HANSEN’S DISEASE (LEPROSY)

Revised 08/22/2010

Leprosy is a chronic, mildly communicable disease of man which primarily affects the skin, mucous membranes, peripheral nerves, eyes, bones and testes due to Mycobacterium leprae, an acid fast bacillus related to the agent of tuberculosis.

Hansen's Disease is synonymous to leprosy. Because modern day leprosy is erroneously associated with the biblical leprosy, patients suffering from leprosy are often ostracized and discriminated. A strong movement developed in the Western hemisphere to substitute the name Hansen's disease to leprosy. In many countries the word leprosy is still currently used. However the word leper carries a pejorative connotation and should no longer be used in medical texts.

Epidemiology

Skin to skin transmission has long been suspected to be the main route of transmission. Although bacilli are present in very large number in ulcers, they cannot be found on the unbroken skin. There are a few anecdotal cases of skin transmission: inoculations during surgical procedures and tattooing. Insects have been suspected but careful studies have shown that their role in transmission would only be a minor one (if any).

It seems that the airborne transmission is the more probable route of transmission. Nasal washings from untreated lepromatous cases have from 10,000 to 10,000,000 M.leprae. A majority of the lepromatous patients have bacilli in their nasal secretions. The primary infection site may be the respiratory tract or the skin. Aerosols with M.leprae have been successful in infecting immunosuppressed mice.

Leprosy is not very communicable. Only 5-10% of the population is susceptible to develop the disease. In the USA the attack rate among close contacts is around 5%. Lepromatous and borderline cases are infectious while indeterminate and tuberculoid are considered as non-infectious. For example, in a study done in 1935, the clinical leprosy attack rates per 1000 person-years were 0.83 for persons with no known contacts with leprosy cases, 1.60 for contacts of non-Lepromatous and 6.23 for contacts of lepromatous cases.

Patients who are adequately treated are no longer infectious a few weeks or months after treatment started. Studies of mothers adequately treated showed that leprosy occurs only among children born prior to the mother’s initiation of treatment.

Humans are the only hosts in most countries. In the south central part of the USA (Texas, Louisiana and Mississippi) armadillos are naturally infected by M.leprae. Prevalence of infection in armadillos range from 4% to 30%. The armadillos seem to play a role in the resurgence of leprosy in Louisiana.

The disease is not hereditary, but a genetic susceptibility may be inherited as shown by the aggregation of cases in some families. Twin data seem to indicate that the concordance of the types of leprosy is high-
er among pairs of identical twins than among pairs of fraternal twins. HLA linked genes seem to control the type of disease that develops.

**Incubation period** : The bacillus reproduces at a very slow rate and therefore the incubation period is an average of 3 to 5 years. It is difficult to find out precisely the incubation period because exposure time and degree of exposure are impossible to determine. Few cases were diagnosed in infants less than 1 year, for other cases the incubation may have lasted 20 years or more.

**Clinical Description**

**Infection with M.leprae** is detected by lymphocyte transformation test, immunogel diffusion, radioimmunoassay and fluorescent antibody absorption test (FLA-ABS). An ELISA test using a highly specific phenolic glycolipid (PGL) antigen of M.leprae is more sensitive but slightly less specific than the FLA-ABS. Although the results of these tests are diverse, they show that infection with M.leprae is far more common than it is generally accepted: From 5% to 50% of contacts. A positive ELISA is present in 95% of cases 1 or 2 years before the development of clinical signs. There is good evidence that a high proportion of early clinical leprosy (up to 12%) will resolve spontaneously.

**Infection**

<table>
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<tr>
<th>Minority susceptible to leprosy disease (5%)</th>
<th>Majority will not develop disease</th>
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**Indeterminate**

- Good resistance: No bacilli
- Skin-Nerve: Tuberculoid

| Borderline |
| No resistance: Numerous bacilli |
| Skin-Nerve-All organs: Lepromatous |

**Indeterminate type**

The indeterminate type is the earliest state of the disease. It consists of a single hypopigmented or erythematous skin lesion which may or may not be insensitive. A skin smear is usually found to be negative. The indeterminate type may heal spontaneously, remain indefinitely at this stage or progress into one of the other types. To confirm a diagnosis of indeterminate leprosy there must be impairment of sensibility (thermal sensibility gets impaired before tactile sensibility). Biopsies are useful to support the clinical diagnosis.

