whole body or its parts, and its aetiology, pathology, and prognosis are fairly well known.

I have found it necessary to digress somewhat in the above paragraphs, to make myself understood in the later sections; also to emphasise that on account of the complexity of the subject, no extravagant claims, or unequivocal statements can be sustained.

III. Case reports

The best approach to the problem is through the consideration of a concrete case.

S. R. male, 35, was referred by the D. M. O. Kurnool, to the Mental Hospital, Bangalore, for diagnosis and treatment. The complaints were:

- (1) Gradual, progressive depression leading to mutism and inertia, at the time of admission;
- (2) Progressive emaciation and weakness;
- (3) Generalised pigmentation, and a raw red tongue.

Duration—one year, since the first symptoms were noticed.

The patient being mute and inert, the history was obtained from his relatives. He belonged to a well-to-do, long-lived agricultural family. Father died young as the result of an accident, mother alive, 60, active and healthy. The patient, was the eldest of five children, (two sons and three daughters), alive and healthy. No history of any psychotic significance in the family, either on the mother's or on the father's side, or in the siblings.

Patient was a full term, healthy baby, who passed the milestones uneventfully and showed no neurotic traits during childhood. He studied up to Third Form, was more interested in practical and manual aspects of the subjects he studied, was a good mixer, and on leaving school at 17, went back to his land. He was married at 25, and had two healthy children at the time of admission. His marital life has been happy, economic condition good, and neither his wife, mother, nor any of his relatives are able to account for his depression. His prepsychotic personality was that of an active young man, interested in people rather than in books, an extravert, energetic, pleasant and good-humoured.

His medical attendant intimated that as there was a history of extra marital relations prior to marriage, a course of six 0.45, neosalvarsan injections was given before being sent to Bangalore, though his Wassermann was negative.

The following were the positive physical signs noticed during examination.

(1) Tall, lean, dehydrated (5 ft. and 10 inches. height, 98 lbs. weight.)

General weakness and exhaustion.

(2) Dull, lusterless, muddy conjunctiva.

Pigmentation.

(3) *Tongue.*—Intensely red, and glazed on the dorsum. But on the ventral aspect so heavily pigmented as to appear deep blue. When the tongue was put out, it had a bizarre effect. The whole of the nose, part of the forehead, (butterfly distribution of Pellagra??) wrist, elbows, ankles, dorsal surface of the toes and fingers, black with pigmentation. The nose looked almost leprosy. No pigmentation of the palms or soles, nor back.

(4) Prominent thyroid and exophthalmos. Pulse rate 120 regular and systolic murmur in the apex. Blood pressure, 110/75. The prominence of the thyroid was not due to generalized wasting, but to actual hyperplasia.—

(5) *Anaemia.*—R. B. C. Count...3½ million.

Haemoglobin ...70%

W. B. C. ... No abnormality.

Blood Picture.—Megaloblasts present, in addition to minor change of shape and size.

W. R. negative ... other body fluids normal.

(6) Alternate constipation and diarrhoea.

(7) No unequivocal neurological signs and symptoms.

Positive mental signs and symptoms. Depressed, mute, inert and negativistic, with spells of acute restlessness, anxiety and agitation—very slow in movement—sleeplessness, and had to be nasally fed.

Diagnosis.—The psychiatric picture was of acute depression of organic origin. The origin: The organic state suggested many possibilities.

- (1) Pellagra.
- (2) Pernicious anaemia.
- (3) Addison's disease.
- (4) Thyroid or other obscure endocrine dysfunction.
- (5) Arsenical dermatitis.

Many other rare possibilities could also be thought out. 3, 4, and 5 were ruled out because,

(a) Pigmentation was not typical of Addison's disease neither in its distribution, range nor nature. In this case, it was regular dermatitis.

(b) The degree of enlargement of thyroid, exophthalmos, pigmentation, dermatitis, anaemia, and mental picture could not all be covered by a diagnosis of Goitre. It was thought more probable that the same aetiological factors responsible for the general illness, were responsible also for dysfunction of the thyroid, and not the reverse.

(c) Arsenical dermatitis has only to be mentioned of the injections he had had and for nothing else.

It must be stated that a detailed investigation of his diet did not reveal any exclusive feeding on corn and maize, as Goldberger and others once thought was the main causative factor in Pellagra.

A provisional diagnosis of avitaminosis was made, and the following treatment was outlined. As the patient was resistive and had to be fed by the nasal tube, injections had to be preferred to oral medication. Daily routine during the first ten days:—

- (1) 9 A.M. I. M. Injection of 5 c. c. Campolan.
- (2) 11 A.M. Nasal feeding—1½ pints of milk, tomato juice and marmite.
- (3) 4 P.M. I. V. Injection of 20 grains of Sodium thio-sulphate in 10 c.c. water.
- (4) 5-30 P.M. Nasal feeding, same as at 11 A.M. except that orange juice was substituted for tomatoes.

Whenever the patient was found to have been restless during the day, medinal 5 grains was added to the evening feed.

- (5) 9 P.M. Coramine—2 c.c.

Locally, for the pigmented area dusting with Cuticura powder was found to give the greatest relief.

Clinical Course.—The patient improved rapidly under the above regime. The pigmentation disappeared in this order—toes,

knee, fingers, wrist, forehead, nose, tongue. It took more than two months before the tongue became red in colour both dorsally and ventrally.

With improvement in his condition the above intensive treatment was slackened. After 20 days, he began to feed by himself. The only solids he was allowed were wholemeal *chapathy* with butter, and vegetable salad.

After six weeks, he began talking in monosyllables, but relevantly; he was discharged recovered from the hospital, four months after admission. He came to see me last week (eight months after discharge) and I found him to be strong and sturdy, very pleasant and sociable. He has not yet been allowed rice or corn, but has been permitted to use ragi and wheat.

Last week, I saw in consultation with Dr. C. V. Natarajan, Chemical Examiner to the Government of Mysore, a Jain patient, (45) male, a strict vegetarian, and a permanent resident of Bangalore, who exhibited practically the same clinical picture—acute depression, pigmentation, dermatitis, anaemia and weakness. Dr. Natarajan also mentioned that he had occasionally come across cases of this type, answering to the clinical description of Pellagra, but that the Editors of the semi-official medical journal in India were very sceptical of his views.

IV. Discussion

These cases illustrate what I have in mind, when I say "Aspects of mental disorder peculiar to Indian condition." The points I would like to invite attention to, are:

- (1) Anaemia—simulating pernicious anaemia.
- (2) Pigmentation.
- (3) Prominent thyroid.
- (4) Mental picture.

One is justified in diagnosing the case either pernicious anaemia, pellagra, or a typical form of goitre. Goldberger stated lately in connection with pellagra that, "The disease is dependent on some yet undetermined fault in the diet, in which the animal and leguminous protein component is disproportionately small, and the non-leguminous vegetable component disproportionately large."

McCarrison has frequently emphasised the importance of avitaminosis in the production of goitrous conditions.

The association of pernicious anaemia-like, goitre-like, and pellagra-like syndromes throw an interesting light on the artificiality of classifications, namely separations of associated similars, and grouping of unassociated similars. It is generally agreed that the lack of Castle's factor (avitamin B complex) elaborated by the mucous membrane of the stomach and stored in the liver, is responsible for the syndrome of pernicious anaemia, in all essentials a deficiency disease—goitre-like states are often due to avitaminosis. Pellagra is definitely due to an ill balanced diet. I have also been often struck by the fact that anaemias are often associated with neuritis, and muscular atrophy, of the type

described in beri-beri. They all do well on intensive vitamin therapy. Of course from the psychiatric point of view, I cannot dogmatise, because only those cases of the group mentioned which show mental symptoms, are brought under my notice. I must qualify it by saying "severe mental symptoms." I have described in a previous article that, where the physical insult to the organism is severe, the mental picture is that of delirium. In a less severe state, fear and delusional states are about equal. Not so rarely is avitaminosis, the starting point of a permanent psychosis of any type, dependent on the physique and previous personality of the individual.

For us Indians, that mental disorder is so commonly associated with such physical syndromes is significant. What we need in the prophylaxis of mental disorder, is not huge mental hospitals, nor intensive psycho-analysis, nor eugenics, but food.

There is another phenomenon which is quite interesting. Probably due to reasons as much psychological as physical, the intensity of maniacal states is much more severe in India than one notices in the West. I have so often noticed it, and some of my medical colleagues have also confirmed the same, that I do not think it is merely a chance observation. It is usual to treat maniacs in Europe, in open wards, but it can never be possible in India. That imbibitions are much more intensive in an average Indian due to his philosophic background, which breaks down under severe stress with proportionally more intensive reaction is a theory, which merely shelves the question.

The only disease directly attributable to tropical conditions is what has been termed tropical neurasthenia. Col. Chopra, in his "Hand-book of Tropical Therapeutics" has described the condition in detail. I have not had much experience with this disorder.

V. Germ Plasm and Environment

A study of the toxic influences on germinal cells has gradually led scientists to believe that "germinal cell can be injured, and this injury is inheritable" (Law of Blastophoria). The plasmic environment of the germ cells cannot of course vary excessively without death resulting, but within the limits which enable life to continue, the action of disease, degeneration and pathogenic agencies have full play, the growing and maturing sexual cells in the ovaries and testes are affected by noxious as well as by beneficial agencies, by changes in food supply; warmth, oxygen, and all the normal necessities of life, as well as by inanition, pyrexia, impure gases in the blood, toxic and cytolytic substances in the blood in fevers, by toxins of defective bowel and renal elimination, by alcohol, food poisoning and the thousand and one factors which assail and affect the human constitution. Is it not a pertinent question to ask, that this lack of proper food for generations, in South Indians specially has not also influenced them mentally, (that it physically has, is evident), during normal

as well as in psychotic states? Do we not carry with us the blemishes of such heritage? It is noticed for example that in one family, a brother might be suffering from achlorhydria, a sister from glossitis, and another brother, from pernicious anaemia. Same type of inherited tissue defects determines an abnormal susceptibility to tuberculosis in one child and schizophrenia in another. Both the disorders are common in India. To my mind the acutely maniacal conditions in India, far more common than in the west, may have a similar relationship with inherited consequences of miserable feeding. It is only pure surmise, but it is a line of investigation worth following.

VI. Malaria and Atebrin

Mental symptoms in the cause of malaria are fairly common. But with the introduction of atebrin, a new significance attaches to this problem. Occasional cases have been described where the mental symptoms have been noticed after the administration of atebrin. I have described one such.

"The patient, a flabby male aged 32, was referred on November, 19th, 1935, by his medical attendant for protective observation because he showed acute maniacal symptoms. He had been having daily attacks of malaria for the past three weeks, and quinine administered; during the eight days before admission he had two pills of atebrin daily, and three injections of atebrin mussonate. Fever had subsided, but 24 hours before admission he had become destructive and violent.

Physical examination.—The patient was very restless, throwing about bed-clothes, and had to be restrained. He was deeply jaundiced. The liver and spleen were not palpable, and there was no evidence of hepatic pain; the tongue was thickly coated and the breath foul. The pulse-rate was rapid, 120 per minute, of low tension, irregular, missing one in every 10-12 beats. The apex-beat was within the nipple line, but a soft uncondensed, systolic murmur replaced the first sound in the apical and pulmonary areas. The pupils were moderately dilated and reactive, but there were coarse, rapid, nystagmoid jerks, about 16 per minute, making it almost impossible for the patient to fixate. He had fine tremors of the tongue and hands. Speech was slow and hesitating, but there was no dysphasia. The reflexes were sluggish except the knee-jerk which was No Babinski sign.

Mental examination.—Restless: psychomotor activity increased of both the small and large joints; disorientated with reference to time and space; he would shout that snakes and fantastic animals were crawling on the floor, and reacted to such visual hallucinations with fear, violence, and emotional instability.

Laboratory findings.—No malarial parasites found. Urine scanty reaction acid, no sugar or albumin found; no bile pigments or atebrin were present. Faeces foul-smelling, but yellow and not clay-coloured.

Progress and Treatment. The patient was given immediately a magnesium sulphate enema, and on account of his restless and non-cooperative state, a paraldehyde enema. He passed a quieter night. He was put on daily injections of cyclotropin (five days) and strychnine gr. 1/20 b.d., and general treatment like daily warm sponging. His mental symptoms cleared up within three days and his jaundice on the seventh day. On November, 29th, 11 days after admission, he was discharged recovered, though somewhat weak.

The Dutch have had a monopoly of Quinine, and it is to their monetary interests that no other anti-malarial drug is put upon the market. Apart from this, in a malaria ridden country like India, every anti-malarial drug must be thoroughly investigated. Acting upon the request of Dr. Urchs of Bayers, I have been engaged for the last one year on the neurological and psychiatric pharmacology of atabrin. The results will be published in due course. But in general it has been found, that:

- (1) Atabrin in general is a safe, harmless, effective drug to administer.
- (2) For the same dosage, and in the same group, the lean asthenics, stand it better than the thickset pyknics.
- (3) One has to be careful in administering atabrin to alcoholics, and arteriosclerotics, who do not stand it well.

Occasionally, one comes across individuals who are sensitive to 'atabrin' and a psychosis is the result. But I suppose, people are found who are sensitive to anything in this world. In such cases when atabrin is being administered the psychosis is definitely the result of atabrin, and not due to malaria.

So, for one practising psychiatry in India, such information is valuable and can safely be included under the comprehensive title selected for the article.

Conclusion

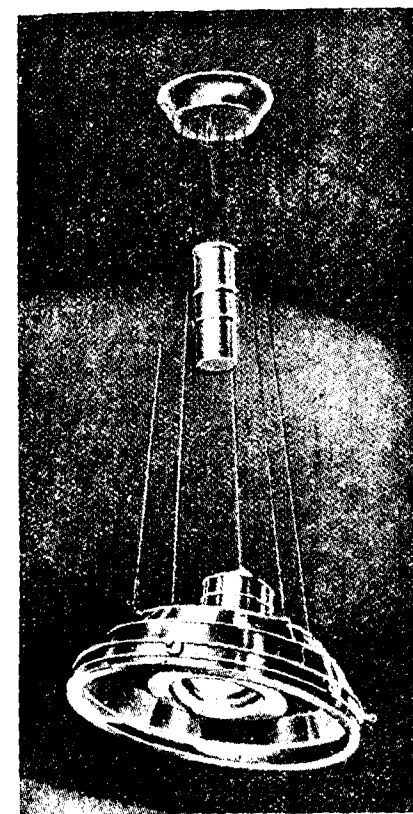
The paper has been much longer than it was intended to be. Hence the purely psychogenic aspects will have to be deferred to a later article. If I have really succeeded in showing that there are aspects of Indian psychiatry worth investigation my task has not been quite fruitless.

Finally, it must be realised that whether in functional or organic psychosis, mental syndromes such as depression, anxiety state, hysterical or paranoid reactions, are abstractions of a simplicity which is always far in excess of clinical reality.

The development of a competent psychiatrist consists mainly in the slow evolution of an adequate sense:

- (1) of the futility of looking for single causes, solitary syndromes, standard courses or specific courses.
- (2) of the double futility of trying to propound constant correlations between any two of these abstractions. (Mapother).

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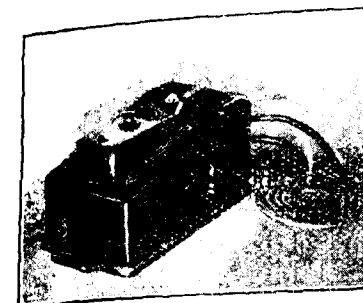
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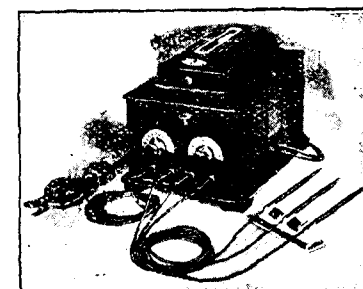


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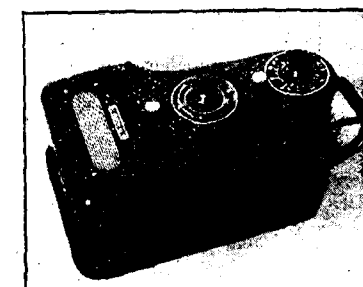
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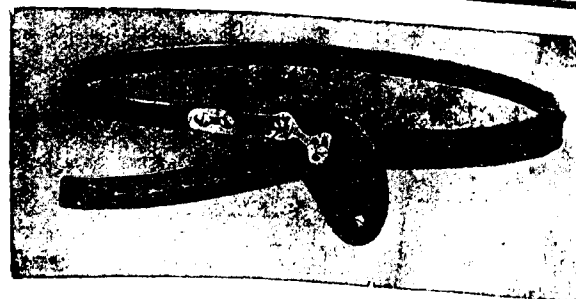
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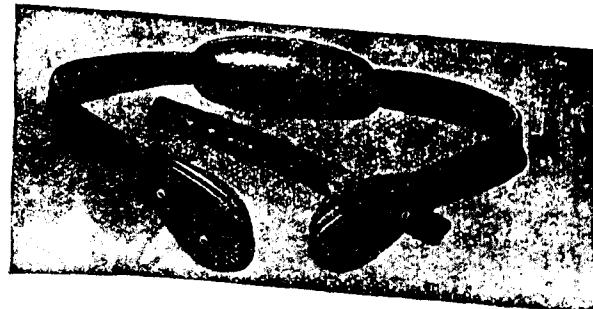
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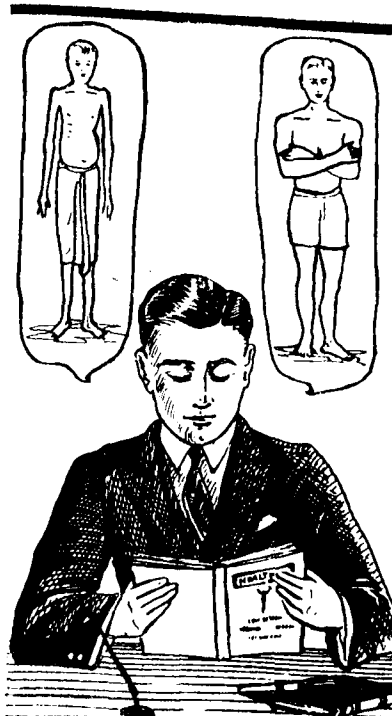
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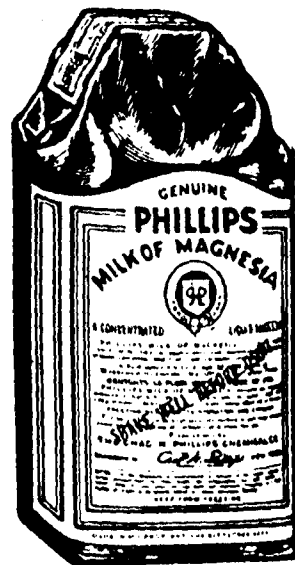
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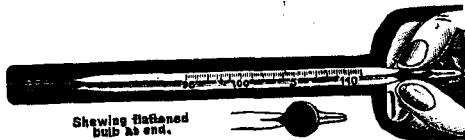
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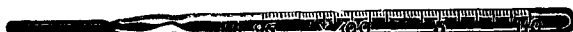