**Tuberculoid type**

In the tuberculoid type there are few (1 to 3) skin lesions; these are large (3 to 30 cm) macules. The lesions are hypopigmented or erythematous. They have a well defined border and are rough and scaly. The periphery is raised and erythematous or hyperpigmented; the center is flat and hypopigmented.

Anesthesia or hyperesthesia is a major characteristic of these lesions. The sensation of temperature, light touch and pain are impaired in that order. Deep sensation is intact and deep tendon reflexes are normal. Loss of the ability to perspire is an important sign. Peripheral nerve involvement is common. The main nerves involved are the ones in the periphery of the lesions, the ulnar above the elbow, the median at the wrist, the common peroneal at the knee, the posterior tibial at the ankle, the radial at the elbow, the greater auricular in the neck, the sural at the lateral aspect of the ankle, and the superficial branches of the V and VII cranial nerves.

Although tuberculoid Leprosy is a relatively benign disease, severe disabilities may develop.
Lepromatous type

The skin lesions are very numerous small macules. They appear copper colored or erythematous. Their margin is vague and they tend to coalesce. They are symmetrically distributed over the body. They are not necessarily anesthetic. There is a generalized infiltration of the skin, often noticed by palpation rather than by visual inspection. Nodules and papules which are characteristic of lepromatous Leprosy are usually numerous. A common site for the development of nodules are the earlobes. Loss of eyebrows (starting with lateral brows) and loss of eye lashes (madarosis) are a common late signs. Alopecia of the scalp is rare.

As the disease progresses, most organs may become involved: Nasal congestion, epistaxis, laryngeal inflammation, eye lesions, renal involvement, testicular atrophy and secondary gynecomastia. Nerve involvement has a slow onset and becomes more generalized to eventually affect all nerves.

Borderline Type Or Dimorphous Type

Clinically and histologically borderline Leprosy has features of both the lepromatous and tuberculoid types. The majority of leprosy cases are borderline, but for particular purposes, they are often classified as Tuberculoid or Lepromatous (T or L). The word dimorphous is sometimes used instead of borderline.

Skin lesions are a mixture of T or L lesions, with a tendency to be more erythematous. The disease is unstable and may progress towards a more polar form (T or L).

Paucibacillary-Multibacillary Cases

Paucibacillary cases are the smear negative I and T cases of Madrid classification (I, TT and BT of Ridley Jopling classification) with a BI=0 at all sites.

Multibacillary cases are L and B cases of Madrid classification (LL, BL, BB and BT of Ridley Jopling classification) with a BI>0. In previous definition, paucibacillary had a BI of 0 or 1 but this was reduced in the latest definitions because of the poor results obtained by therapy in the BT group who had BI of 1.

Ulceration:

Ulcers are the result of repeated trauma on the anesthetic areas on the skin. The feet may be injured by poorly fitting shoes. The patient walking barefooted will often injure his feet and be unaware of the injury. The anesthetic hands are also exposed to injury such as burns while smoking or cooking, and traumas while working will ill fitted tools. Secondary infection of the ulcers is common.

Eye involvement:

The hypoesthesia of the cornea, paralysis of the eyelid muscle, inflammation of the tear duct and lacrosse gland will contribute to the development of lesions due to exposure of the eye. Lagophthalmos is the incomplete closure of palpebral tissue when lids are shut. In lepromatous leprosy, beading of corneal nerves may be a very early sign. The ciliary body, iris, and cornea are directly involved. As the disease progresses, it will lead to loss of visual acuity, cataract development, glaucoma and eventually blindness.

Nose involvement:

Rhinitis and nose bleeds are common in early lepromatous leprosy. The thick mucous fluid contains large numbers of bacilli and is probably a major source of infection.
Peripheral nerve involvement:

Sensory and motor impairment of the peripheral nerve will lead to the following deformities:

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Deformities</th>
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| Ulnar nerve | - Clawing of fingers IV and V  
              | - Adductor weakness of thumb  
              | - Pinch impairment            |
| Median nerve| - Clawing of fingers I and II always associated  
              | - Opposition and adduction of (to ulnar) thumb impairment  
              | - Abnormal grasp mechanism    |
| Lateral Popliteal nerve | - Loss of dorsiflexion  
                      | - Loss of eversion             |
| Posterior Tibial nerve | - Clawing of toes               |

Bone involvement:

Secondary infection leads to osteomyelitis and ultimately to bone absorption. Because fingers and toes are the areas most affected, there is partial loss of phalanges occasionally leading to total loss of digits. Direct invasion of bones by *M. leprae* may provoke bone destruction. The nasal bones and hard palate are often involved. Concomitant mucous membrane involvement may result in the perforation of the nasal septum.