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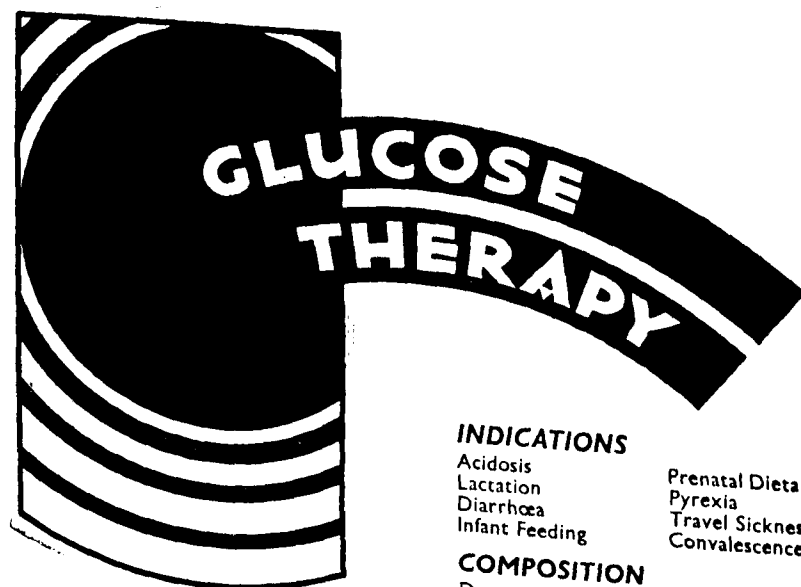
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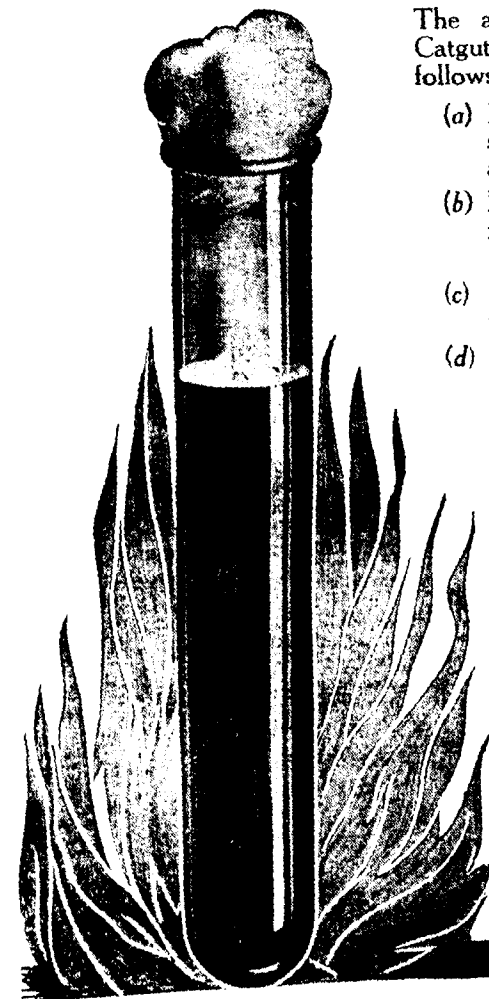
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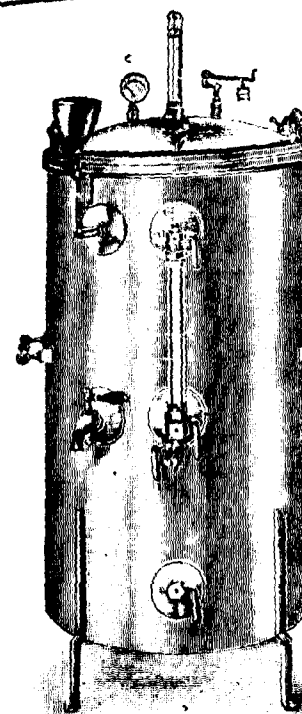
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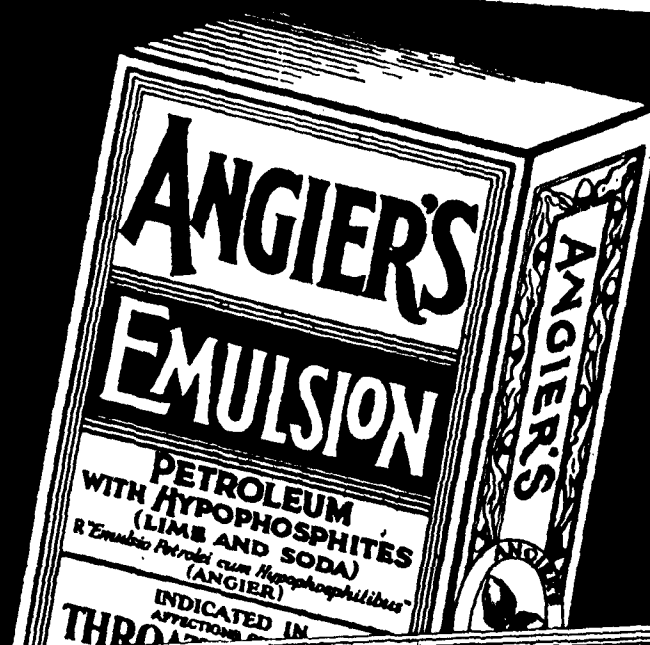
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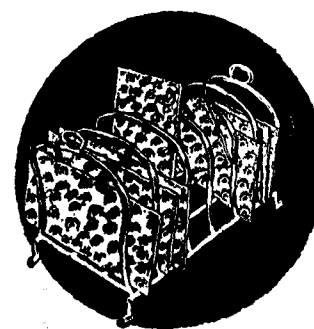
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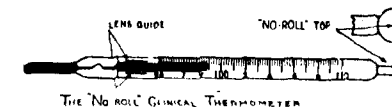


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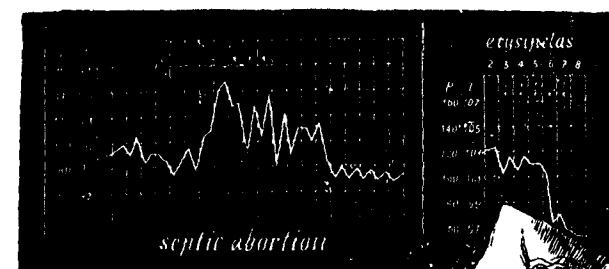
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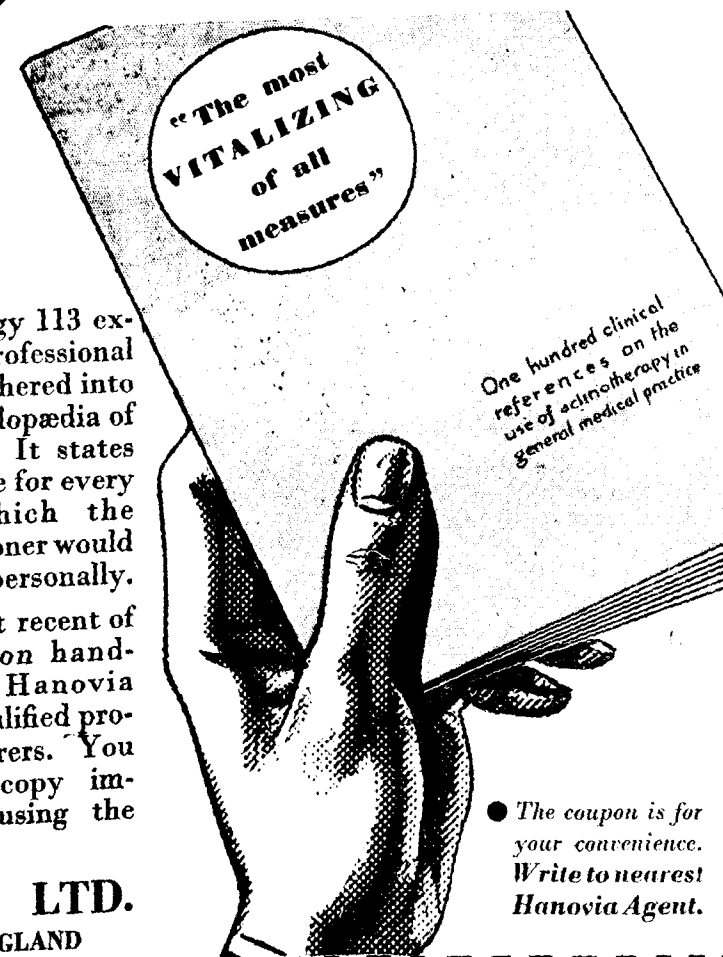
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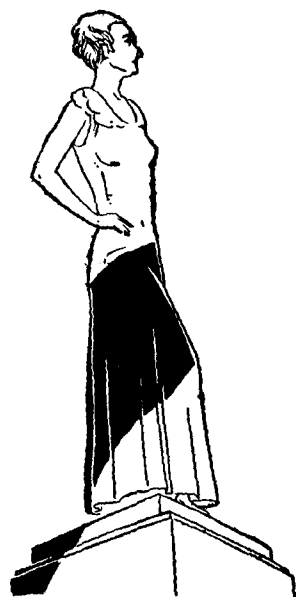
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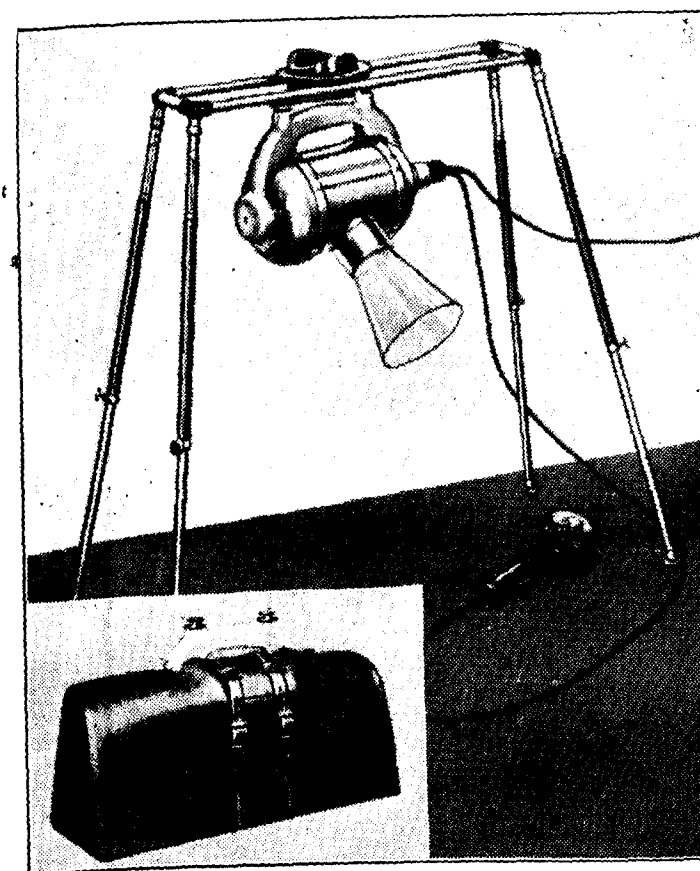
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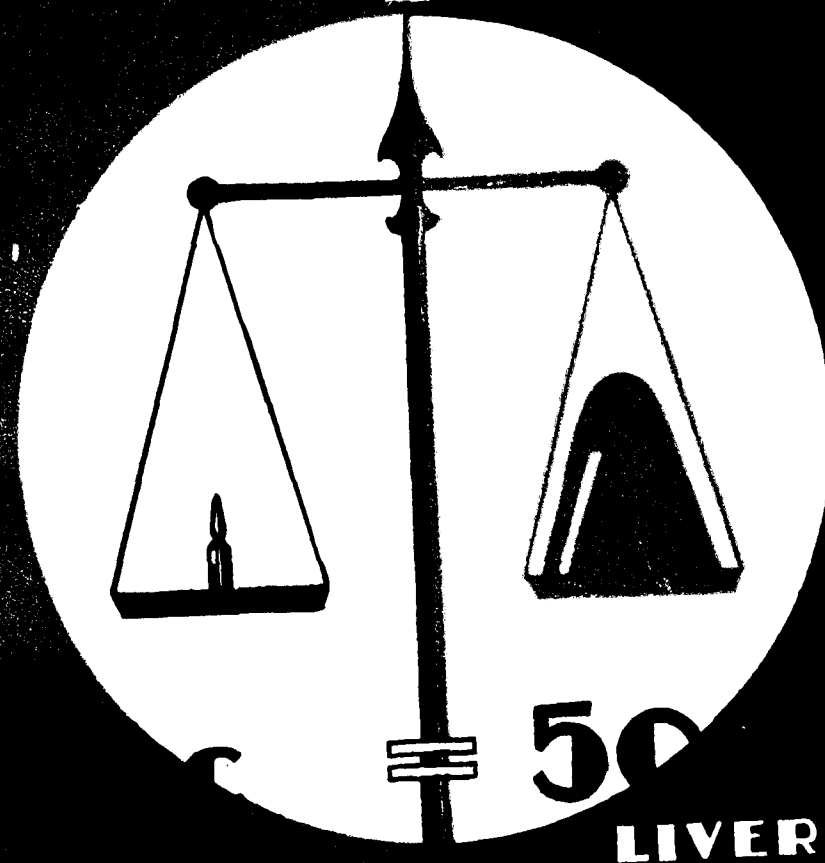
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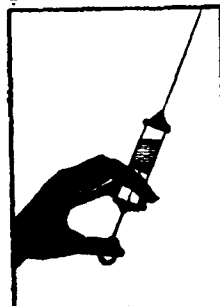
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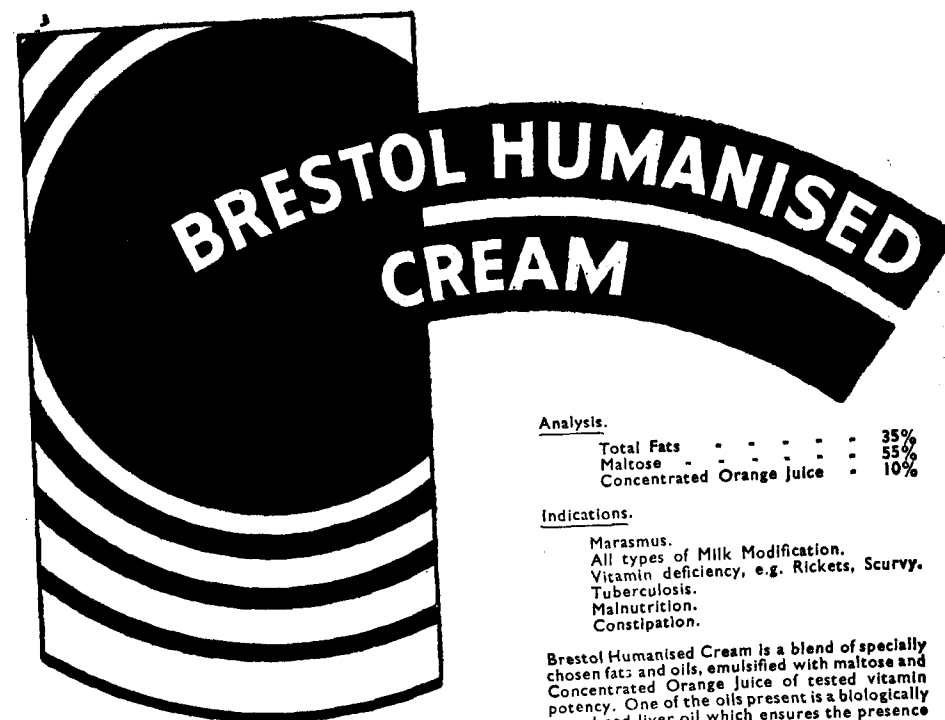
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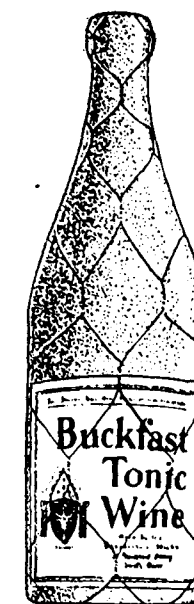
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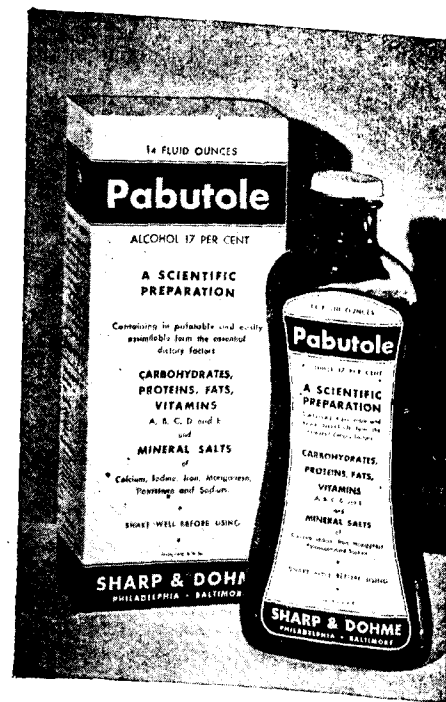
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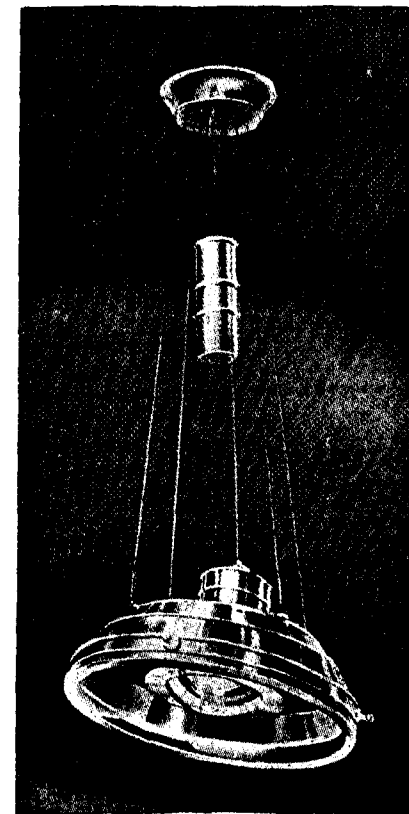
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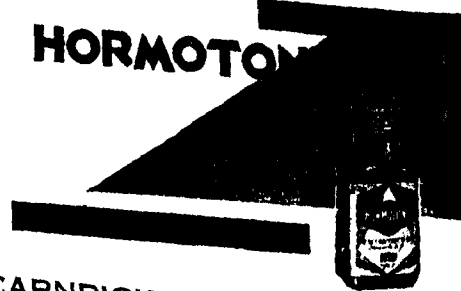
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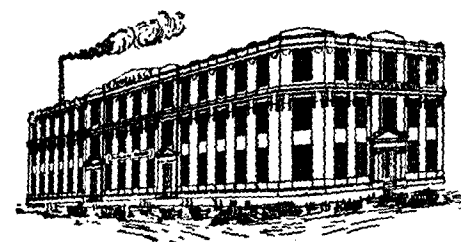
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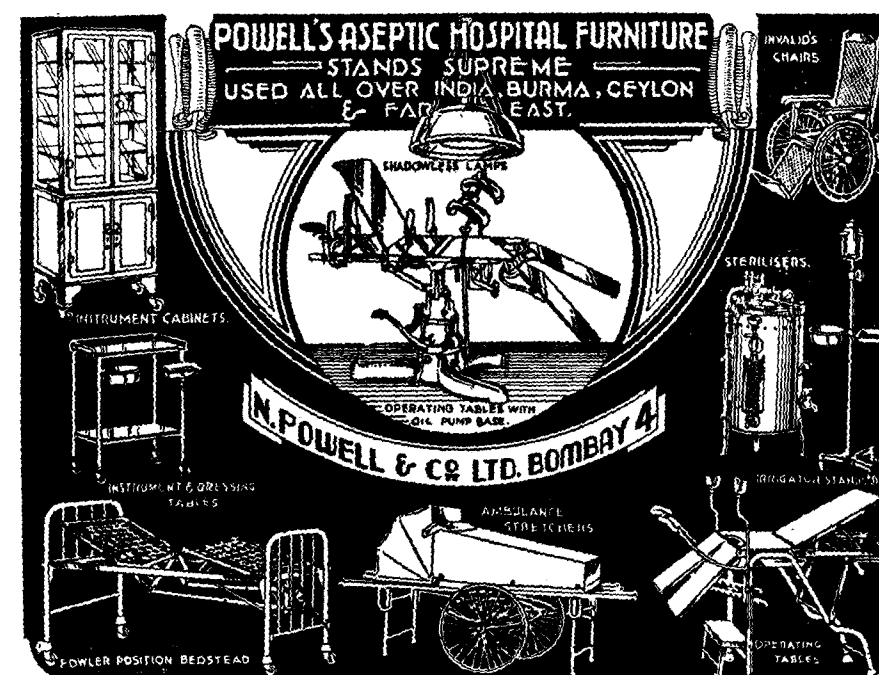
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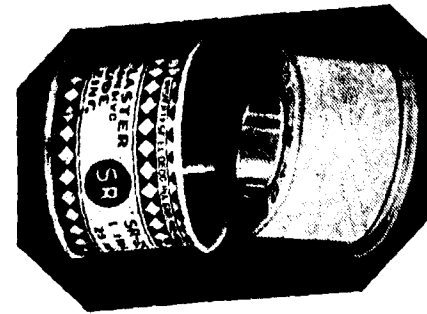
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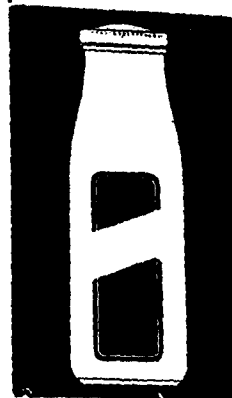
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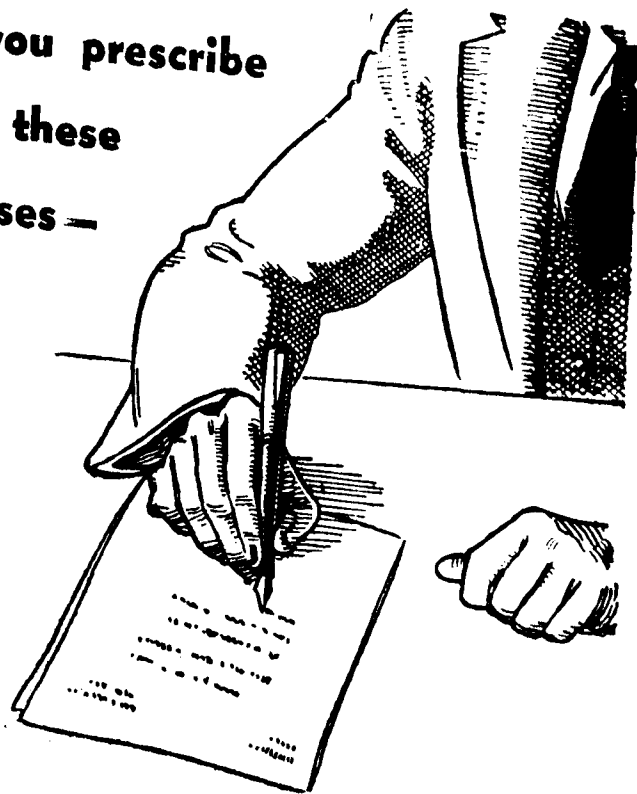
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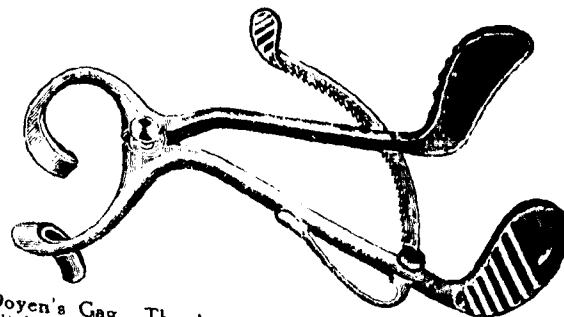
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Fig. 5577. Tonsil Guillotine, Mackenzie's, the same as Beehag's, sharp, three sizes, $\frac{1}{2}$, $\frac{3}{4}$ and 1 inch, with reversible metal handle, steel nickel plated—to use on both sides with right hand. Rs. 21-0.