Inactive Leprosy

The diagnosis of inactive leprosy is important. It means that the disease process has been stopped. The purpose of continued treatment is to consolidate this stage and to prevent later relapses. The patient is no longer infectious. Although physical examination, skin smear and biopsy are all used for the diagnostic, subjective diagnoses are sometimes made depending on clinical and histological impressions. The following criteria are required:
1. Negative skin smears for one year
2. Absence of clinical activity and/or reactions
3. Negative skin biopsy after one year.

Note: Inactivity does NOT mean that all *M. leprae* have been killed in the body. It means that they cannot be found by the usual methods; however, special studies may show the existence of some viable bacilli.

Diagnosis

A complete history, physical examination and laboratory tests are the keys to the diagnosis of leprosy. Most leprosy cases either have lived in an endemic area or have been in close contact with a known case of leprosy.

Sensory Changes, chiefly anesthesia, are the most common symptoms reported by the patients.

The key to the diagnosis is a thorough clinical examination.
- The total skin area should be examined carefully. Use the brightest available natural light, side lighting may be useful. Examine from close and far distance.
- Light touch can be tested by touching the skin with a wisp of cotton, while the patient's eyes are closed. Some surface areas with thick callous skin are normally less sensitive: elbows, knees, soles, fin-
gers of manual workers. Stroking a hair may cause a sensation. Test tubes with hot and cold water may be used to test temperature perception.
- Palpate for enlarged nerves especially the areas where a superficial nerve may be involved. Watch for wincing during nerve palpation, indicating pain.
- Examine the extremities for contracture and trophic changes.
- Look for a Motor deficit:
  --Facial Nerve - Blink reflex is normally noted when the examiner waves his hand before the eyes.
  --Ulnar, Median, and Radial Nerves - The patient is asked to approximate the tip of the thumb to the tip of the straight little finger with the hand outstretched in the pronated position.
  --Lateral Popliteal Nerve - Patient is asked to dorsiflex the great toe against resistance.
  --Posterior Tibial Nerve- The patient is asked to spread the toes.
- A detailed examination of the eye is necessary. Check the eyebrows, eyelashes, motricity of the eyelids, the cornea, conjunctiva and iris.
- Inspect the mouth and throat.
- Examine the earlobes.

Skim Smear

A skin smear may demonstrate the presence of acid fast bacilli in the skin. If bacilli are located in the deep layers of the skin or in the nerves they will not be found on the smear.

The bacilli in the skin are quantified with the Bacteriological Index (BI). It ranges from 0 to +6 according to the number of bacilli per oil immersion field at microscopic examination. The rate of clearance under treatment is approximately 1 per year.

The Morphological Index (MI) is the percentage of solidly stained bacilli of normal size and shape. These bacilli are thought to be the viable ones, that is the ones that are alive and may infect someone. There are problems in standardization and reproducibility that cause the index not to be routinely used in practice.

Interpretation of BI and MI: Indeterminate and Tuberculoid leprosy may have a negative or 1+ BI. A nontreated advanced lepromatous may have a 4 to 6+BI with MI from 10 to 50%. A Borderline may show lower BI and MI. With an efficient treatment the MI will go to 0% rapidly and the BI will generally fall by 1+ every 1 or 2 years. Subjective interpretation by individual technicians vary considerably. Therefore, it is important that the same person perform the microscopic examination to insure consistency in reporting.

A skin smear may be useful for diagnosis: when positive it indicates leprosy but skin smears are used primarily for the surveillance of the patient's bacteriological status.

Technique of the skin smear:
A skin smear may be taken on any suspect lesion, or on the earlobe or eyebrow, the elbow or the knee. This procedure is easy and totally harmless. It can easily be done by physicians, nurses and lab technicians.