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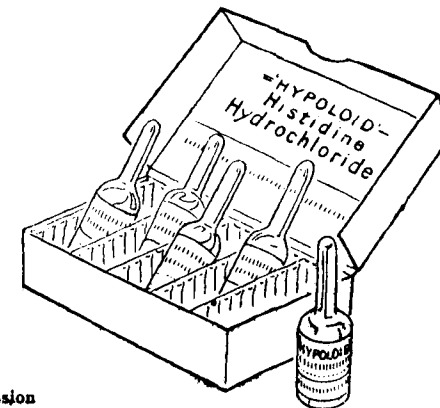
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APRIL 1937

No. 4.

ORIGINAL ARTICLES

Food Poisoning*

BY

Lt. Col. B. G. Mallya, M.D.F.R.C.S.E., L.R.C.P., I.M.S.,
 Civil Surgeon, Howrah

AND

Dr. S. Chakraborty, M.B.,

House Physician, Howrah General Hospital.

CASES of food poisoning occur in large numbers and are reported more often in the general press than in the medical journals. The reason for this is obvious. A group of persons partaking of the same food or drink is affected at the same time and the incident is promptly reported in the press. Medico-legally, food poisoning may be suicidal, homicidal or accidental. What we are concerned with here is accidental food poisoning. Tinned meat and fish accounted for a large number of cases of food poisoning and it was formerly thought that the poisoning was due to the products of protein decomposition and the term "ptomain poisoning" was erroneously used. It is now generally accepted that the poisoning is due to the action of micro-organisms or their toxins.

Poisoning may occur as a result of ingestion of meat, fish, cheese, shell-fish, sausages and mushrooms. Some persons are

* Specially contributed to the "Antiseptic".

liable to develop allergic symptoms from eating fish, eggs etc. This condition is known as food idiosyncrasy. Food poisoning should suggest itself in a case where there is sudden onset of vomiting, abdominal pain and diarrhoea shortly after taking food. All those who have partaken of the same food suffer from food poisoning but the severity of the symptoms may vary. In cases where the action is on the gastro-intestinal tract, the causative agent is a micro-organism. Toxins act on the brain and spinal cord and produce visual disturbances and muscular paresis. In cases of allergy, the symptoms are vomiting, urticarial rash, dyspnoea and restlessness.

As a rule, food poisoning may be diagnosed by the presence of symptoms which suggest either a bacillary infection or action of a toxin. Bacillary infections produce symptoms of acute gastro-enteritis and collapse within 1 to 3 days. Toxins act on the nervous system and produce paralysis and the action of toxins is immediate.

Treatment of the former class of cases consists of clearing of the intestinal tract and administering stimulants for collapse. For the latter class of cases, anti-toxic serum administration is required.

Four seamen were admitted into the Howrah General Hospital with loss of sensation and power in the lower limbs as well as the distal parts of the upper limbs. On enquiry, they said that while their boat was in Port Said, they had their usual evening meal at five o'clock and about six o'clock some developed vomiting within an hour of partaking the meal. There were twenty six of them who received food from the same kitchen and all of them suffered from acute gastro-enteritis. There was only a variation in the time of onset of symptoms. There were no deaths. Most of them recovered fully in due course and on the arrival of the boat in Calcutta three weeks later, some went to their homes and four were admitted to the Howrah General Hospital.

None of them had any temperature during their prolonged stay in Hospital. One of them had pleomorphic vesicular eruptions all over his body. Anaesthesia of the lower limbs passed off gradually and earlier than the hyperaesthesia of the limbs and the pain in the limbs was so severe that large doses of sleeping draughts did not give them any relief. The loss of power in the lower limb was more marked than in the upper limbs. Knee and ankle jerks were absent. Progress was slow but steady.

One of the patients said that the bhandari (cook) mixed some poison in the food. This is imagination as the bhandari was one of our patients and would have taken care not to poison himself.

At Port Said, camel's meat is issued to seamen. The question is if meat poisoning causes acute gastro-intestinal as well as nervous symptoms.

Available evidence is against meat being the cause of this food poisoning. What is the causative agent then? Some physicians with whom these cases were discussed hold the view that these are cases of metallic poisoning and arsenic is the one that does cause acute gastro-enteritis and loss of power and sensation of the limbs a fortnight later. As the cook is in the hospital along with the others, he could not have in any way been responsible for poisoning the food. Some other person might have done so to get the cook into trouble. This mystery could have been cleared by an analysis of the vomit. Thus arsenical poisoning must remain as a possibility. The points in favour of arsenical poisoning are: the onset of acute gastro-intestinal symptoms soon after taking food, followed by loss of power and sensation in the limbs, hyperkeratosis of the skin, of the palms, of the hands, and the soles of the feet.

Case Reports.—1. M. H—male, aged 26, admitted on the 26-8-36, to the Howrah General Hospital with a history of sudden attack of vomiting and diarrhoea along with slight rise of temperature after his evening meal about three weeks before admission. This continued for seven days. Twelve days later, he felt tingling and pricking sensations in the extremities and a pain in the throat.

On examination.—Pupils were normal. Gait was slightly ataxic, voluntary movements were present. Muscle sense was absent in both the extremities, markedly on the left side. No sensation of light touch and pin prick over both the extremities.

Reflexes.—Triceps, Biceps, Supinator—present: Knee and ankle jerks—absent; Plantar reflexes—flexor response. Abdominal reflexes—present; Albumin in urine—present. Wassermann—negative. Blood count nothing abnormal. Stool examination report—nothing abnormal.

9-9-36.—Patient says that the tingling and pricking sensations are gradually diminishing.

Dizziness on standing.—Reflexes same. Diminished sensation of light touch and pin prick of both the extremities. Loss of sense of position of the left foot.

24-9-36.—Patient says that the sensations are more marked in the hands and feet than other parts of the limbs.

Sensation present in the limbs except over hands and feet. Patient left hospital on 30-9-36.

CASE 2. A. G., male, aged 28, admitted on 26-8-36, to the Howrah General Hospital with a history of sudden attack of diarrhoea and vomiting with high fever after his evening meal about three weeks before admission. The vomiting subsided after three days but the fever continued for seven days. Later he noticed gradual loss of sensation and power in his limbs. In addition he had a skin eruption all over his body.

On examination.—Pleomorphic reddish brown nodular vesicles all over the body more marked on the face and limbs. Pin prick and heat sensation lost all over the body. Patient unable to stand up and could sit down with difficulty.

Reflexes.—Knee and ankle jerks—absent; Triceps, Biceps, Supinator and Abdominal, — present; Plantar reflex — flexor response. Wassermann—negative. Stool, urine and blood examination—Nothing abnormal.

11-9-36. Patient can sit down. Dizziness on standing; reflexes same.

Diminished sensations of pin-prick over the terminal portions of both hands and feet.

Loss of sense of position in both hands and feet. Skin eruptions disappeared.

24-9-36. Sensations present except in hands and feet. Patient discharged on 3-11-36.

CASE 3.—B. Meah, male aged 25, was admitted on 26-8-36, in the Howrah General Hospital with a history of sudden attack of vomiting and diarrhoea about three weeks before admission after taking his evening meals. This attack lasted for three days. Seven days later he felt burning sensation over his upper and lower extremities below his elbow and knee.

On examination.—Voluntary movements present. Dizziness on standing. Diminished loss of sensation, in the limbs below his elbow and knee.

Reflexes.—Knee and ankle jerks absent. Gums swollen. Teeth—unhealthy. Tongue—slightly coated. Sub maxillary glands enlarged. Few dark spots each about the size of a pea present in the palms and soles. Stool examination report: Few ova of *Ankylostoma Duodenali* present. Wassermann—negative. Urine and blood count examination report—Nothing abnormal.

9-9-36. Patient complains of tingling sensation below ankle and wrist. Diminished loss of sensation (pin prick) in the palms and soles.

Reflexes.—Knee and ankle jerks absent. Patient left hospital on 24-9-36.

CASE 4.—A. Haque (cook, male aged 35, admitted on 26-8-36, in the Howrah General Hospital with a history of sudden attack of burning sensation in the extremities about three weeks before admission, four days later he noticed reddening of the ankles. 26 members of the ship suffered from diarrhoea (patient says) all of a sudden after taking their evening meals, about three weeks before admission.

On examination.—Patient could sit down but unable to stand; Oedema of legs present. Voluntary movements not impaired; Knee and ankle jerks absent; Pin-prick sensation present; Urine—Albumin present. Wassermann—moderately positive. Blood count and stool examination report—nothing abnormal.

10-9-36. Patient complains of tingling sensations in the limbs below his elbow and knee.

Oedema of legs disappeared. Dizziness on standing.

Reflexes—Same. Pin-prick sensation present.

20-9-36. Patient says that the tingling sensations are disappearing. Patient left hospital on 26-9-36.

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—*The Eye, Ear, Nose and Throat Monthly.*

Diet in Pregnancy*

A General Survey of the Deficiencies in India and their Effects

BY

Capt. P. C. Dutta, M.B., (Cal.), F.R.C.S., (Edin.),
D.G.O., (Dub.), I.M.S.,

Civil Surgeon, Shahpur, The Punjab.

ON the health of the expectant mother depends the health of the child and consequently that of the whole nation in the long run. If she is ailing or does not get proper nourishment for herself and the growing foetus, the child will be unhealthy and the stigma may stick to him for the rest of his life. Every parent is responsible for the children he begets; bringing into the world an unhealthy, puny and miserable creature, who, from its very birth, is unfit for the struggle for existence, is a crime. Malnutrition in pregnancy is one of the chief causes of defective physical development and infant mortality in India. If the child does not die in infancy, he grows up a weakling and is a liability to the family and nation.

The question of diet is complicated in this country by age-long prejudice, social customs and illiteracy. The older generation believes in what has been the custom from time immemorial (forgetting that the conditions under which they are living now are totally different), despises anything that they did not do, while the (half-educated) would-be young mother considers herself too ultra-modern to live on a conservative and nutritious diet.

The diet of the expectant mother is not a matter for which hard and fast rules can be laid down. All that is required is that she should get a good mixed diet consisting of fresh foods, mostly carbohydrates, low protein and low fat. It has been rightly said that the prevention and treatment of diseases in pregnancy are to be found rather in the larder and kitchen than in the pharmacy. Expensive preparations will be unnecessary if the diet were mixed and adequate.

It must be realised that the deficiencies if they are not too acute, will produce only a latent effect. Nothing grossly abnormal may be detected but all the same it will hinder the proper development of the foetus and the child. The resistance to infection is lowered and the power to combat with adverse circumstances is lessened. It may cause general debility and other vague

* Specially contributed to the "Antiseptic".

symptoms. Only when the deficiency is beyond a certain limit, do we get acute symptoms *e.g.*, osteomalacia and pernicious anaemia in the mother and rickets in the child.

The fundamental difference between the deficiency symptoms occurring in the pregnant and non pregnant states is the relatively rapid onset of disturbances in the former. The foetus is a parasite. Like all parasites it does not care what happens to the host as long as it gets its own share. When the latter cannot supply these from her reserve store, it robs the mother of its most vital substances and naturally her system is depleted of these.

Carbohydrates.—These are essential for nutrition, neutralisation of the fatty acids, maintenance of the normal alkali reserve and foetal growth. The average Indian diet contains more than enough of this element. But it has to be pointed out that the source of the carbohydrate is cereals in this country. It has been shown that the cereals have an antagonistic effect on calcium absorption. Calcium deficiency is very common in pregnancy in India and this in my opinion is due mainly to this excessive amount of cereals in our food. So the cereal part of the diet should be cut down by half in pregnancy and replaced by carbohydrates from other sources, *e.g.*, tubers, roots, sweets, etc.

Proteins.—An excessive intake of proteins throws extra strain on the kidneys and is liable to lead to toxæmia, so the proportion should be low. But at the same time there is nothing to show that a moderate amount is more liable to produce toxæmia in a pregnant woman with healthy kidney. Moreover it is now believed by a number of authorities that toxæmia of pregnancy is a deficiency of iron, iodine, calcium and vitamins A, B, and D, which produces hepatic dysfunction leading to toxæmia. The average Hindu woman takes very little meat, some of them are total vegetarians, but there is nothing to show that the Hindus suffer less from toxæmia than the Mahomedans.

Fats.—Animal fat is the natural source of vitamin A supply. The proportion of fat should be low in pregnancy. Rich and highly spiced food should be avoided.

Vitamins.—*Vitamin A.*—The chief sources of vitamin A are milk, butter, cream, and animal fats. It is abundant in cod and halibut liver oils. It is present in the embryo of green vegetables. It is absent in vegetable oils—simple process like pressure fails to extract the vitamin from the seeds.

Deficiency of this vitamin reduces the resistance to infection. It is essential for proper growth and health. The demand for this vitamin is increased during pregnancy. Vitamin A reserve

in a woman following pregnancy is very often lower than the normal individual. If a woman has a liberal supply of this vitamin during pregnancy, her resistance to infection in puerperium is increased. In this country where the average doctor has to work under conditions far from ideal, he would do well not only to look to her diet but also prescribe one of the many proprietary vitamin A and D preparations available in the market.

Vitamin B.—This is present in most of the natural food stuffs *e.g.*, eggs, pulses (seeds), cereals (germ and bran), yeast. During pregnancy, the vitamin B content in the system is markedly diminished, specially in the lung, liver and brain, due to increasing consumption of the vitamin by the foetus. An even greater diminution is found during puerperium. The mammary glands during pregnancy and puerperium contain an increased supply of the vitamin.

Polyneuritis or beri beri syndrome is quite common in pregnancy in this country. This is usually due to vitamin B deficiency. Cases of polyneuritis during pregnancy has lately been reported from other countries where beri beri is absent. Symptoms in the milder cases are, tingling and numbness of the fingers, gnawing pains along the arm specially at night. In more severe cases symptoms of paresis and later on anaesthesia or hyperaesthesia may be present. The symptoms generally appear in the later months of pregnancy.

Deficiency of vitamin B complex is responsible for the megalocytic anaemia or pernicious anaemia of pregnancy which is very prevalent in this country. This will be more fully described later.

Vitamin C.—Deficiency of this vitamin does not occur during pregnancy. It may be that the growing foetus needs very little of this; so there is no unusually increased drain on the maternal system.

Vitamin D.—This is present in fish liver oils, yolk of egg, milk and butter. Ergosterol which is normally present in the skin is converted to vitamin D when the latter is exposed to sun's rays. This vitamin is essential for the absorption and utilisation of calcium by the system. So lack of this vitamin will cause calcium deficiency. This is more fully discussed later.

Vitamin E.—It is present in wheat germ oil, olive oil etc. There is ample supply of this vitamin in our ordinary diet. Deficiency of vitamin E produces sterility, and in pregnancy will cause abortion.

Calcium. Its role in pregnancy.—It is required for the growth of foetus. Mother stores an excessive amount of calcium in her system for the foetus. For the efficient storage of calcium not only an ample supply of calcium and phosphorus is required in the diet but also vitamin D which is essential for the absorption and utilisation of calcium by the system.

If the storage of calcium in the maternal system is insufficient, the foetus draws the same from the tissues of the mother and the latter suffers from the effects of calcium deficiency. The symptoms of deficiency will depend on the amount of calcium depleted from her system. In minor degrees it may cause only flabbiness of the muscles specially of the uterus (uterine inertia) and heart (tachycardia) and vague pains and aches in the body. In more severe cases, tetany and cramps, and in the most serious cases, osteomalacia may be caused.

The child is also liable to suffer from deficiency of calcium. This may cause malnutrition or defective dentition and in the more serious cases, rickets.

Minor degrees of osteomalacia is very common in pregnancy in India. It is also common among Indians living outside India, in places where it is very rare among the indigenous people. So it is fair to conclude that there is some defect in our diet which is responsible for this. Three factors are mainly to be blamed for this deficiency.

I. INSUFFICIENCY OF CALCIUM AND PHOSPHORUS SUPPLY:

Milk is the best natural source. It is usually the only source in most Hindu households. In a big joint family, the young would-be mother hardly gets any. In towns it is expensive. In England and other countries when they are having milk bars all over the country, it is considered to be a very timid drink for the well-to-do in this country.

2. *Deficiency of the vitamin D.*—A certain amount of the vitamin is present in milk, but the main source is from ergosterol present in the skin which is converted into vitamin D by the action of sun's rays on the skin. As has already been said, the amount of milk ingested is very small so the main source of supply is from the skin. In a 'Purdah-ridden' country like this, women very seldom get the benefit of sunshine. In big cities it is worse. In villages like those of the Punjab, where a village consists of a mass of houses, one stuck to the other, seldom do the women get any sunshine,

3. *Effect of cereal food.*—Cereals form the main element of our food. It has been shown that they have an antagonistic action to vitamin D. Rickets can be precipitated by increasing the cereal content in the diet of a young animal who is on a minimum vitamin D diet. The diet of all Indians, in or out of India, rich or poor, and that of the Chinese has an exceedingly highly cereal content and these are the two countries where osteomalacia is most common, though they get plenty of sunshine. This ingredient of food is also cheap, so the hungry poor has mainly to depend on this to sustain himself. Therefore to avoid calcium deficiency in our diet which contains a barely minimum vitamin D and calcium it is essential that the amount of cereals should be cut down by half and replaced by carbohydrates from other sources, e.g., tubers, roots, sweets etc.

Anaemia of Pregnancy.—The commonest types of anaemia associated with pregnancy are due to deficiency in certain constituents of food which are essential for the regeneration of blood. Four types of anaemia are generally associated with pregnancy.

I. DEFICIENCY OF IRON IN FOOD :

(1) In minor degrees of deficiency, the woman develops anaemia due to lack of iron, manganese and copper in the food. It is relieved by putting the patient on a diet that contains enough of the above elements. Iron is present in egg, and all green vegetables, specially spinach. (Palak.)

2. In major degrees of deficiency, the woman was probably suffering from a low grade iron deficiency anaemia before pregnancy and this is aggravated by the strain of pregnancy. The foetus also needs iron for its blood formation. Deficiency of iron in pregnancy is one of the main causes of nutritional anaemia in infants. In these cases the mother develops hypochromic anaemia. They will need massive doses of iron e.g., Ferri et Ammon. Cit. gr. 30 T. D. S. or Bland's Mass gr. 15, six times a day as well as food which contains a generous amount of iron.

II. DEFICIENCY OF VITAMIN B COMPLEX.

Castle's extrinsic factor, which is supposed to be a certain type of vitamin B, is essential for the formation of red blood corpuscles.

This is the most serious type of anaemia associated with pregnancy in this country and unfortunately it is quite common. Oedema and albuminuria are often present. Megalocytes are always present in the blood and the colour index is above one.

The condition generally proves fatal if not recognised early and efficiently treated. Dr. Lucy Wills has shown that many of these cases react excellently to liver and ventriculin treatment on the intrinsic principle of Castle, while others fail to do so because they lack that extrinsic principle (vitamin B complex) in their diet which stimulates reticulocytic response. This can be supplied by administration of Marmite $\frac{1}{2}$ to 2 ounces per day in addition to the usual dietetic measures. Small amounts of repeated blood transfusions will help.

III. MYCROCYTIC ANAEMIA.

This type of anaemia, although not caused by dietetic deficiency is truly a deficiency disease. The deficiency in these cases are due to hypochlorhydria or achlorhydria of the gastric juice which is responsible for defective blood formation. The symptoms are aggravated by the foetus robbing the mother of the haemoglobin forming materials for its own use. Blood picture shows hypochromia with microcytosis, normoblasts may be present but megalocytes are absent. Treatment with liver, iron and hydrochloric acid gives satisfactory results.

Iodine.—Deficiency of this lowers the metabolic rate. Cases of sterility and habitual abortion are occasionally benefited by small doses of iodine. We do not know the exact mechanism of this. It may be due to stimulation of thyroid which speeds up every gland of internal secretion, particularly the endometrium and chorion and these are thereby enabled to filter or neutralise the effects of toxins or secretions which disturb carbohydrate metabolism. Deficiency of iodine also causes diminished resistance to infection. Sea fish is one of the important sources of iodine.

Summary.—1. Calcium deficiency is one of the most important factors in the usual Indian Diet.

2. Anaemia in pregnancy is mainly due to deficiency of iron and vitamin B complex in our diet.

3. Vitamin A deficiency must be guarded against to avoid puerperal sepsis.

4. Polyneuritis due to vitamin B deficiency is not uncommon.

Short Notes on Arthritis'

BY

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THIS disease is very common in this country but one is bound to get confused in finding out the Etiology as several types are responsible in some cases.

The following varieties are generally found—Gonorrhoeal, Gouty, Acute infective, Neuropathic, Pneumococcal, Pyaemic, Traumatic, Senilis, Sicca, Tubercular, Typhoid and Rheumatic.

In pneumonic variety upper extremities are more affected than lower—larger joints are more attacked than smaller ones.

Here I should like to deal with gonorrhoeal variety of arthritis only.

Gonorrhoeal variety of arthritis.—Articular inflammation with gonorrhoea may be due to toxæmia from the primary focus which may be again aggravated or kept up, by added infection from secondary organisms in the urethra, or from some septic focus such as the teeth, or the tonsils etc. Generally, it is due to gonococcal septicaemia as the result of metastasis carried by the blood stream to the joint.

Primary focus of infection is generally the prostate and vesiculi seminalis in the male and the cervix in the female. Time of onsets is therefore usually at the later stages of the primary infection. Involvement of joints is generally polyarticular than mono-articular.

Clinically and pathologically they are acute and subacute or chronic.

Acute type varies from flying arthralgia to synovial inflammation with scanty or abundant effusion of fluid to purulent exudate.

In subacute or chronic type there is scanty effusion and the synovial membrane, articular surfaces and periarticular tissues are all thickened with adhesion and limitation of movement.

Treatment.—Vaccine therapy is the sheet-anchor in the complications of Gonorrhoea. As a larger dose of antigen is more effective, detoxicated vaccines are now-a-days preferred which can

* A paper read at the Seventh Assam Provincial Conference of the All India Medical Licentiates' Association at Jorhat, and specially sent to the "Antiseptic" for publication.

be given even in a dose of 5000 million organisms from the start. The toxicity of endotoxins are generally so great that it is only possible to administer comparatively much smaller doses of vaccines than therapeutically effective. This is specially the case in Gono-infection. So detoxicated vaccine is preferable as it is relatively non-toxic. The antigenic properties are the same. Much larger dose causes no severe reaction. So it can be used in febrile condition—no chance of negative phage.

Vaccine therapy is greatly enhanced when combined with Protein-shock such as milk injection or T. A. B. vaccine. Arthigon, a Polyvalent vaccine with urotropine gave me excellent result in a case of Gonorrhoeal Epididymitis. When vaccine plus Protein-shock is not very effective, it may be combined with chemotherapy such as sulfarsenol or contramine. Intravenous Iodine helps better absorption and resolution.

All other treatments to improve general condition and help elimination and arrest focal infection as also local treatment of the joint should as usual be resorted to.

I have dealt with the general aspect of the disease, as far as possible, in a brief outline. Now I wish to give a short note of an obstinate or fulminating case of Gonorrhoeal Arthritis.

A cooly woman, aged 34, Telenga by caste, was admitted in this hospital on 13-7-36 with an attack of Arthritis in her left knee joint. She was a widow and multipara with three children—all healthy and alive.

Previous History.—She was a healthy woman of strong build. No complaint of any pain or Rheumatic troubles any time. No P. V. examination allowed. No history of venereal disease traced out.

Present Condition.—General health was not very much affected. She was running a temperature varying between 101° to 103° F. for the last two weeks without remission. Pulse was good and regular with moderate tension and volume. Nothing abnormal in heart or lungs.

Liver—2" below the costal arch.

Spleen—1" " " "

Genito-Urinary System.—Urine was scanty and yellowish. Occasional burning sensation during micturition.

Result of Urine Examination:—

Sp. gravity—1020.

Reaction—Highly acid;
Phosphate—Present.
Albumin—Abundant,
Bile—Present.

Microscopically no pus-cell or any other organism found.

Morbid Anatomy.—Her left knee joint was tremendously swollen. Synovial membrane, Periarticular tissues and cartilage and possibly bone were infiltrated and hypertrophied with extensive damage. It may be that the capsule and surrounding tissues were much more infiltrated and swollen—hence the enlargement of the joint. There was very little accumulation of synovial fluid as only about 2 drs. were aspirated for examination but none came out on subsequent attempts though on palpation, I thought, there was certainly sufficient effusion.

Locomotor System.—The affected joint was immobilised and all other joints were free and unaffected. It was extremely painful and tender.

The leg was kept always bent at right angles. The pain was so severe and excruciating that she fretted and fumed on the slightest movement and passed restless days and sleepless nights. She rather preferred to die than linger in this agony.

Microscopic Examination.—Blood examination showed, leucocytosis with relative increase in Polymorphs. No. M. P. found on repeated examinations. On examination of synovial fluid, Gram-negative intracellular diplococci found. No other Pyogenic organisms detected.

Diagnosis.—Osler is of opinion that in the later stage, the pus or the serum from the affected joint, is sometimes found to be sterile, the gonococci having died after causing the inflammation and that such an occurrence is always suggestive, as sterile fluid or pus is rarely found in an acute abscess due to ordinary pyococcic infection. However in my case, gonococci were found and the diagnosis was obviously gonorrhoeal arthritis.

Treatment.—Pot. Iodide and sodiisalicylas with alkaline mixtures, occasional purgatives, hexamine and acid sodium phosphate etc. were administered as required with other symptomatic treatment. Aspirin, Antipyrin, Sodium-amytal, Chloreton, Medinal, Bromide and Chloral etc. were given to relieve pain.

Milk injections with intravenous iodine were started from the very first day.

Prognosis on treatment.—Nothing ameliorated the symptoms. The pain was unbearable. He was a menace to the other patients as well—not even the doctor was spared from her shouting day and night.

Though inadvisable, she was put to morphia and atropine gr. $\frac{1}{4}$ and 1/200 respectively every night. Liq. morphine and Bromide were continued at day time. These had to be continued for about six weeks. 40 tablets of morphia and 4 oz. Liq. morphine were consumed in all.

The following injections were given:—

Milk Injection (dose 5—15 c.c.)	...	9
Iodine intravenously	...	8
A course of gono-vaccine B. I	...	6
Sulfarsenol 30 cgms. each	...	3
Sodii Iodide and Sodii Salicylas each gr. 10	...	1
Total	...	27

Local treatment.—Antiphlogistin, Ext. belladonnae and glycerinum, Scott's dressing. Liniment Iodine, Lint. Pot. Iod. C. Sapon, Saturated sol. of sodii bicarb etc. were all tried in vain.

Diet.—Plenty of fluids such as milk and barley water, albumin water, glucose D, and vegetable soup etc. were given.

General course of the disease.—Injections were begun on 14.7.36 and finished on 26.9.36. In spite of all treatment the condition was unchanged for about seven weeks. Temperature of course gradually came down to 99°—100° F. though without remission. Other joints such as shoulder joint, elbow joint, wrist joint, and knuckles all were swollen and painful one after the other. But they subsided quickly just to reappear shortly and disappear again. Another curious complaint—burning sensation on her abdomen came up suddenly and lasted for a week. Perhaps this was more severe than the joint pain. Her general condition was much run-down; she became extremely anaemic 30% Hb. She had no vitality or defensive mechanism in her system. I was patiently awaiting her fatal end.

But to my surprise and great joy, at the end of the 10th week, her temperature came down to normal—Pulse improved, pain and swelling began to subside. She was gradually coming round, she picked up remarkably well, and subsequent recovery was uneventful within a very brief period.

Ankylosis was treated by active and passive movement and massage. The patient was discharged cured on 9.11.36 when she could walk and work almost as normal, though slightly crippled.

In this connection I may be allowed to cite another case of Gonorrhoeal Epididymitis treated with vaccine and Protein-shock. A cooly boy, aged 20, was admitted in the hospital with a huge scrotal tumour—Epididymitis complicated with abundant accumulation of fluid in the sac. Urethral discharge was very scanty. It showed gram-negative intracellular diplococci on microscopic examination. The case was treated with Gono-vaccine B. I. and milk injection along with the urethral irrigation with acriflavine solution 1 in 1000. Curiously enough, the swelling was very much diminished and the fluid was being absorbed after two or three injections within a very short period. Subsequent injections completely cured him clinically. The absorption of fluid in this case, was an unexpected and wonderful phenomenon to me.

I have treated a few other cases of interstitial keratitis with this combined method of treatment—the result was exceedingly satisfactory, though of course, it requires no further comment.

Comments.—The patient could bear, possibly on account of unbearable pain, a long course of morphia without any depression or untoward feelings and any habit formation, though the patient was in a very prostrated condition.

Milk injection had no local or general reaction even in a dose of 15 c.c. This might be due to filtration of casein as suggested by my chief-doctor, W. J. Moloney, M.B. Personally I prefer fresh milk sterilized than its various market preparations which do not keep well long.

Sodii Iodide and Sodii Salicylas intravenously given to relieve pain produced severe reaction. Temperature shot up to 106.8°F. with shivering and rigor immediately after the injection, though it did not last long. I experienced no such reaction in my previous cases.

My excuse for writing out the note of this case is that the recovery of the case particularly with protein-shock and Chemotherapy combined with vaccine is perhaps unique and encouraging in a persistent case like this.

My grateful thanks to my Chief Medical Officer, Dr. W. J. Moloney, M.B., for his kind permission to write out this note and use the hospital record.

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Clark, A. J., *Applied Pharmacology*, 1933, p. 572.

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Puerperal Fever—its Causes and Treatment*

Dr. N. C. Appayya, L.R.C.P., (LOND.) M.R.C.S., (ENG.)
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AYURVEDA, which is about 3000 years old, mentions it and undoubtedly Susruta, Charak, and other great physicians of ancient India knew all about it and ordered that the parturient woman should be separated from others of the family and looked after, as if she were being treated for a major illness. In the West, there is no record of the older physicians having known anything about it until about hundred years ago. Oliver Holmes of America found in 1842, that a large number of parturient women died because of contagion and he published a paper called "The Contagiousness of Puerperal Fever." He firmly believed the infection was carried from one person to another by means of inoculations. Unfortunately, his teaching met with failure. In 1847 Semmelweis of Vienna also thought that puerperal fever was due to absorption of decomposed animal matter. He gathered so much material for proving that the cause of puerperal fever was due to absorption, into blood, of animal matter and laid down all the antiseptic principles that we now know, in conducting labour cases, in his hospitals and in private practice; but like that of Holmes, his theory was not accepted and it fell into disuse and he died of dementia. Later, when Pasteur, Lister and others came into the field and proved the germ theory, then only the works of Semmelweis and Holmes were recognised.

Ætiology.—Puerperal fever is nothing more nor less than wound infection by bacteria and cocci. If there was no wound in parturition then there would be no puerperal fever. But, unfortunately there could be no parturition without wounds of the genitalia and there could be no sterile passages through which the passengers can be born. Therefore our aim should only be to reduce the extent of wound caused by child birth and prevent, as far as possible, the bacteria and cocci present in the genitalia from coming into contact with the wound and thus getting into the circulation. We can also aim at reducing the number of bacteria

* A paper read before the Trichinopoly District Medical Conference on the 23rd December 1936, and specially sent to the "Antiseptic" for publication.

present in the genitalia and round about and prevent the organism getting into the wound from without.

Modes of transmission to the genital tract. It is well known that organisms are carried from without, in a large number of cases, by the hands and throat of the accoucheur, the clothes, the utensils, the air and in fact everything that can be found in the room. It is possible to conduct a labour case in the same way as we conduct an abdominal operation and thus reduce, to the minimum, the infection of the wound from without. In spite of all these precautions, it is still found that some women become septic. Now it is known that these septic cases are not really due to the faults of the accoucheurs, but due to the weakened condition of the mother and the absorption of organisms from the urinary and alimentary tract. This we call autogenous infection.

Among the sources of external infection the most frequent cause is the hand and the coat of the accoucheur, the midwife and of course, the barber woman. Here the causes are due to want of sterilisation of hands, the genitals and the instruments and dressings. Among the others, the air, the preparatory tub bath of the Westerner, but not the most hygienic bath of the Oriental. The husband himself may be the cause, if sexual intercourse takes place within the last three weeks of delivery and lastly the patient herself may cause her infection by carelessness of her fingers and clothes.

Exciting causes.—Pregnancy itself is an exciting cause. Next come the debilitating diseases such as tuberculosis, typhoid, etc. toxæmias of pregnancy, gonorrhœa, syphilis, prolonged and difficult labour causing interference by the barbar woman, midwife and lastly, the doctor. I believe the meddlesome midwifery of the barbar woman, midwife and the enthusiastic but ignorant doctor is the commonest cause of puerperal fever.

Types of infection.—Infections may be classified as "Sapraemia and Septicaemia." The former is chiefly caused by the retention and decomposition of the placenta, lochia, blood clot etc., while septicaemia is due to the absorption of toxic matter like B. Coli, streptococci, gonococci and typhoid bacillus.

Sapraemic cases usually get well without much trouble to the doctor or to the patient, but septicaemia gives a lot of trouble to both.

Treatment.—The treatment of puerperal fever, after it occurs, depends upon its cause. Whenever a woman who has delivered of a child gets rise of temperature and keeps up for over 24 hours after the second day of delivery, then she is said to be

suffering from puerperal fever. This fever may be due to sapraemia, and all that is necessary in such cases is to keep the woman in bed, regulate her bowels and give her a dose of quinine and Ergot mixture. Her vagina should be cleaned twice a day and her clothes, binders and napkins to be changed after a sponge bath. Usually her temperature comes down to normal after the blood clots are expelled from the uterus which enables the free flow of lochia.

In case of gonorrhœal infection the diagnosis is difficult as other organisms than gonococci may be present in the lochia. If the child's eyes are affected then it is more or less certain that the infection is gonorrhœa. In gonorrhœal infections the big joints may be affected and abscess of the cellular tissue may occur. After a course of G. Vaccine and intravenous iodine, the patient recovers but often she becomes an invalid and sterile owing to the two tubes being blocked, and the pelvic cellular tissue being affected. Tetanus, which is very rare, is often fatal when it occurs. The usual antitetanic treatment should be carried on. It is useless to give small doses of A. T. S. and when given, it must be repeated every eight hours if necessary.

You all know that the treatment of all diseases consists of (1) general (2) local (3) specific.

1. The *general* treatment of a case of puerperal fever consists in putting the patient to bed in a well ventilated room, with clean clothes and bedding, attending to her diet and keeping up her general health.

2. *Local* treatment consists of cleaning the vulva, vagina, and if thought fit, cleaning the interior of uterus in cases where there is reasonable ground to believe that bits of placenta or membranes are left behind in the uterus. I need not tell you that the intra-uterine manipulation is a very difficult one and it should not be undertaken in a light-hearted manner. Midwives must not be entrusted with this work.

3. *Specific* treatment consists in finding out the cause of fever and treating it. Where streptococci are found, 22 cc. anti-streptococci vaccine are to be injected every other day until the temperature comes down to normal. Tear of the perineum must be immediately stitched and cleaned twice a day. The wound should be kept as clean as possible and healing should be brought about with the least delay. In cases where the fever is due to haemolytic streptococcal puerperal infection, antistreptococcal vaccine has been disappointing. A large number of patients die inspite of best treatment. Of late, the obstetricians

- 3rd passage—.7 c.c. S. Broth culture of 2nd passage failed.
- Repeated —5 c.c. S. Broth culture of 2nd passage centrifuged deposit in 1 c.c. N. S. Pn. restored.
- 4th passage—.5 c.c. S. Broth culture of 3rd passage failed; not tried further.
6. Pneumo C. H. 6 (sputum). Type II.
1st passage—.1 c.c. emulsion. Pneumo restored as usual
2nd passage—not done. The strain died suddenly.
7. Pneumo CH. 4. (sputum).
1st passage—.8 c.c. emulsion. Pneumo restored.
2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
3rd passage—.5 c.c. S. Broth culture of 2nd passage failed.
Repeated —.7 c.c. S. Broth culture of 2nd passage failed —not tried further.
8. Pneumo CH. 35 (sputum)
1st passage 1 c.c. emulsion. Mouse died as usual—heart blood culture grossly contaminated. Pneumo could not be restored.
9. Pneumo CH. 36 (sputum)
1st passage—.9 c.c. emulsion. Pneumo restored as usual, not tried further.
10. Pneumo CH. 34N (sputum)
1st passage—1 c.c. sputum emulsion. Mouse died as usual—heart blood culture heavily contaminated with *B. proteus*—Pneumo lost.
11. Pneumo CH. 39 (sputum)
1st passage—1 c.c. sputum emulsion; Pneumo restored.
2nd passage—5 c.c. S. Broth culture of 1st passage centrifuged deposit. Pneumo restored.
3rd passage—1 c.c. S. Broth culture of 2nd passage. The mouse was killed by chloroform on the 2nd day of injection. Heart blood culture—no growth.
12. Pneumo Poly (from our Vaccine Lab.)
1st passage—.9 c.c. culture emulsion N. S. (from growth in solid media) injected; failed.