- Pinch the skin to reduce blood flow.
- Wipe the area with an alcohol sponge.
- Make a small slit with a sterile scalpel blade or razor blade approximately 5mm long and 2mm deep.
- Wipe away any blood which exudes.
- Scrape the edges of the wound with the blade.
- Spread the materials obtained on a microscopic slide. Obtain as little blood as possible.
Skin Biopsy

Indications for biopsy: Possibility of Hansen’s Disease, including: Long-standing skin lesions, not responding to conventional topical treatments, skin lesions in persons with history of travel/residence in developing country

Biopsy:
Select the most active margin of the most active lesion
Obtain a full-thickness biopsy – it is important to see a bit of subcutaneous fat.
Either elliptical or punch biopsy is OK (4 mm punch is sufficient)

Fixation and processing:
Routine 10% neutral buffered formalin
Send to NHDP in formalin OR embedded in paraffin by your pathology laboratory
Documentation: basic patient information and clinical questions, including DOB or age. Patient treated?
If yes, how long? Use of steroids?
Address: Clinical Laboratory, National Hansen’s Disease Programs
1770 Physician Park Dr., Baton Rouge, LA 70816, Tel 225-756-3733

Examination of a skin biopsy gives the definitive diagnosis and classification of the type of leprosy. The edge of skin lesions or the nodules are considered to be the best sites for obtaining a biopsy. The biopsy is made with a punch or by surgical incision. It should be deep enough to include subcutaneous fat. The specimens are best preserved in neutral buffered formaldehyde solution (a solution containing phosphate buffer and 10% of formaldehyde 37-40%).

Cultivation & Animal Models

It has not yet been possible to culture the *M. leprae* in vitro. It only multiplies and produces disease in a very limited number of animal species. The nine banded armadillo is the only source of large amounts of *M. leprae* for research and production of vaccine. Inoculation of the normal mouse foot pad is the basic tool used to study *M. leprae* drug resistance and the protective effect of candidate vaccines. Immunodeficient mice (thymectomized, irradiated, bone marrow reconstituted, nude) or neonatally thymectomized rats are also used. Several primates can also be experimentally infected.

Surveillance

Hansen’s disease is a condition reportable within 5 working days of diagnosis.

Case Definition

Clinical description

A chronic bacterial disease characterized by the involvement primarily of skin as well as peripheral nerves and the mucosa of the upper airway. Clinical forms of Hansen's disease represent a spectrum reflecting the cellular immune response to *M. leprae*. The following characteristics are typical of the major forms of the disease:

- **Tuberculoid**: one or a few well-demarcated, hypopigmented, and anesthetic skin lesions, frequently with active, spreading edges and a clearing center; peripheral nerve swelling or thickening also may occur
- **Lepromatous**: a number of erythematous papules and nodules or an infiltration of the face, hands, and feet with lesions in a bilateral and symmetrical distribution that progress to thickening of the skin
Borderline (dimorphous): skin lesions characteristic of both the tuberculoid and lepromatous forms
Indeterminate: early lesions, usually hypopigmented macules, without developed tuberculoid or lepromatous features

Laboratory criteria for diagnosis

Demonstration of acid-fast bacilli in skin or dermal nerve, obtained from the full-thickness skin biopsy of a lepromatous lesion

Case classification
Confirmed: a clinically compatible case that is laboratory confirmed

Treatment

Following are the general NHDP recommendations. NHDP recommendations are for daily rifampin, and for longer duration of treatment than the WHO recommendations, largely due to WHO’s cost considerations for developing countries. Treatment that is more intensive and of longer duration is medically preferable.

Treatment guidelines for immunologically competent individuals, (e.g. those without immunodeficiency, immunosuppression, prolonged corticosteroid use, etc.) are as follows.

<table>
<thead>
<tr>
<th>Adults</th>
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<tbody>
<tr>
<td><strong>Tuberculoid (TT &amp; BT) (WHO classification Paucibacillary, “PB”)</strong></td>
</tr>
<tr>
<td><strong>Agent</strong></td>
</tr>
<tr>
<td>Dapsone</td>
</tr>
<tr>
<td>Rifampicin</td>
</tr>
<tr>
<td><strong>Lepromatous (LL, BL, BB) (WHO classification Multibacillary, “MB”)</strong></td>
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<td>Rifampicin</td>
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<tr>
<td>Clofazimine b</td>
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- The recommended durations of treatment are sufficient, even though large numbers of dead bacilli may remain in the tissues for several years, before they are eliminated by physiological processes. There is no evidence that additional, prolonged treatment hastens the elimination of these dead organisms.

- Clofazimine, used for decades to treat HD around the world, is no longer available on the open market. Because it is no longer distributed commercially, the only way we can obtain the drug in the U.S. is to once again treat it as an investigational new drug (IND). The NHDP holds this IND for its use in treating HD in the U.S.