- Repeated —7 c.c. Pneumo emulsion from growth in solid media+.3 c.c. pyocyanous emulsion. Mouse died within 12 hours—culture showed *B. pyocyanous*—thus failed.
- Repeated —.8 c.c. Broth culture of Pneumo+.2 c.c. Pyocyanous mouse died. Pneumo restored.
- 2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
- 3rd passage—.7 c.c. S. Broth culture of 2nd passage+.1 c.c. Pyocyanous—mouse died. Heart blood cultured. Pneumo restored; not tried further.
13. Pneumo B₂. Isolated from sputum.
1st passage—.8 c.c. S. Broth culture—failed.
Repeated —5 c.c. S. Broth culture centrifuged deposit in 1 c.c. N. S. Pneumo restored from Heart blood culture.
2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
3rd passage—.5 c.c. S. Broth culture of 2nd passage. Pneumo restored.
4th passage—.2 c.c. S. Broth culture of 3rd passage failed; not tried further.
14. Pneumo Shyamlal, Isolated from sputum. (4 months old strain).
1st passage—1 c.c. S. Broth culture failed
Repeated —5 c.c. „ „ centrifuged deposit failed.
Repeated —10 c.c. S. Broth culture centrifuged deposit failed.
Repeated —15 c.c. S. Broth culture centrifuged deposit failed; mouse died—heart blood culture was heavily contaminated. Pneumo could not be restored.
15. Pneumo. 332. Isolated from sputum.
1st passage—1 c.c. Glucose Broth culture—failed.
Repeated —1 c.c. S. Broth culture+.2 c.c. Pyocyanous. Pneumo restored.
2nd passage—.7 c.c. S. Broth culture of 1st passage. Mouse did not die—it was killed and heart blood cultured; no growth.

- Repeated —5 c.c. S. Broth culture of 1st passage centrifugalised deposit : Pneumo restored.
16. Pneumo. S. 37. (Isolated from sputum).
 1st passage—1 c.c. S. Broth culture—failed.
 Repeated —.9 c.c. Broth culture+.1 c.c. Pyocyanous ; failed.
 Repeated —.1 c.c. S. Broth culture+.2 c.c. Pyocyanous. Pneumo restored
 2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture of 2nd passage failed.
 Repeated —1 c.c. S. Broth culture of 2nd passage. Pneumo restored ; not tried further.
17. Pneumo. Roy (sputum)
 1st passage—.8 c.c. sputum emulsion. Pneumo restored
 2nd passage—.9 c.c. S. Broth culture of 1st passage+.1 c.c. pyocyanous—Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture of 2nd passage. Pneumo restored.
 4th passage—.3 c.c. S. Broth culture of 3rd passage, failed. The strain died suddenly ; could not be further tried.
18. Pneumo I. I. M. R.—Type I.
 1st passage—2 c. c. S. Broth culture centrifugalised deposit in 1 c.c. N.S. Pneumo restored.
 2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture of 2nd passage failed.
 Repeated —.7 c.c. S. Broth culture of 2nd passage Pneumo restored ; not tried further.
19. Pneumo. B₄. N. M. I. (sputum)
 1st passage—1 c.c. sputum emulsion. Pneumo restored.
 2nd passage—.7 c.c. S. Broth culture of 1st passage failed.
 Repeated —1 c.c. S. Broth culture of 1st passage ; Pneumo restored.
 3rd passage—1 c.c. S. Broth culture of 2nd passage (mouse died on the 3rd day) ; Pneumo restored.

20. Pneumo B₉. N. M. I. (sputum)
 1st passage—.8 c.c. sputum emulsion. Pneumo restored.
 2nd passage—1 c.c. S. Broth culture of first passage. Pneumo restored.
 3rd passage—.8 c.c. S. Broth culture of 2nd passage. Pneumo restored.
 4th passage—.5 c.c. S. Broth culture of 3rd passage failed—not tried further.
21. Pneumo M. (Isolated from Throat swab).
 1st passage—10 c.c. S. Broth culture centrifugalised deposit in 1 c.c. N.S. Pneumo restored.
 2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture of 2nd passage failed.
 Repeated —.7 c.c. S. Broth culture of 2nd passage. Pneumo restored (Mucous growth).
 4th passage—.5 c.c. S. Broth culture of 3rd passage failed.
22. Pneumo. Bag (sputum)
 1st passage—.5 c.c. of sputum injected. Pneumo isolated pure.
 2nd passage—1. c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture of 2nd passage failed. Heart blood was cultured after killing the animal which did not die even on the 2nd day ; culture—nil.
 Repeated —.8 c.c. S. Broth culture of 2nd passage Pneumo restored.
 4th passage—.4 c.c. S. Broth culture of 3rd passage mouse died after 36 hours. Pneumo restored.
 5th passage—.1 c.c. S. Broth culture of 4th passage Pneumo restored.
 6th passage—.05 c.c. S. Broth culture of 5th passage. Pneumo restored.
 7th passage—.03 c.c. S. Broth culture of 6th passage failed.
23. Pneumo. Pyor (Isolated from Dental Swab).
 1st passage—5 c.c. Broth culture centrifugalised deposit in 1 c.c. N.S. Pneumo restored.

- 2nd passage—1 c.c. S. Broth culture of 1st passage, failed.
 Repeated —1 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture of 2nd passage. Pneumo restored.
 4th passage—.2 c.c. S. Broth culture of 3rd passage, failed.
24. Pneumo Mallick—(Isolated from Throat swab)
 1st passage—5 c.c. Broth culture centrifuged deposit in 1 c.c. N. S. Pneumo restored from heart culture of mouse which died on the 3rd day.
 2nd passage—2 c.c. S. Broth culture centrifuged deposit. Pneumo restored.
 3rd passage—.5 c.c. S. Broth culture. Pneumo restored.
25. Pneumo B. 11. N.M.I. (sputum)
 1st passage—.5 c.c. sputum emulsion. Pneumo isolated.
 2nd passage—1 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.7 c.c. S. Broth culture of 2nd passage failed—not tried further.
26. Pneumo B. 7. N.M.I. (sputum)
 1st passage—.8 c.c. sputum emulsion. Pneumo restored.
 2nd passage—5 c.c. S. Broth culture of 1st passage centrifuged deposit in 1 c.c. N. S. Pneumo restored.
 3rd passage—1 c.c. S. Broth culture failed.
 Repeated —1 c.c. S. Broth culture of 2nd passage. Pneumo restored.
 4th passage—.3 c.c. S. Broth culture of 3rd passage. Pneumo restored.
 5th passage—.1 c.c. S. Broth culture of 4th passage failed—not tried further.
27. Pneumo L. A. 43-5 (from Lister Institute)
 1st passage—.5 c.c. S. Broth culture. Pneumo restored.
 2nd passage—.1 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.05 c.c. S. Broth culture.

- 4th passage—.02 c.c. S. Broth culture of 3rd passage. Pneumo restored.
 5th passage—.005 c.c. S. Broth culture of 4th passage failed.
 Repeated —.007 c.c. S. Broth culture of 4th passage. Pneumo restored.
 6th passage—.001 c.c. S. Broth culture of 5th passage failed—not tried further.

28. Pneumo. R. (sputum)
 1st passage—.2 c.c. sputum injected. Pneumo isolated.
 2nd passage—.5 c.c. S. Broth culture of 1st passage. Pneumo restored.
 3rd passage—.1 c.c. S. Broth culture of 2nd passage. Pneumo restored.
 4th passage—.05 c.c. S. Broth culture of 3rd passage. Pneumo restored.
 5th passage—.01 c.c. S. Broth culture of 4th passage failed—not tried further.

Discussion.—It is evident from the records that with the increase in the numbers of passage, the virulence of each strain has been more or less increased though not at an uniform rate. Such deviations might be due to the following probable causes:—

(1) In these series, since each passage was delayed by a week or so, due to some unavoidable reasons, the virulence of the strain which was increased as a result of passage might have come lower due to repeated subculture.

(2) The strain might have been deteriorated in its virulence on account of the fact that as a result of metabolic product of the organisms inside the injected animal, a peroxide gas might have been generated (Topby & Wilson—Principles of Bacteriology and Immunity p. 65 and Florence Oram—Journal of Immunology Vol. xxvi, 1934) which exerted some antiseptic effect on the organisms resulting in a fall in its virulence. To counteract the effect of the gas, a very small dose of B. Pyocyanous culture (1 c. c.) along with the pneumococcus was injected. In this way it was however possible to restore the pneumococcus of low virulence insufficient to kill animal independently (No. 12, 15, 16, 17 of the series).

(3) The heart blood was always cultured after animal's death, before which the animal might have developed some anti-toxin in the presence of which the strain might have deteriorated.

in its virulence as it may happen under such circumstances that an original smooth strain may turn to a rough variant.

(4) Most of the animals died within 12 to 15 hours after injection. As it was not always possible to culture to heart blood immediately after death, the virulence of the pneumococci might have fallen due to the action of other saprophytic organisms on the dead animal. In some cases this fallacy was overcome by culturing the heart blood from just dead animal and thus virulent strains could be restored.

In conclusion, it may be said that though pneumococci get increased in their virulence with the animal passage, this phenomenon is by no means uniform and steady and that different strains though behaving differently as regards their virulence to mouse, it is apparent that this virulence cannot be enhanced indefinitely nor beyond 5 or 6 passages. (Other records are however 8 to 13 passages. Dr. Napier and Dr. Dharmendra, I. M. G., April 1935). This peculiarity may be due to various reasons having their origin either in the culture itself or to other factors such as media used, laboratory life of the culture, or the animal selected. Further work is necessary to verify the actual cause of the inability to 'passage' *Pneumococcus* beyond a limit. The number of the experiments done here are too few to assert any definite conclusion but it is hoped to publish on a future date the observations after further works.

Lastly we wish to thank Dr. A. K. Sen, Director of the Laboratories for kindly allowing us to publish these preliminary observations and for his various valuable suggestions.

Other References:—

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2. *Fleming and Pettit—Recent Advance in Serum and Vaccine Therapy.*
3. *Park and Williams—Pathogenic Micro-organisms.*

SINUS THERAPY

A recent contribution to sinus therapy is the introduction of iodine vapor into the diseased cavity through the exchange of air between the nasal chamber and the sinus, or its direct introduction by means of a cannula.
—*The Eye, Ear, Nose and Throat Monthly.*

CASES AND COMMENTS

A Case Report

BY

W. S. Mathias, Z. D. S.; Z. O. S. (Vienna).

Coonoor, The Nilgiris.

Patient's name—F. W. Barton. *Sex*—Male. *Age*—39. *Occupation*—Military Engineering Department.

Patient was advised by a dentist to see me with a view to "making good" an upper central incisor with a small mesial filling which apparently had an apical abscess, and which he was very anxious to save. His erupted teeth were good, normal in number and in perfect arch. Vitality test proved that the tooth was not dead. I advised a radiograph. Being a military man, he sought X-ray diagnosis from Government to which he was entitled. No two persons agreeing either about diagnosis or treatment, he came back to me. On radiographing him, I diagnosed a supernumerary unerupted tooth lying across the apical third of the right upper central incisor. It was explained to him that an operation would be necessary to extract it and he was advised to undergo it in order to save the central incisor. Patient, instead, got his other tooth done to "put off the evil day" and came to me 2½ months later with a small fistula above the apex of the incisor. The follicle of the unerupted tooth had become infected and secondarily also the periapical region of the central incisor which was proved dead.

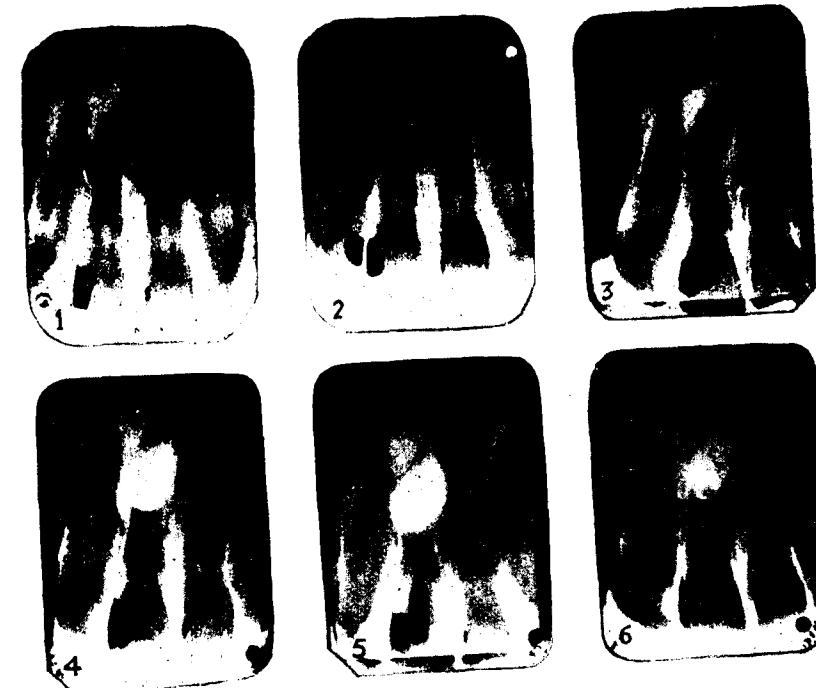
Immediate treatment and extraction with a flap operation was advised and the patient was told that it might be necessary during the course of the operation to remove also the central incisor. Unfortunately for him, two more radiographs taken from directions mesial and approximal to the incisor, to determine the lie of the tooth, proved definitely that the unerupted super-numerary tooth lay posterior to the roots of the right upper incisors. This made the case much more difficult to deal with. Operation and chiselling of bone from the palatal side of the incisor region was inadvisable due to the anterior palatine arteries, veins and nerves emerging from the anterior palatine foramina in this region.

The only solution I could think of was to do resection of a portion of the root of the central incisor with a view to getting at the tooth behind it and at the same time saving the central incisor.

The root canal of the incisor was treated and filled with cement. A look at the radiographs will show this clearly. It was impossible to resect enough root to uncover the unerupted tooth as this would have left the incisor very insecure. A little more length of root than it is usually removed for an apicectomy was resected and in addition a little bone above the unerupted tooth. The tooth was shifted partly into the cavity thus made and disengaged from the root of the central incisor. The flap of mucoperiosteum was cut into and lifted from well below the apical region. The operation was fully aseptic. Anaesthesia was done by injecting the infra-orbital foramina extraorally and the incisive foramen palatally. Healing took place by organisation of blood clot. Half the sutures were removed on the 5th day and the remainder on the 7th. Patient was given an injection of omnadine but did not need to take the analgesic powder given to him as he did not have enough pain to keep him awake at night. The central incisor was tightened up and it is quite firm now. No marks of the operation are to be seen.

The radiographs No. 1. and 2 show the tooth before operation, No. 3. shows the incisor after extirpation of the dead pulp and the first root canal treatment. No 4 was taken immediately after operation. No. 5 about three weeks later and No. 6 about 10 weeks after operation. Rapid regeneration of bone is seen and No. 7 seems to prove what many authorities hold—that cementum can be deposited on the healthy cut end of a resected root. A fresh radiograph taken a month or two hence will be sent to the journal later on.

It is gratifying to see how the practice of dental or oral surgery is advancing in South India. With the surgical background, always kept in mind, dental surgery must have a great future in this country and the case reported shows what can be achieved.—[ED. "A".]



No. 1 & 2. Tooth before operation.

No. 3. Incisor after extirpation of the dead pulp and the first root canal treatment.

No. 4. Tooth immediately after operation.

No. 5. do. 3 weeks do.

No. 6. do. 10 „ do.

A Short Note on Intra-uterine Glycerine

Medication in some Puerperal Infection Cases

BY

J. M. Datta,

A. M. O. Khobong, T. E.

TRUE puerperal septicaemia is very rare amongst the labour women of this garden. Cases of puerperal sepsis which are generally met with, are either due to sapraemia or to some local septic condition. Amongst 1095 confinements in this garden during the last six years, there were 23 cases of puerperal infection of which only one case was diagnosed as puerperal septicaemia.

Last year, I happened to get six cases of puerperal infection within a period of one month. All those cases were treated by intra-uterine injection of sterile glycerine with 5% tincture of Iodine added to it with satisfactory results. Short descriptions of individual cases are given below.

CASE No. 1.—A Ghashi woman, aged 25, 4th para. Hb. 50% delivered in the hospital on 25th July 1936; developed fever 101°F. on the 30th and then she was out of our control till 3rd August *i.e.* 10th day of delivery, when she was re-admitted with fever 101.6° F. pulse 110, respiration 22 and she looked very ill. A painful hard tumour was felt over the right side of the pelvis, uterus was subinvolved and very tender. Lochia was scanty and very offensive, vagina and cervix were inflamed and tender. Parametritis with septic endometritis was diagnosed. Hot vaginal douche twice daily, injection of iodine solution intravenously, commencing from m. xl in 10 c.c. of distilled water, increased by m. x every third day. P. P. mixture etc. did no good. Intra-uterine glycerine with 5% Tincture of Iodine twice daily was commenced from the 26th day of delivery. Fever came down to normal, tender mass disappeared and uterus completely involuted within 7 days. This case unfortunately died of mucous colitis and exhaustion on the 42nd day of delivery.

CASE No. 2.—A Munda woman, aged 20, 3rd para, a placenta praevia case, delivered on 12th August 1936 and had fever 100°F. on the 3rd day of her delivery. Lochia slightly offensive on the 5th day, fever 102.6° F. pulse 110, respiration 28, uterus involuting slowly and not tender, and slight tenderness towards the left side of

the pelvis on deep pressure. Vagina normal, sloughing ulcer on the cervix, small quantity of pus mixed with blood discharging from the external os when the anterior lip was raised by the catheter. Sloughing lacerated cervix with endocervicitis was diagnosed. Injection of puerperal antistreptococcus serum, 20 c.c. pituitrin, P. P. mixture, hot vaginal douche gave little result. Intra-uterine glycerine with Iodine twice daily was commenced on 7th day of delivery, fever came down to normal on 9th day, uterus rapidly involuted and the patient then made an uneventful recovery.

CASE No. 3.—A Munda woman, aged 40, 8th para was admitted on 23rd August 1936, 7th day after delivery, with fever 101° F. sub-involution of uterus which was very tender and offensive and lochial discharge. On P. V. examination, an extensive puerperal ulcer on the perineum, vagina normal, large quantity of pus coming out through a lacerated and sloughing cervix on passing the uterine tube; sloughing cervix with endometritis and retained lochia was diagnosed. Intra-uterine glycerine with Iodine twice daily was at once started. Fever came down to normal, uterus completely involuted within 7 days and she was discharged cured within 3 weeks.

CASE No. 4.—A Chik girl, aged 18, 2nd para, admitted on 22-8-36, 3rd day after delivery with fever 102° F. and sub-involution of the uterus. Injection of pituitrin, P. P. mixture, and hot vaginal douche were given without any result. Lochia became offensive on 5th day. Vagina and cervix were normal. Intra-uterine glycerine with Iodine twice daily was started on 10th day and a large blood clot came out on the 11th day. Another clot came out on the following day. Fever came down to normal on 20th day of delivery, uterus completely involuted and then she had an uneventful recovery.

CASE No. 5.—A Dome woman, aged 23, 3rd para, Hb 40% delivered in the hospital on 26th August 1936. Fever 101° F. developed on the 5th day, lochia became scanty and offensive and there was sub-involution of the uterus. On the 20th day of delivery a slightly tender mass was felt on the left side of the pelvis. Irregular fever continued. Any medication through vagina was refused but on the 35th day, when her condition grew very bad, she consented. On P. V. examination—a small sloughing ulcer was seen on the cervix. Sloughing cervical ulcer with parametritis was diagnosed. Temperature came down to normal on the 2nd day after the intra-uterine glycerine with Iodine twice daily, uterus completely involuted and the mass disappeared within 8 days. She then recovered without any further trouble.

CASE No. 6.—A Dome woman, aged 22, 5th para, was admitted on 6-9-'36, 10th day after delivery with fever, 103.4° F. dry and coated tongue, pulse 120, respiration 28, and looked very ill. Uterus tender and not involuted. Lochia very offensive. Chest normal, P. V. examination:—Vagina and cervix normal, much pus mixed with blood came from uterus when uterine tube was passed inside. Septic endometritis with retained lochia was diagnosed. Intra-uterine glycerine with Iodine twice daily was at once started together with injection of antistreptococcus puerperal serum 20 c.c. and other usual treatment of puerperal sepsis. Fever came down to normal within 6 days, uterus not palpable after 9 days, and she was discharged cured within 3 weeks.

Since the introduction of Remington Hobb's intra-uterine glycerine treatment, no material change has been effected so far as injecting fluid is concerned. Many still prepare sterile glycerine as originally introduced and do not like adding any antiseptic due to the fact that this will not only fail in its purpose of sterilizing the birth canal for want of sufficient concentration, to kill the infecting organisms, on account of the constant outward flow from the placental surface but may rather do harm by destroying the protective leucocytic carrier. Some workers however have used non-corrosive antiseptics such as acriflavine, mercurochrome etc. with much success. References are not available to me in support of the Iodine with glycerine method used in this series of cases, but some of my colleagues used this solution with satisfactory results. Whether these good results have been due to simple glycerine or to the addition of tincture of Iodine, I cannot say as I have not had a series of control cases. This is a matter for discussion.

Method of Administration.—One long uterine catheter specially made for this purpose, one 10 or 20 c.c. record syringe and cusco's bivalve vaginal speculum are all that are necessary for the purpose. After carefully douching the vagina with hot lysol solution (3 i to O. i. or 3 i to a pint), taking care that the douche nozzle does not touch the margins of the vulva, cusco's bivalve speculum is slowly introduced as far as it will go. Screwing it widely open, the os only will come into view with suitable adjustment. After swabbing the cervix with Tr. of Iodine, Uterine catheter fitted to syringe already filled up with the solution is slowly and carefully introduced inside the uterine cavity taking care that the uterine curve of the instrument is in the proper direction. The pelvis of the mother is then raised about 30° and 30 c.c. solution injected. She is allowed to remain in this position

for an hour after which pelvis is lowered and the head of the bed is raised to promote drainage. This is repeated twice daily. Some patients complain of much pain during opening of the speculum and on introducing the catheter. But this is transitory. In spite of tilting the pelvis, some amount of fluid comes out of the uterus when uterine catheter is removed. This cannot be avoided. The intra-uterine cannula devised by Dr. Parekh and made by Messrs. N. Powell & Co., may serve the purpose of retaining medicament inside the uterus as long as necessary though I have got no practical experience of it.

Summary.—(1) 6 cases of puerperal infection treated by intra-uterine glycerine with 5% Tincture of Iodine have been described.

(2) Whether it was the addition of Iodine that added to the efficacy of treatment has been discussed.

(3) Method of administration has been described.

Conclusion.—No originality is claimed for the treatment described in this short note. Iodine was added to glycerine with the idea of inhibiting the growth of pathogenic micro-organisms. But this is of doubtful value because of low concentration of Iodine used. It has already been said that corrosive antiseptics may do harm; but in this series of cases, no bad effects due to the addition of tincture of Iodine have been noticed.

My grateful thanks are due to Dr. W. F. Whaley, our chief medical officer, for his kind guidance in preparing this note and his permission for publishing it.

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2. Tropical Midwifery—Section: Fever during Puerperium by V. B. Green Armytage, p. 111.
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A Case of Tuberculosis of Spine

Occurring after healing of lung affection and presenting unusual difficulties regarding prognosis

BY

Rai Dr. G. C. Chatterjee Bahadur, M.B., (CAL.) F.R.I., (LOND.),
Calcutta.

VAGARIES of tuberculosis present unusual difficulties regarding prognosis in comparison with other diseases, which can only be appreciated by one who has opportunities of following the course of a large number of cases, for a pretty long period. He will then be able to appreciate the writing, on this line, of some of the observers, of which, a quotation is given (Fishberg) below:—

“There are “incipient” cases detected as early as humanly possible which have no chance, irrespective of the treatment applied, while there are many in the third stage whose chances of survival and even of efficiency are excellent”.

The patient under consideration, a lady aged 30, mother of several children, the last one, about 2 years old, born during the period under observation, was first seen by me, in her house, about the middle of August 1936, suffering from tubercular affection of the left apex, and also of dorsal region of spine where there was marked lateral and angular curvature. These have been diagnosed by x-ray by her previous advisers, who were treating her up to the time of my visit with what they thought the recognised methods of treatment, namely solganol and therapeutic compression over the left lung which had been done several times. It is necessary to state here, before I proceed further with the description of the case, to show the injudiciousness of their action, in that she was at that time practically symptomless regarding her lung affection—no fever—no cough—no physical sign and there was well balanced physiological state of her health. The thing which influenced them to do it, was the statement, made by the radiologist without any justification, of what he saw in his own two radiographs taken at an interval of 2 months, which I have since in my possession, that the disease was progressing. From early infiltration which he saw in the first negative, he made out break down and small cavity formation in the 2nd negative taken 2 months later but which does not show it. The only sensible advice which they gave was putting up Plaster of Paris

jacket over the body, in order to help in the alleviation of the spinal disease, but which was not put up for some reason or other, and also perfect rest in bed. At the time, I saw her, her only complaint was shooting pain in the lumbar region, which was so severe, as to keep her restless day and night. At that time I found there was marked lateral curvature of the lower dorsal spine, with an angular curvature also which was tender to touch. The following history was elicited—she had symptoms of apical lesion (left side) 4 years previously and the pleurisy in the right base of the right and later in the left. In October 1935 she had two or three attacks of haemoptysis. In the present year, the spinal affection took place without much fever but the lung affection which had really healed, and of which there were no symptoms, was made out by the radiologist who mistook the cycatrix to be an active disease as stated before and in which he later on saw evidence of breaking down, which made her previous medical advisers commit the mistake of putting in air into an already healed lesion. The subsequent history and course of treatment followed, is as follows. As she was apyretic, a course of tuberculin treatment was given, with rapid increase in dose, which did not produce any reaction. With this, rapid increase in weight took place. A metallic corset was put over the body, instead of Plaster of Paris jacket, to put rest to the spine. The tenderness over the spine where there was marked angular curvature gradually disappeared. She was allowed later on to sit up with the corset on, without feeling much discomfort. Later on, she was allowed to walk.

Discussion and Conclusions.—Leaving aside the bonafide mistakes made by the radiologist and of the physicians who were in-charge before, if we take the case as a whole, the strange part of the case is, why this case had run so mild a course and that in a woman who had conceived during this period. Secondly, though immunity had evidently been produced by the first attack in the lung as indicated by healing of lung lesion, yet why it did not prevent the spreading of the disease to the spine. Thirdly, rapid recovery by tuberculin in spite of lateral and angular curvature of so pronounced a character of the spine.

The next point to be considered is the mistake made by the radiologist whose reports are reproduced here. Of the two negatives which are in my possession, one is reproduced here, the other is not reproduced as it is not necessary, as there is so little of affection, not to speak of cavity, there is slight infiltration in the left apex in both the plates.

Copies of x-ray reports taken previously. First taken on 12-11-35. Screening-diaphragm—comparative restriction in the movement of the left arch of diaphragm costo phrenic angles clear, cardiac—nothing special. Posterior mediastinum clear, lungs—comparative hypo-translucency left base and left upper lobe region including apex.

No. 2973 } Film 6 ft. erect Ant. :
6 ft. erect. Ant. } (1) Infiltration left apical and sub-apical region, pleural thickening.
(2) Suspicion of very early infiltration, medial half right apex and infra-clavicular region.
(3) Thickened pleura base.

2nd x-ray examination report taken two months after. 29-1-36. Film No. 3201, chest comparative study with the previous examination of 12-11-35.

1. Shows break down in the left apical region—small cavity formation.
2. Suspicious areas in right apical and sub-apical region have not cleared up. Rather shows slight progress.

A Case of Guinea-worm mistaken for Caries of Spine

BY

Capt. B. S. Sandhu, I.M.S.

Superintendent, Central Prison, Ahmedabad.

A WELL built prisoner, aged 24 years, Hindoo, was admitted to the Presidency Jail, Calcutta, with multiple swellings all over his body especially, on the back, in the lumbar region, both the fore-arms, and on the left leg. On the day of his admission, the swellings were about the size of a walnut and were painless. This case was kept under observation for a pretty long time. He showed no signs of malaise. There was no rise in temperature and he took his meals as usual. On the twentieth day of his disease, there was fever, the temperature shooting upto 102.4° F. and with this, the swellings on the back, in the lumbar region also began to increase in size definitely. About one pound of blood-stained pus was withdrawn by trocar and cannula from the swelling in the lumbar region. It was suspected that this swelling might

have some connection with the lumbar spine and hence the case was sent to the Civil Hospital for X-Rays, so as to exclude caries of the lumbar spine. X-Rays showed no bony lesion. Though there was no fever now, his weight continued to decrease and he lost 10 pounds in a few days. After a fortnight again, about a pint of blood-stained muco-purulent discharge was withdrawn by trocar and cannula from the same swelling in the lumbar region. This did not produce any good result, and in a fortnight's time, the swelling increased and began to move down towards the buttock. Still he had no fever and did not complain of any pain. At this juncture, I thought it wise to resort to surgical interference. On incising the abscess in the lumbar region, I found calcified remains of Guinea-worm in the centre and these were broken into bits. After a few days, another abscess was incised and in this way, all the swellings were incised one after the other. He was given 1 gr. of Tartar Emetic intravenously every other day and in all 8 grs. were injected. The patient made an uneventful recovery and was discharged cured.

Discussion.—Though Guinea-worm is very common in the Deccan, N. W. Frontier, and in certain other parts of India, yet it is very rare in Bengal, and this fact led to confusion and wrong diagnosis in the beginning. I have treated about 200 cases of Guinea-worm in Waziris between October 1929 and May 1932, yet with this experience of mine, I was completely led astray, in the early part. On several occasions, I have drawn fluid from the blisters which have shown embryos under the microscope, and though the muco-purulent discharge was examined microscopically in this particular case, no embryos could be seen, probably due to mixed infection. The abscesses were mistaken to be either pyæmic or tubercular in origin especially the latter and having some connection with the lumbar spine. X-Rays did not reveal any calcareous mass in the film and only the incision made the diagnosis absolutely clear. History revealed that the patient had been a bearer to some British Officer working in Northern India, and probably he must have contracted the infection there.

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EDITORIAL

Faints and Fits

A LARGE number of clinical conditions first become manifest by a passing loss of consciousness with or without convulsions. In a very interesting article on this subject, in our distinguished contemporary, the British Medical Journal, Dr. Gilchrist has collected and tried to correlate, in a very admirable way, the results of recent investigations and advances in this field.

Just as there is no satisfactory definition of a faint, so also great difficulties beset the definition of a fit. Realising these handicaps, no attempt is made to define either the faint or the fit, recognising that the one merges at times almost insensibly into the other. This discussion is therefore limited to a consideration of the attacks characterised by sudden and transient weakness, loss of muscular control, amnesia of a varying degree, if not actual unconsciousness, with or without convulsive movements.

Recent researches have demonstrated beyond doubt that the autonomic nervous system plays an important part in the regulation of the conscious state. The recognition of cerebral ischæmia as a cause of the convulsive state led to the differentiation of attacks of unconsciousness into two broad groups—the syncopal, due to transient circulatory defects, and the epileptiform, primarily of cerebral origin. In between these two widely separated causes comes the autonomic nervous system. A convenient classification

to understand this subject would be as mentioned below according to the dominant mechanism.

1. Seizures resulting from a disturbance of the autonomic nervous system.
2. Syncope—the result of an intrinsic disturbance of the heart beat.
3. Seizures, more directly of cerebral origin, due to local structural or general metabolic defects.

A study of the common syncopal attacks is sufficient to indicate a disturbance of the usual balance between heart rate and blood pressure. Beginning with a sense of uneasiness and muscular weakness, restlessness, the symptoms gradually advance to vague abdominal discomfort, nausea, feeling of insecurity, blurring of vision, loss of power, coldness, sweating and final collapse. These are associated with a progressive fall in blood pressure, a decrease in heart rate and are called by Lewis—vaso-vagal attacks, to describe the underlying mechanism, cardiac inhibition, sudden hypotension, and diminished cerebral blood flow. Recovery is gradual, slower than onset and in the succeeding hours leaves the victim listless and unsettled. Simple syncope occurs under a variety of circumstances. The erect posture, long hours without food, hot stuffy rooms, or exposure to cold and damp, all play a part; but there is evidence to suggest that the emotional tone of the victim at the moment plays a very important part in the precipitation of the attack. Received by one or other of the special senses the stimulus itself does not cause fainting, but the emotional idea—often disgust or loathing—dominant in the mind of the individual at the moment, may be the main determining factor in the initiation of these circulatory reflexes, which produces a sudden change in the mechanics of cerebral blood flow. The slow pulses, abrupt loss of tone, are in effect a transient disturbance of the reflex control of the circulation exerted by the autonomic nervous system.

More conclusive evidence of the influence of the autonomic nervous system is afforded by recent studies of Weiss and Ferris, who have revealed in a group of people liable to fainting attacks, a peculiar sensitivity of the carotid sinus to mechanical pressure in the neck. Thus it was possible, by pressing on one or other sinus with the fingers, to reproduce an exactly similar train of symptoms as those for which the patient sought advice, showing clearly a hypersensitive state of the sinus mechanism.

There is another variety of fainting which occurs rarely but has certain characteristics which clearly indicate a disorder of the

autonomic system. It is uninfluenced by carotid sinus stimulation. Its leading symptom is readily induced fainting on assuming the upright position. There is a marked drop in the systolic and diastolic pressures when the patient stands erect and this is accompanied by actual syncope or marked weakness and giddiness. This postural hypotension is accentuated in hot weather. Localised or generalised deficiency of sweating and the failure of the heart to increase markedly in rate when patient stands erect, gives a clue to the nature of the disorder. Adrenaline while it augments the heart rate, does not restore peripheral vasomotor control. The heart is uninfluenced by atropine. This condition is probably due to a decreased activity of the autonomic nervous system amounting to a virtual paralysis of the peripheral vasomotor constrictor fibres.

In this brief review, it has been shown that identical symptoms can be produced in a variety of ways. In each and all, the activity of the autonomic system is disturbed in varying degree.

We shall next consider syncope of cardiac origin. In this can be grouped those cases in which the major defect lies in a sudden change in the dynamics of cerebral blood flow consequent upon an intrinsic disorder of the heart beat. The outstanding example is the Stokes—Adams seizure associated with certain forms of heart block. Attacks may vary in severity, from the mildest, associated with only a transient giddiness to the most critical, in which, during a long period of asystole lasting three or four minutes, muscular twitchings with cessation of respiration and urinary incontinence may occur. Though the definition and the origin of the name is not clear it is best to confine the term Stokes—Adams seizure to those forms of syncope associated with a defect, transient or permanent, in the conducting mechanism of the heart beat.

There is again a group of cases in which fainting attacks are due to a transient paralysis of ventricular activity. These people do not suffer from a persistent chronic block, but only from time to time does the bundle of His cease to function. In this a heart that beats in normal rhythm with no impairment of auriculo-ventricular conduction, suddenly and without any warning shows ventricular asystole accompanied by a faint of corresponding severity. This event is followed by independent ventricular beating for a few minutes and then almost as suddenly normal rhythm is resumed.

The diagnosis is not difficult if the absent pulse is noted

during the attack or the steady slow rate for a short time thereafter, but the presence of the normal rhythm in the intervals between seizures makes it somewhat difficult to diagnose.

A diminished cerebral blood flow may result from an excessive ventricular rate when the volume output per beat is greatly diminished. In these cases mild attacks occur and are usually preceded by violent and rapid palpitation. Fainting from this cause is more common in the later age groups.

There are a few other cardiac causes for faints. After coronary thrombosis the acute fall in blood pressure may sometimes be responsible, or the damage done to the heart muscle may give rise to a ventricular tachycardia with epileptiform seizures at the time of development of the abnormal rhythm. Syncope occasionally accompanies angina of effort. Valvular lesions rarely are associated with syncope. Of these, however, aortic stenosis is more commonly associated with syncope than is regurgitation.

Syncope in the mind of the layman is closely associated with cardiac disease. Fortunately in young people organic vascular damage is easily excluded. It is, however, justifiable to assert that fainting attacks in middle aged or elderly people are mostly due to cardiac disease.

We shall next consider seizures of cerebral origin. The features of the major epileptic attack are very well known. Petit Mal and hysterical fits are often confused with faints but the diagnosis is not difficult. Convulsions are seen in such a variety of conditions that a common denominator applicable to all has not yet been found. Recent work supports the view that the fit is accompanied by an altered conduction of nerve impulse and increased irritability of the nerve cells, alkalosis and waterlogging being factors of importance in the initiation of the unknown mechanism of the fit. Oxygen lack also has its advocates. Available evidence indicates that cerebral anæmia by itself is not the cause of the epilepsy. Vaso-motor changes have been found in eclampsia, lead-poisoning and glomerulo-nephritis but the cause of these vaso-motor changes remains a mystery.

Reference must however be made at this stage to the peculiar attacks known as "Narcolepsy", and "Pyknolepsy". In the former sudden short attacks of sleep occur unexpectedly. Pyknolepsy is confined to children. It consists of attacks, unassociated with mental deterioration and often indistinguishable from Petit Mal except perhaps in their frequency and resistance to all known forms of treatment. Fortunately these fits end spontaneously by the time puberty is reached.

There is a syndrome originally described by Gowers and named vaso-vagal attack. This in view of the discussions in the previous portion of this article may be termed vaso-medullary seizure. Its relationship to epilepsy is obscure and perhaps no more than fortuitous. Arising unexpectedly but often after a period of mental strain or anxiety, the attack is sudden and lasts for ten minutes or half an hour. The most striking, urgent and dreaded of the symptoms is a sense of impending death. A sense of utter powerlessness, weakness, praecordial discomfort, dyspnoea, choking feeling, pallor, cold perspiration and an appearance of grave illness, are common. The pulse is slow and of poor volume. The attack is not a hysterical phenomenon though it is often labelled, "nerves" or "heart" attack. These attacks though alarming are never fatal, and, whatever the precise mechanism, yield to sedative drugs and assurances regarding the future.

Only a few aspects of a very interesting clinical problem are touched in this article. The readers would do well to peruse the original in which the statements are illustrated with case notes.

GLEANINGS FROM MEDICAL PRESS

Medicine and Therapeutics

A New Medicament for the Treatment of Gonorrhoea.—

The new medicament is named Argycuprol and contains 10 per cent. silver and 0.1 per cent. copper in the form of nucleinates. The silver is alleged to kill the gonococci, while the copper causes the disappearance of the non-specific flora, at the same time producing a mild astringent effect. The Argycuprol is obtainable in yellow glass ampoules, each end of which can be opened by the patient with the aid of a file. The content is emptied into a small dish and the required amount drawn into a syringe. Spots of fluid on the clothing are easily removed with soap and water.

The box containing the preparation must be kept closed and the ampoules protected from the light.

The addition of equal parts of distilled water was subsequently tried and this weaker solution was found to be efficacious in killing the gonococci.

The syringe must be able to hold at least 10 cm. At first it is only half filled, urine is voided and then the content of the syringe under pressure is slowly injected into the anterior urethra, the

meatus is kept closed for $1\frac{1}{2}$ minutes by the finger and then the solution is allowed to flow out.

Soon the solution can be retained 5-6 minutes. The urethral discharge diminishes quickly, and smarting, burning, etc., soon cease after the first injections.

The absence of pain during the injections is further claimed for this method.

The patients are at the same time treated by the well-known general measures.—*Friedlander. Wien med. Woch., 1936, No. 50 p. 1394.—Medical World.*

A contribution to the Treatment of Hyperthyroidism with Dijodtyrosin.—The thyroid gland produces two hormones: thyroxin, which contains 4 iodine atoms to a molecule and, according to Kendal and Simonsen, represents only 14 per cent. of the total iodine content of the thyroid gland though in an active form, and the antagonistic diiodtyrosin which contains 2 iodine atoms and represents the chief quantity of the inactive reserve iodine of the thyroid gland and, according to Abelin, robs thyroxin of its toxicity, lowers the fundamental metabolism, promotes the formation of glycogen in the liver, etc. This is not a simple iodine effect but that of the intact molecule.

A man of 26, with a retrosternal struma, circumference of neck 46 cm., respiratory troubles, increase of the basal metabolism by 27.4 per cent. was given 1 tablet of 0.1 gm. diiodtyrosin Roche daily (small doses are the most effectual!) for 40 days. After this treatment he felt well, was capable of work, his weight remained constant, circumference of the neck 41 cm. The treatment was continued for another three weeks with 1 tablet only, three times weekly; circumference of neck 40 cm., the struma soft, the sternum free. After giving 0.1 gm., daily for a few weeks longer the thyroid gland was normal; he felt well and had recovered his full capacity for work.—*A. Mettler. Ther. d. Gegenw., No. 10, 1936.—Medical World.*

Modern Methods of Preventing Measles.—J. S. Baird and L. W. Pumphrey, Pennsylvania M. J. 39: 765 (July) 1936.