In order for physicians to obtain the drug for treating HD, they will have to be registered as an investigator under the NHDP IND. This will require submitting a signed FDA form 1572 and a curriculum vitae to the NHDP. A packet of information including the form 1572 as well as consent forms, etc., will be provided. An Institutional Review Board (IRB) of the Centers for Disease Control has agreed to act as the central IRB for the use of Clofazimine for Hansen's Disease, so that individual physicians do not need to arrange this themselves. For further information, or to request investigator status to use Clofazimine, please call the NHDP at 1-800-642-2477.
For immunologically compromised patients, these protocols may be modified, and consultation with the NHDP is recommended.

**Alternative Anti-Microbial Agents**

- **Minocycline**, 100 mg daily, can be used as a substitute for Dapsone in individuals who do not tolerate this drug. It can also be used instead of Clofazimine, although evidence of the efficacy of its anti-inflammatory activity against Type 2 reactions is not as substantial as the evidence for Clofazimine.

- **Clarithromycin**, 500 mg daily is also effective against M. leprae, and can be used as a substitute for any of the other drugs in a multiple drug regimen.

- **Ofloxacin**, 400 mg daily, may also be used in place of Clofazimine, for adults. This is not recommended for children.

In the United States, the occurrence of leprosy in children is rare. We strongly recommend contacting the NHDP for management of leprosy in children; the following are general guidelines.

<table>
<thead>
<tr>
<th>Treatment for children</th>
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<tbody>
<tr>
<td><strong>Tuberculoid (TT &amp; BT) (WHO Paucibacillary, “PB”)</strong></td>
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<tr>
<td><strong>Agent</strong></td>
</tr>
<tr>
<td>Dapsone</td>
</tr>
<tr>
<td>Rifampicin</td>
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| **Lepromatous (LL, BL, BB) (WHO Multibacillary, “MB”)** |
| **Agent** | **Dose** | **Duration** |
| Dapsone | 1 mg/ Kg daily | 24 months, and then therapy discontinued |
| Rifampicin | 10-20 mg/ Kg daily (not > 600) | |
| Clofazimine | 1.0 mg/Kg daily | |

As there is no formulation less than 50 mg, and the capsule should never be cut open, alternate day dosing may be used at 2 mg/kg.

-Dapsone Monotherapy is no longer recommended, however some countries still continue using it.

**Disappearance Of M.Leprae:**

The lepromatous patient placed under an effective treatment will become non-infectious in a few weeks. However, it usually takes 3 to 10 years before M. leprae will completely disappear from a skin smear. Even though the skin shows no evidence of the presence of bacilli, some may persist in the peripheral nerves and internal organs.

**Treatment of leprosy during pregnancy and lactation**

Leprosy is exacerbated during pregnancy, so is important that the standard multidrug therapy be continued during pregnancy. The Action Program for Elimination of Leprosy, WHO, Geneva has stated the standard MDT regimens are considered safe, both for the mother and the child, and therefore, should be continued unchanged during pregnancy. A small quantity of antileprosy drugs is excreted through breast milk but there is no report of adverse effects as a result of this except for mild skin discoloration of the in-
fant due to clofazimine. The single dose treatment for patients with single lesion paucibacillary leprosy should be deferred until after delivery.

**Treatment of patient with concomitant active tuberculosis.**

If the patient has both leprosy and active tuberculosis, it is necessary to treat both infections at the same time. Give the appropriate antituberculosis therapy, addition to the antileprosy multidrug therapy for the type of leprosy in the patient. Rifampicin is common to both regimens and it must be given in the doses required for tuberculosis.

**Treatment of patients with concomitant HIV infection**

The management of a leprosy patient infected with HIV is the same as that of any other patient. The information available so far indicates that the response of such a patient to MDT is similar to that of any other leprosy patient and management, including treatment reactions, does not require any modifications.

**Reactions**

More than 25 percent of patients with Hansen's disease (leprosy) may have reactive episodes ("reactions") of varying degrees of severity during the course of their disease. Some of them occur before treatment is started or after therapy is completed, but most occur during therapy, particularly during the first year.

Reactive episodes appear to be less common in patients treated with clofazimine. Reaction should not be regarded as a side effect of any drug, rather it is apparently due to the destruction of bacilli and the immune response to released bacterial antigens. Chemotherapy should be continued in spite of reactive episodes but with the episodes themselves suppressed as needed by other therapy.