In sixty-six children (average age, 4 years) the prevention of measles with placental extract was attempted. In 58 per cent measles was successfully prevented. In the remaining 42 per cent, generally speaking, the attack was modified—with an average incubation period of eighteen days. The striking feature

of the study, however, appears to be in the 100 per cent. prevention attained in fourteen children with whole (unfractionated) placental extract. Obviously, fractionation is desirable if it is possible, in the interest of refinement and concentration of the material, but in the group treated with the several different protein fractions only 46 per cent. were fully protected. This appears to agree with McKhann's observations that the fractional preparations, *i.e.*, those containing only euglobulins or pseudo-globulins, do not have the same measles antibody potency as the whole extract. It is hoped that a method will be speedily found by which the antibody potency of the whole placental bulk will be made available. It is this material which, as shown, gave perfect prevention.—*A. J. D. C.*

The Treatment of Pericarditis.—Acute pericarditis demand rest in bed, cool compresses or an ice-bag on the region of the heart. Careful bloodletting or the local application of leeches relieves the circulation. Acute pain is satisfactorily combated by applying cupping glasses on the skin in the region of the heart repeatedly for a few minutes. In cases of pericarditis of rheumatic origin, large salicyl doses should be given as early as possible; if this does not agree with the patient, large doses of pyramidon, possibly injections of calcium in addition. It is important that the patient should have an antiphlogistic diet, *i.e.*, containing large quantities of vegetables and poor in salt with restricted amounts of fluid. A careful watch should be kept on the heart's action in order, if necessary, to step in with caffeine, cardiazol and similar analeptics which should always be kept handy. Digitalis is ineffectual, even contra-indicated, where an effusion exists. If, in spite of all these measures, the alarming symptoms of influx stasis, especially of stagnation in the hepatic and cervical veins supervene, relieving the heart by puncturing the pericardial cavity is always indicated; this alleviates the condition and may often save the patient's life. The point of puncture should be chosen at the level of the protrusion of the pericardial fluid which can be demonstrated by screen examination, *i.e.*, usually at the 4th or 5th intercostal space on the left side, at a distance of three to four fingers' breadths from the sternum. In exceptional cases, puncture from behind comes into consideration. To avoid injuring the heart, an exploratory puncture with a thin needle should be made previously. In some circumstances the quantity of exudate withdrawn may amount to some hundreds of cubic centimetres. It is, however, quite unnecessary to insist on complete evacuation

of the pericardial sac. Even the withdrawal of a few c.c. often removes the symptoms which threaten life and starts the process of absorption of the exudate. Where the absorption is delayed, it should be promoted by diuretic, euphyllin and a dehydrating diet. Short wave diathermy may also be tried, though it is not yet possible to give a definite verdict on its effect in pericarditis.—*H. Deicher and H. Tiedke, Fortschr. d. Ther., No. 16, 1936.—Medical World.*

"Salyrgan" in Heart Failure.—In the treatment of heart failure with oedema "Salyrgan" is a valuable addition to our armamentarium. It is usually considered to be inadvisable to use it if the renal function is unsatisfactory, if the urine contains blood, or if there is any evidence of colitis. I have occasionally used it cautiously even in the presence of one or more of these contraindications when other methods have failed to relieve the oedema, and have so far not had cause to regret this. It is usually more efficacious if given in combination with ammonium chloride, but since this usually causes a good deal of gastric discomfort, "Salyrgan" alone may be tried first and is frequently effective. Even after all visible oedema has disappeared there may still be a moderate amount of latent oedema in the liver, lungs and alimentary canal, and unless this be removed, the full benefit of the drug will not be obtained. Increase of weight gives warning of return of oedema before there is any external evidence of it, and is an indication for further injections of "Salyrgan."

In patients with cardiac asthma or nocturnal dyspnoea, in whom there may be little or no visible oedema, but some hepatic and pulmonary congestion, marked symptomatic benefit is sometimes obtained from two or three injections of "Salyrgan."—*Hurley, Med. Journ. of Australia, Dec. 12, 1936, p. 808.—Medical World.*

Clauden for Therapeutic Obliteration.—Clauden is an extract of pulmonary tissue and is used for local haemostasis.

Clauden is warmly recommended for the obliteration of hydroceles. The simple method can be carried out by any practitioner. After disinfection and the application of a novocaine wheal the fluid is evacuated by puncture and 4-5 c.c. clauden injected if the hydrocele is small, anything up to 10 c.c. into a large one. In about a day's time redness and swelling of the corresponding half of the scrotum develop; it hardly causes any pain and does not prevent the patient following his occupation;

it disappears by the end of a week. If the desired result does not occur, the injection may be repeated in three to four weeks. Out of 28 cases thus treated, one to two injections were sufficient in two, in five cases three were needed, one required four injections. All the 23 patients, whom it was possible to examine after about a year, were free from troubles and relapse.

Ganea treated traumatic torsions, dropsy of joints, haemarthrosis with clauden also. If, in cases of injury to the knee, the skiagram shows no lesions of bones, the exudate should be evacuated, the needle introduced for that purpose left in place and 10 c.c. clauden injected through it. The point of puncture is covered with a simple sterile dressing fixed with strapping. This treatment should be applied as soon as possible after the accident.—*Hachmeister, Munchener Med. Wschr. No. 36, 1936; Ganea, Med. Welt., No. 34, 1936.—Medical World.*

Treatment of Haematemesis and Melæna with Unrestricted Diet.—Meulengracht five years ago decided to change the customary treatment of bleeding gastric ulcers from a greatly restricted to a practically unrestricted diet. The reasons were that patients died of exhaustion after hæmorrhages in spite or perhaps as the result of scrupulous dieting, that occasionally patients with protracted hæmorrhage stopped bleeding when they were given food, and that ambulatory patients with severe melæna frequently recovered without changing their diet. Moreover, it seemed to him inadvisable to let a patient starve at a time when, as the result of the shock of hæmorrhage, the intake of fluids, salts, vitamins and sufficient calories seemed especially necessary. The diet recommended is a so-called full puree diet. The patients are given five meals a day and are permitted to eat as much as they want. Foods are selected that will not cause irritation and are readily digestible. The noon meal includes a variety of dishes; namely soups, meats, vegetables and fruits. The other meals consist of bread and butter, gruels, milk and so on. Milk is also given between meals. The daily number of calories averages about 2,300. Three times daily the patients are given a teaspoonful of a mixture of 15 gm. of sodium bicarbonate, 15 gm. of magnesium subcarbonate and 2 gm. of extract of hyoscyamus; a tablet of iron lactate is also given three times each day. With this method of treatment the author had only three fatalities in 273 cases of massive hæmorrhages from gastric ulcers or erosions. In comparison with other statistics, which report a mortality of from 7 to 10 per cent., the mortality rate of his material is

unusually low. If with this method of treatment the mortality of bleeding gastric ulcers can be reduced to about 1 per cent, a surgical intervention will hardly be necessary during the hæmorrhagic stage. An operation for the gastric ulcer may become necessary at a later date.—*E. Meulengracht, Wien. klin. Woch., Dec. 4, 1936, p. 1481.—Medical World.*

A Study of Hepatic Oedema.—Though much work has been done on oedema in other parts of the body, oedema of the liver has had little attention. Keschner and Klemperer have now studied its incidence in 505 unselected autopsies and in a further selected series. The condition was diagnosed histologically when the capillary walls could be seen to be separated from the liver cells, and when the pericapillary spaces were distended by a granular (protein) material which could be stained. Of the 505 unselected cases 79 showed these changes. "Back pressure," attributed to cardiac failure, accounted for nearly half of these. Of all the cases of cardiac failure examined 39 per cent. showed hepatic oedema, yet of 9 cases of lipoid nephrosis with generalised oedema not one showed it. There were 60 deaths in uræmia and they have the following interesting results: of 24 cases under the heading of "malignant nephrosclerosis" no less than 18 had hepatic oedema, while of 36 cases of uræmia due to causes other than this, such as pyelonephritis and polycystic kidneys, only 14 showed the change. Hepatic oedema was commoner in influenza than in any other infectious disease, and it was noted in 5 out of 11 cases of diabetic coma. It was also common in association with exophthalmic goitre. Keschner and Klemperer refer to the type of hepatic oedema "that follows back pressure as 'mechanical oedema' and to the other variety, in which there can be no factor of back pressure as 'primary oedema,' and they suggest that it is due to increased permeability of the capillary wall. They claim that the demonstration of primary hepatic oedema is at present the only morphological evidence of increased capillary permeability.—*Lancet.*

Surgery

A New Treatment of Male Impotence (Functional) by Lumbar Sympathectomy.—The rami-communicantes of the fourth and fifth lumbar are divided on the two sides after laparotomy.

The operation has been done five times, with remarkable success, by Professor Voldani. Erection is augmented even in men who have not succeeded in accomplishing the sexual act for several years.

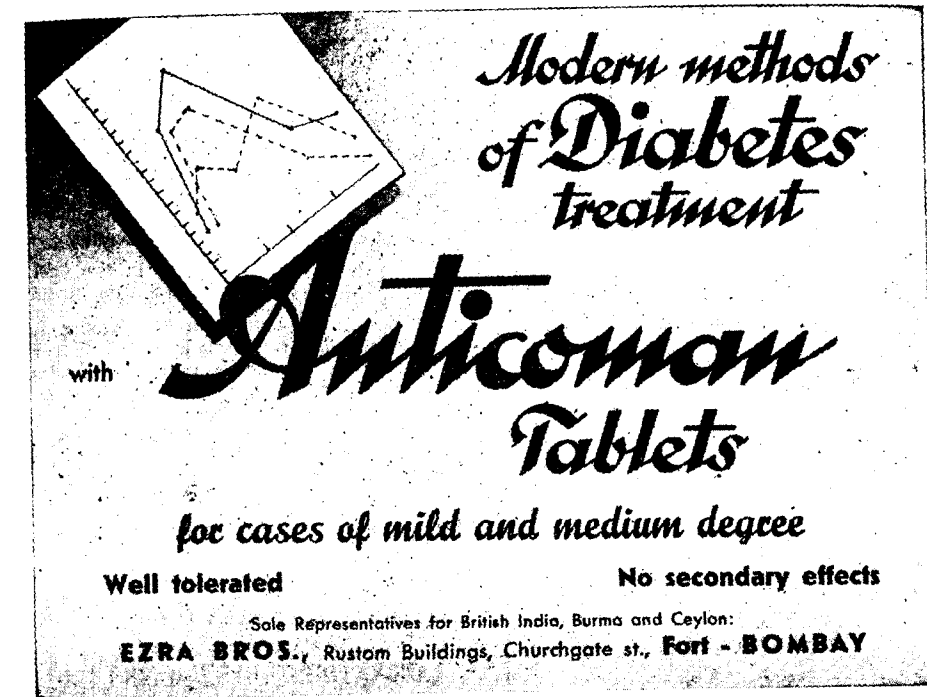
These sympathetic fibres are believed to act by inhibiting erection and one explains feeble erection in association with ejaculatio præcox by a morbid excitation of the lumbar ortho-sympathetic or of the cerebral centres.—*Nicola Pende, La Presse Médicale, No. 8, Jan. 27, 1937, p. 147.—Medical World.*

Prognosis of Gastric and Duodenal Perforation.—E. Schneider (Dtsch. med. Wschr., November 6, 1936, p. 1825) finds that, as far as his own experience is concerned at the University Hospital in Freiburg-i-Br., the frequency of 'acute perforations of gastric and duodenal ulcers has diminished during the past three years. In the course of the past six years he has had to deal with fifty-five ulcer perforations, only five of which were in women. This comparative liability of males to ulcer perforations seems to be ubiquitous. Professor Schneider has classified his fifty-five cases in four groups, according to the length of the interval between perforation and operation. There was only one death among the twenty-five patients operated on within six hours of perforation. There were four deaths among the fourteen patients operated on between seven and twelve hours after perforation. When this interval was between thirteen and twenty-four hours four of the twelve patients in this third group died. All the four cases operated on more than twenty-four hours after perforation ended fatally. The patient's life would therefore seem to hinge on the early advent of a doctor and his prompt recognition of the needs of the case. With the patient's fate determined by the earliness of a correct diagnosis the simple clinical examination is of far greater importance than any highly technical test which, if it implies delay, is rather a danger and a hindrance than the reverse. With regard to the character of the operative treatment the author is in favour of appropriate treatment for the peritonitis and the closing of the ulcer with sutures. This is undoubtedly the simplest, quickest, and most conservative procedure; and only when this suturing has been completed and stenosis of the pylorus is found to exist should a posterior retrocolic gastro-enterostomy be performed.—*B. M. J.*

Obstetrics and Gynaecology

Induction of Labour.—K. Ehrhardt and E. Henss (*Med. Klinik*, December 11, 1936, p. 1700) summarize their experience with castor-oil-quinine-hypophysin treatment, which was successful in 80 per cent. of properly selected cases. The treatment was carried out according to the following plan: at 7 a.m. two tablespoonfuls of castor oil; at 8 a.m. 4 grains of quinine; the same dose of quinine repeated again at 9 a.m. and 10 a.m.; at 11 a.m. an intramuscular injection of 0.2 to 0.3 c.cm. of hypophysin; the same dose of hypophysin repeated again at 12 noon, at 1 p.m., and 2 p.m. Multiparae respond to the treatment better than primiparae. In successful cases the duration of labour was shortened in primiparae. The artificial induction of labour was particularly successful in cases where delivery has been overdue one to two weeks. The proportion of forceps cases was not higher than in deliveries at term. The proportion of uterine atony was 2.2 per cent. This percentage is approximately the same as in spontaneous deliveries. The castor oil-quinine-hypophysin treatment may facilitate the expulsion of a dead foetus or a hydatid mole.—*Lancet*.

Treatment of myasthenia gravis.—Hyland of Toronto describes the effect of several well accredited methods of treatment upon six patients suffering from severe myasthenic weakness. He found that neither physostigmine (eserine) salicylate by mouth nor Antuitrin by intramuscular injection had any apparent influence. Irradiation of the thymous gland with X-ray was followed, in some cases only, by a remission lasting 4—12 months. A combination of glycine (5—15 grammes) by mouth with ephedrine (0.25 g. three to five times daily) by subcutaneous injection brought more lasting improvement, but its action varied much from patient to patient, and in the same patient on different occasions. In four cases dramatic relief of weakness followed the use of Prostigmin, but this was transient and a caveat is entered against repeated administration of this drug on the grounds that it may be followed by alarming deterioration. This is perhaps to exaggerate the dangers; and the statement that prostigmin is "impracticable for continued use" cannot be accepted since there are already several cases on record in which quite large doses of the drug have been given daily for over a year without ill effect. Although, as Hyland says, "the increased usage of the muscles permitted during the temporary remission may prove actually harmful if repeated too often," no harm is likely to be done if the patients are warned against over-exercise of their newly returned strength.—*Lancet*.



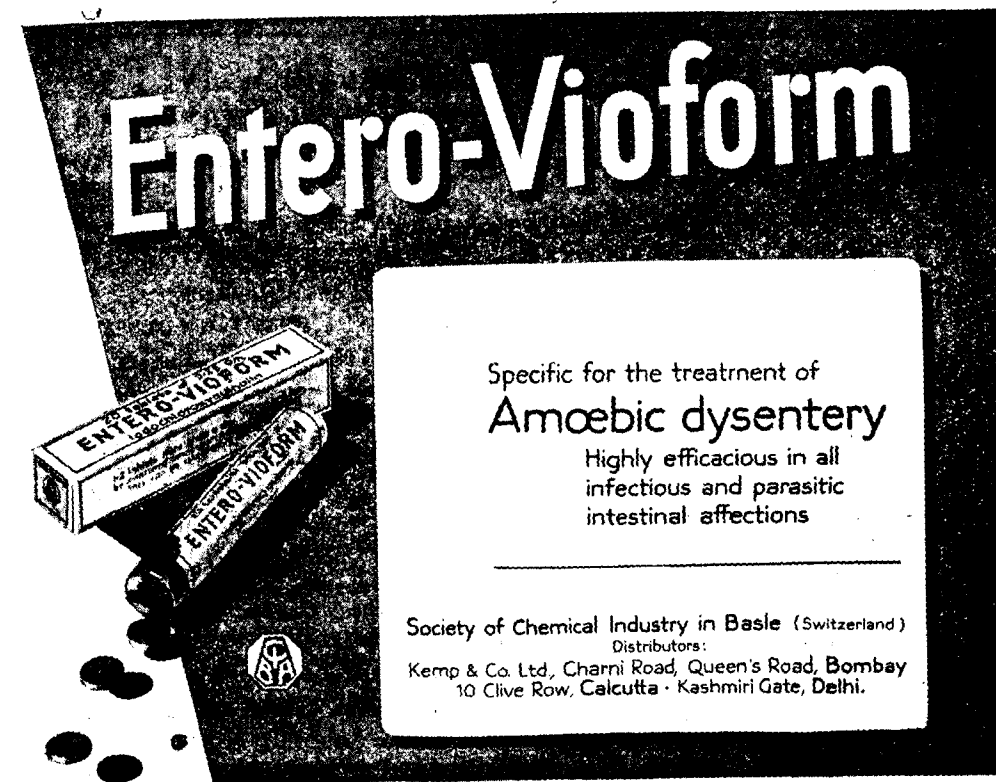
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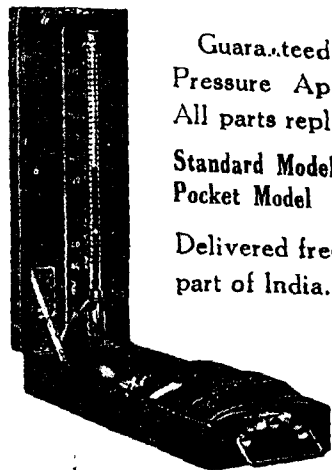
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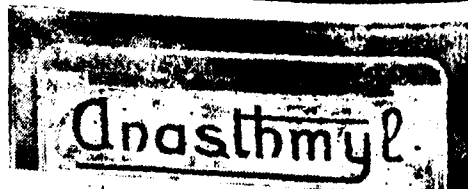
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PROCEEDINGS OF CONFERENCES, SOCIETIES, ETC.

The Kistna District Rural Medical Practitioners' Association.

An ordinary meeting of the above Association was held at Masulipatam in the D. M. O's office premises on Sunday, the 24th January, '37 at 4.30 P.M. when Lt. Col. K. V. Ramana Rau, I.M.S., District Medical Officer, presided. Mr. N. Rama Rau, L.M.P., Secretary, after reading the minutes of the previous meeting which were duly adopted, put forth the following resolutions which were unanimously passed by the members:

- I. This Association requests the Government to continue the usual subsidies for the Rural Medical Practitioners when they are sent for Refresher courses.
- II. This Association thankfully receives the proposal of Dr. V. Rama Kamath of Madras in suggesting Dr. Vittal Pai of Pallavaram to succeed late Dr. S. Rama Subbu to officiate as Honorary Secretary of the Madras Provincial Rural Medical Practitioners' Association and requests them both to arrange to call for a provincial meeting as early as possible to formally elect Dr. Pai as Secretary.

Then there was a discussion between the Members and the President about the disabilities of Rural Practitioners and the meeting came to a close by 5 P.M. after which the Kistna District Medical Association had their proceedings in the same place.

The Medical Practitioners' Association, Nuzvid.

A monthly meeting of the above Association was held at Agiripalli on Sunday the 31st January '37 at 3.30 P.M. with Dr. S. Hanumantha Rau in the chair. Mr. A. Venkateswara Rau, the local Rural Practitioner was at home to the members. The meeting commenced with prayer after which, minutes of the previous meeting were read by the Secretary Mr. N. Rama Rau which were duly adopted. The members had a long discussion over several clinical cases of professional interest of which some were the following:—

1. Mr. A. Venkateswara Rau read his case notes upon a

case of a female suffering from chronic suppurative Bronchitis and also another case which he thought to be one of chronic Appendicitis. On discussion, the latter case was decided to be one of faecal concretions simulating Appendicitis.

2. Mr. N. Rama Rau read case notes upon 5 cases of Tuberculosis of which three were primarily intestinal in origin and later developed lung infection, one was multiple cold abscesses and the other purely Tuberculosis pulmonum. In all these cases, though the first three were well advanced and of grave prognosis, and later on proved fatal, he administered unckaloubo extract which he claims to have relieved the Toxæmic symptoms and all the patients felt a sensation of well-being without much suffering. He further said that these few cases were no conclusive proof of the efficacy of the drug, but yet it is worth a trial in cases of Tubercle.

3. Mr. S. Hanumantha Rau narrated the history of a case of T. P. that started with fever typical of Malaria (no blood examination was done) and later on developed cough and lung affection.

The meeting came to a close by 6.45 P.M.

Bombay Medical Council.

The following extracts from a summary of the proceeding of the meeting of the Bombay Medical Council held on 1st February 1937 are published for information:—

The Council proceeded to consider the request made by the Representatives of Newspapers and certain Visitors at the meetings of the Council for the supply to them of the papers connected with the Agenda and resolved that it is not practicable to furnish copies of such papers to the Press or the Visitors.

The Council proceeded to consider further the application of Mr. G. S. Kasyapi, L.M. & S., for the restoration of his name to the Bombay Medical Register and resolved that sufficient time had not yet elapsed before his request could be entertained.

The Council proceeded to consider the application of Mr. Shripad Narhar Kulkarni, M.B., B.S., for the restoration of his name to the Bombay Medical Register and resolved that his name be restored to the Register.

The Council proceeded to consider further the draft revised Code of Medical Ethics and resolved (i) that it is not desirable to issue any binding rules or to insist on a rigid Code which shall have the force of law but that full freedom should be retained to treat any case on its merits and (ii) that further elaboration of the ethical principles which should guide members of the profession is not necessary.

The Council proceeded to consider further the reference from the Medical Council of India regarding the conditions of medical practice in the French Establishments in India affecting Indian Nationals possessing medical degrees included in the First Schedule to the Indian Medical Council Act, 1933, and resolved to endorse and agree with the resolution passed by the Madras Medical Council at their meeting held on 27th October 1936, viz., that "This (the Madras Medical) Council resolves to request the Medical Council of India immediately to recommend to the Government of India to correspond with the French Government in order that registered medical practitioners in the Provincial Registers in this country may be allowed to practise in French India, and in case the French Government refuses to comply with this suggestion, that the Government of India may be requested by the Medical Council of India to enact suitable legislation to prevent practitioners from French India from practising in British India unless they are in the Provincial Registers"

The Council proceeded to consider the reference from the Medical Council of India regarding the conditions of medical practice in Portuguese India affecting Indian Nationals possessing medical degrees included in the first Schedule to the Indian Medical Council Act, 1933, and resolved (i) that no further action in the matter appears called for, as the reciprocity at present existing between the Government of Bombay and the Government of Portuguese India, has been continued, but (ii) that the medical Council of India should be informed that the Bombay Medical Council would urge that the Government of Portuguese India be requested to allow all medical practitioners registered under the Bombay Medical Act, VI of 1912, to practise in Portuguese India.

The Council proceeded to consider the request of the Dean, the Nair Hospital Dental College, Bombay, for the Council's support in bringing a Dentists' Registration Act on the Statute Book and resolved that the Council would be prepared to assist in the preparation of a Dentists' Registration Act for Bombay, if a

demand for such an Act is made by an organised Dental Profession.

The Council proceeded to ballot for the election of an Executive Committee for the year and, in accordance with the result of the ballot, declared the following six members, as duly elected :—

Dr. D. A. D'Monte.
Dr. Rajabally V. Patel.
Sir Mangaldas V. Mehta.
Lieut. Colonel W. C. Spackman, I.M.S.
Khan Bahadur Sir Nasarvanji Choksy.
Lieut. Colonel Sir Jamshedji N. Duggan.

The Council proceeded to consider certain points raised by the Registrar for orders in connection with the next General Elections of Members of the Council, due to be held during the year, and resolved that :—

- (1) the existing practice of treating the Medical Register as the Electoral Roll under clause (1) of Rule 12 of the Rules of the Council, be continued ;
- (2) all practitioners registered up to the 10th August 1937 should be included in the Electoral Roll ;
- (3) 12th August 1937 should be fixed as the date up to which nomination papers should be received in the Council's Office ;
- (4) 19th August 1937 should be fixed as the date for the issue of voting papers ;
- (5) 10th September, 1937 should be fixed as the latest date for the return of the voting papers and the counting of votes ; and
- (6) that the various instructions required to be published should be published in the *Bombay Government Gazette*, the *Times of India*, the *Bombay Samachar*, the *Daily Gazette*, Karachi, the *Kesari*, Poona, and the *Sind Official Gazette* and through all Medical Associations and Societies on the list of the Office of the Council.

The Council proceeded to consider a motion given notice of and made by Mr. R. A. Amesur and seconded by Dr. P. T. Patel and supported by Mr. M. N. Talati, proposing the amendment of sections 2, 5 and 19 of the Bombay Medical Act, VI of 1912, and the motion was lost, a majority of the members voting against it.

The Council proceeded to consider a motion given notice of by Mr. M. N. Talati, regarding the grant of fees and travelling allowance to the members for attending the meetings of the Council and the Committee, but the motion fell through for want of a seconder.

The present meeting being the last on which Major-General Bradfield had to preside in view of his impending departure to take up the appointment of the Director General, Indian Medical Service, Sir Temulji Nariman then moved :—

That the Council place on record their appreciation of the cordial way in which General Bradfield had always conducted the meetings of the Council and the great help he had rendered in the disposal of the business which came before them".

The motion was seconded by Sir Nasarvanji Choksy and on being put to the vote, was carried unanimously.

After General Bradfield had made a suitable reply and thanked the members, the meeting terminated.

The Tinnevely District Medical Association, Palamcottah,

Affiliated to the Indian Medical Association.

The premises of Dr. G. N. Kantayya, B.A., M.B., B.S., (Madras), of Palamcottah, was the scene of a distinguished Medical gathering on the occasion of the opening of the Rev. Kantayya's Radiological Institute, on Monday the 15th of March 1937 at 4.30 P.M.

Sixty-two members of the Association, including four women doctors, gathered from all parts of the District. Seven other medical practitioners who are not members of the Association, were also present besides a few non-medical visitors.

Dr. G. N. Kantayya, welcomed the guests and requested Lieut. Col. T. S. Shastri, I.M.S., District Medical Officer, Tinnevely District, and President of the Association, to declare it open. Lieut. Col. Shastri, I.M.S., in declaring the Institute open said :—

"This enterprise of one of our colleagues is praiseworthy and worthy of active encouragement. It serves as a challenge to the Government Headquarters Hospital, which is just across the road, the foundation-stone of the Edward VII Memorial X-Ray Block having been laid in it by His Excellency the Governor of Madras, in November last, and

no further progress having been made with its construction. We hope good work will be turned out of it and that all of us will help its progress.

The apparatus which is a fairly high powered one, with special arrangement for Fluoroscopy was supplied and erected by Messrs. International General Electric Company, Ltd., Bangalore.

Monthly Meeting

Dr. G. N. Kantayya, entertained the guests with tea and refreshments. After sweet music, the apparatus was demonstrated to the Medical Public, after which the usual monthly meeting of the Association was held under the Presidentship of of Lieut. Col. T. S. Shastri, I.M.S.

The Secretary Dr. K. Rama Ayyar, M.B., B.S., (Andhra), read out the minutes of the monthly meeting of the Association held at Nanguneri, on Saturday the 27th of February 1937.

The following interesting clinical cases from the Government Head-quarters Hospital, Palamcottah, were demonstrated:—

1. Strangulated Hernia with a tumour.
2. Compound fractures of Tibia and Femur.
3. Duodenal ulcer with kala-azar.
4. Paralytic ileus of colon.
5. Compound fracture of humerus.
6. Duodenal ulcer with many bleedings.
7. Cystic Goitre—very big size—The excised tumour was shown.

Dr. V. Rama Kamath's letter inviting the Association to be present at a dinner of Unification of the Medical Profession in Madras was read out and discussed. The general opinion elicited was that the profession should join and strengthen some common organisation like the Indian Medical Association and enable it to work steadily and vigorously.

Dr. P. S. Srinivasan, of the Government Head-quarters Hospital Palamcottah, read out case-notes of the above seven cases operated on by Lieut. Col. T. S. Shastri, I.M.S., They were fully discussed by the members. Then the President brought the meeting to a close by his remarks on the subjects and summed up the salient and important features of all the cases. He thanked Dr. G. N. Kantayya for his kindness in organising this most enjoyable function and entertainment.

Thereafter the excellent "Association Dinner" brought the happy gathering to a close late at night.

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'Tabloid' Brand Sulphonamide—P. 0.5 gm.—(p-amino-benzenesulphonamide), manufactured by Burroughs Wellcome & Co. London.

'Tabloid' Brand Sulphonamide—P. 0.5 gm., presents p-amino-benzenesulphonamide for administration by mouth in haemolytic streptococcal infections. It has been shown experimentally that, following administration of this chemotherapeutic substance, the blood of man and animals becomes bactericidal to haemolytic streptococci.

Before deciding to use 'Tabloid' Sulphonamide—P in the treatment of severe streptococcal infections, such as puerperal septicaemia, erysipelas, etc., it is suggested that patients should be submitted to full bacteriological examinations to establish clearly the presence of haemolytic streptococci in the lesion or in the blood stream. 'Tabloid' Sulphonamide—P should be administered orally in a total daily dose of six to ten 0.5 gm. products after meals, the full dosage being continued for six days after the temperature becomes normal. In the event of some gastric disturbance following large dosage—the only troublesome symptom so far reported—the dose might be reduced temporarily. 'Tabloid' Sulphonamide-P may be tried intramuscularly when oral administration is found to be impossible. In such a case the low solubility of the substance (1.2 per cent.) makes the total amount that can be conveniently given by this method undesirably small. For intramuscular injection one 'Tabloid' product should be boiled up in 40 c.c. of 0.7 per cent. Saline. and the solution cooled to body temperature before injecting.

'Tabloid' Brand Sulphonamide-P, 0.5 gm., is issued in bottles of 25 and 100.

CORRESPONDENCE

College of Physicians and Surgeons of Bombay

D. K. Bharucha, Esq., Secretary, College of Physicians and Surgeons of Bombay, writes:—

"I have the honour to state for your information that the Council of the College of Physicians and Surgeons of Bombay at their meeting held in January 1937, decided to institute an examination for the Diploma in Ophthalmic Medicine and Surgery

and appointed a Committee to draw up regulations and syllabus of study for that examination.

The Committee submitted its report to the Council at their meeting held on the 12th March 1937, when it was resolved that the report be approved and adopted; and that the first examination for the Diploma in Ophthalmic Medicine and Surgery be held in July 1937.

A copy of the regulations is forwarded herewith for information."

Regulations Relating to the Examination for the Diploma in Ophthalmic Medicine and Surgery

The College of Physicians and Surgeons of Bombay shall grant a Diploma in Ophthalmic Medicine and Surgery (D.O.M.S.).

2. The seal of the College shall be affixed to every Diploma.
3. The examination shall be held annually in Bombay on the last Monday in July.
4. The examination shall be open to all those registered with the Bombay Medical Council*.

5. Candidates must apply to the Secretary of the College for permission to appear at the examination at least two weeks before the date of commencement of the examination, and shall at the same time pay a fee of Rs. 100. Fees once paid shall not be refunded, but may be reserved for the subsequent examination only at the discretion of the College Council, provided that the application for such reservation is made at least seven days before the date of the commencement of the examination and good and sufficient reason is shown for the same. A candidate who fails to pass or to present himself at an examination may be admitted to one or more subsequent examinations on a fresh application being made and a fresh fee being paid.

6. No candidate will be admitted to the examination unless he produces the following certificates:—

- (a) of having attended as a post-graduate for a period of nine months the clinical practice of an Ophthalmic Hospital or the Ophthalmic Department of a general hospital (having at least 20 Ophthalmic beds) recognized by the College of Physicians and Surgeons.

* Candidate shall, at the time of applying for permission to appear at the examination, produce a certificate stating that he is registered with the Bombay Medical Council.

The conditions of this certificate will be fulfilled by holding an appointment as House Surgeon or House Physician or as clinical assistant or as post-graduate at one of the recognised Ophthalmic hospitals or Ophthalmic department at a general hospital.

- (b) of having attended the following courses for a period of three months at an institution recognised by this College for the purposes of the M. C. P. S. Examination:—

- (i) a course of study in Optics;
- (ii) a course of study in the Anatomy and Physiology of the Eye;
- (iii) a course of study in Pathology and Bacteriology with special reference to Ophthalmic Medicine and Surgery;
- (iv) a course of Ophthalmic Operative Surgery.

7. The examination shall be written, practical, clinical and oral. There shall be four papers, each of three hours' duration and each carrying 100 marks in each of the following subjects:—

- (a) Anatomy and Physiology of the Eye and Optics;
- (b) Bacteriology and Pathology of the Eye;
- (c) Ophthalmic Medicine and Surgery;
- (d) Relation of Ophthalmology to general Medicine.

There shall be practical, clinical and oral tests, carrying 400 marks as under:—

(i) Clinical Examination	... 100
(ii) Dark Room Examination	... 100
(iii) Pathological Specimens of the eye-ball and microscopic slides	... 100
(iv) Ophthalmic Instruments, appliances and viva voce	... 100

8. To pass the examination, the candidate must obtain 40 per cent. of the full marks in each paper, 50 per cent. of the full marks in the practical, clinical and oral tests taken together.

9. The subjects for study shall be—

- (a) Anatomy and Embryology of the Eye, including the contents of the orbit, bones in relation thereof and the central nervous system as far as it relates to the Eye;
- (b) Physiology of Vision;
- (c) Physiological Optics;
- (d) Errors of Refraction;

- (e) Ophthalmic Medicine and Surgery;
- (f) Pathology and Bacteriology of the Eye;
- (g) Relation of Ophthalmology to general Medicine;
- (h) Operative Surgery.

10. Every person before receiving his diploma in Ophthalmic Medicine and surgery shall make and sign the following declaration:—

"I.....do solemnly and sincerely declare that holding the diploma of the College of Physicians and Surgeons of Bombay, I will obey every lawful order of the Council of the College, and that I will demean myself honourably in the practice of my profession, and to the utmost of my power maintain the dignity and honour of the College."

Dr. A. R. Choudhury, Naagan, writes to us as follows:—

The following is a "quotation" picked up from the old collections in connection with "Cobra Venom in Modern Practice"—By Capt. J. Singh Chouhan, Calcutta, appearing in *Antiseptic*, February, 1937.

Crotaline.—Syn. Cobra Venom.—Dose—Hypodermically— $\frac{1}{200}$ grain, gradually increased to $\frac{1}{15}$ grain. (0.0003 to 0.0026 gm.).

In epilepsy $\frac{1}{200}$ grain and then 3 to 5 similar injections at 8 day intervals, then two of $\frac{1}{15}$ grain with 14 day intervals—these generally would suffice, if not, one or more $\frac{1}{15}$ grain doses at monthly intervals; if even then, symptoms have not disappeared, give $\frac{1}{15}$ grain dose if necessary. Modify according to age and nature of the case.

Prepared from the venom of *Crotalus Horridus* (Rattle snake). The venom of the living reptile is desiccated by drying under a bell jar in the sun. Ampoules are prepared containing a glycerine and water solution with tricresol added as preservative.

'Self' in the *Medical World* in 1908, made the observation that people bitten by snakes remain proof against attacks of Epilepsy for years.

R. H. Spangler of Chicago claims, that epilepsy can be cured by the injections. He observed that an epileptic in Texas was cured by a bite from a Rattle snake. *Crotaline*—appears to act on the central nervous system, the condition of the blood and metabolism of the affected person. The period of coagulation of the blood is lengthened.

Spangler claims to have succeeded, with the venom treatment, in increasing the time of coagulation in eighteen patients from about two and a half minutes to about six minutes, the normal period being about seven minutes.

A case is reported of a girl, eleven years of age, who took the treatment for seven months. The first week she had 3 attacks in a period of 24 hours.

But there were only three minor attacks during the rest of the term of the treatment, when she was declared perfectly well.

In lung affections—Phthisis, and Asthma, has also been tried subcutaneously, seldom given internally".

Reference:—See also Calmette and Mezie *compt. Rend.* 1904, 158,846. P. J. 1/14,609.

NOTICE

International Union against Tuberculosis Press Communiqué

The Executive Committee of the International Union against Tuberculosis, General Secretary, Professor, Fernand Bezancon, met in Paris on 9th January 1937, at 2-30 p. m. The main object of this meeting was to come to a decision concerning the date of the 10th Conference of the International Union which should have taken place in Lisbon in September 1936 under the Chairmanship of Professor Lopo de Carvalho and which, owing to unforeseen circumstances, had to be adjourned.

It was agreed that the 10th Conference should take place in September 1937, either at the beginning of the month, should it be possible to meet in Lisbon, or at a date to be decided later should the Conference have to be held in another city.

In their wish to pay due regard to the active and intelligent endeavours of their Portuguese Colleagues who, under normal conditions, would have been entitled to expect a most successful meeting, the Members of the Committee expressed the hope that the Conference could take place in Lisbon next September, the final decision to be taken only towards the end of April or the beginning of May 1937.

The Executive Committee on the other hand decided that the 11th Conference of the International Union against Tuberculosis should meet in 1939, instead of 1938, in Berlin, subject to the statutory approbation of the next General Assembly of the Union.

The Members of the Committee were unanimous in expressing their gratitude to the Italian Government for having placed at the Committee's disposal six scholarships at the Carlo Forlanini Institute in Rome.

They approved the Regulations of the Leon Bernard Memorial Fund according to which a prize shall be awarded every other year by the Executive Committee to the author of an essay on Tuberculosis from a social point of view, written in French or in English.

Finally, it was decided that the Secretariate of the Union would ask the Councillor Members in the different countries to select certain scientific questions which would be proposed for research and which might eventually be inscribed on the agenda of future Conferences of the Union.

The Coronation

Medical men, visiting London for the Coronation, will naturally wish to take the opportunity to familiarize themselves with recent developments in all branches of their calling.

British manufacturers of medical equipment will most cordially welcome any opportunity they may be afforded to acquaint visitors with their latest productions.

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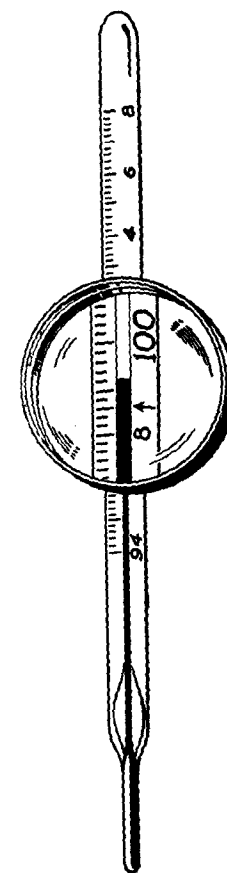
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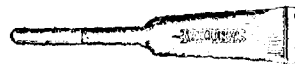
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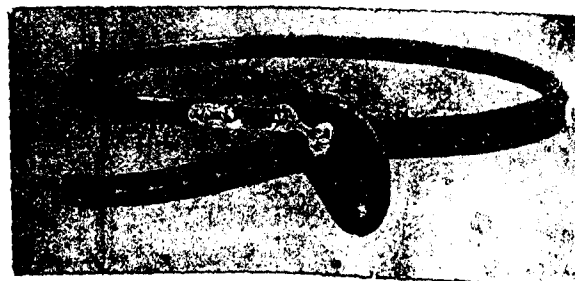
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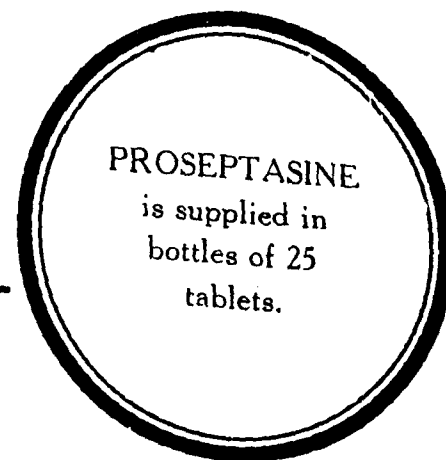
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