Reactions can be broadly divided into two main categories: erythema nodosum leprosum (ENL or type 2 reactions) occurring almost exclusively in borderline lepromatous and lepromatous patients, and reversal (type 1 reactions), occurring in borderline tuberculoid, mid-borderline and borderline lepromatous patients. A third type of reaction known as the Lucio's phenomenon is relatively rare. It occurs in patients with diffuse lepromatous leprosy who are from Mexico and some other areas. The patients develop multiple ulcers of varying size that are often difficult to heal. It is only occasionally seen and is managed with corticosteroids and treatment of the underlying infection.

**Erythema Nodosum Leprosum**

Erythema nodosum leprosum usually manifests with fever and painful erythematous nodules, but peripheral neuritis, orchitis, lymphadenitis, iridocyclitis, nephritis, periostitis and arthralgias may also occur. Mild episodes may require no therapy, or symptomatic measures such as aspirin administration may suffice. Several drugs are useful for the management of severe episodes.

**Corticosteroids** are effective in all patients and should always be used if an acute neuritis is present to prevent permanent nerve injury. Usually, 40-60 mg of prednisone daily (dosage of 1 mg/kg per day) is sufficient. When the initial episode has been completely controlled for several days, an attempt may be made to taper the drug dosage over a period of 2 - 4 weeks. Reaction often recur, however, and the dosage may be increased. If the process becomes chronic, prolonged therapy may be needed. In these patients it may be useful to try tapering the prednisone to alternate day therapy. The dosage is reduced still more slowly until either the drug is eliminated or the lowest possible maintenance level is reached. However, because steroid-associated side effects are often a problem, other forms of therapy should be considered in chronic cases. When prolonged use of corticosteroid is required, giving the rifampin only once monthly as in the World Health Organization regimens should be considered to avoid its adverse effect on corticosteroid levels.
Thalidomide (Thalomid) is extremely effective in treatment of ENL. The initial regimen is 100 mg four times daily, and reaction is usually controlled within 48 to 72 hours. The dosage is then tapered over 2 weeks to a maintenance level, usually 100 mg daily. Regular attempts should be made to taper or discontinue the drug, but patients may need to continue taking thalidomide for months to years before ENL reactions no longer recur. Side effects are few with drowsiness being the most common. Thalidomide absolutely cannot be given to fertile females because of its well known teratogenicity, except under strict conditions outlined by the manufacturer.

Clofazimine is also effective for the control of ENL. A dose of 100 mg two or three times daily usually is necessary, and the reaction should come under control during a period ranging from a few weeks to a few months, depending on its severity. Normally, reaction control is maintained with prednisone in these patients, and the dosage of prednisone is gradually diminished as the clofazimine begins to act. Because gastrointestinal symptoms may develop with high doses, clofazimine dosage should be reduced to 100 mg daily within a year, if possible. Pigmentation from clofazimine is usually quite marked in these patients. They should be fully cognizant of this complication before therapy is started, as well as educated about the potential benefits of this antibiotic.

**Reversal Reactions**

Clinically, reversal reactions are usually evidenced by edema and erythema of pre-existing lesions. Neuritis and occasionally new lesions or fever may also occur. If there is neuritis or ulceration, high doses of corticosteroids should always be used, e.g., prednisone at 1 mg/kg daily. This type of reaction usually is controlled within 24 to 48 hours, and only a short course of therapy may be necessary if the patient has minimally active disease and no neuritis. However, those with neuritis may require prolonged treatment (4 to 6 months) if neural damage is to be reversed. Patients with prolonged reactions may sometimes be managed with alternate day steroids as noted for ENL.

**Other Complications**

**Neuritis** or **silent neuropathies** (neuritis without nerve pain or tenderness) may occur independently of any reactive episode. Immediate treatment with high doses of corticosteroids is necessary to avoid permanent injury and recover lost function insofar as possible. Treatment should be continued for 4 - 6 months to recover as much function as possible using a gradually tapered course of corticosteroids.

**Iridocyclitis** (inflammation of the iris, ciliary body and choroid coat) is a medical emergency and best managed by an ophthalmologist. Atropine drops and corticosteroid drops must be started at once if permanent damage is to be avoided. Tear substitutes are used in patients with lagophthalmos and/or decreased lacrimation.

**Orchitis** (inflammation of a testis) may occur with or independently of a reactive episode. It usually responds quickly to corticosteroids but sterility may result.

**Injuries are common in all patients with Hansen's Disease who have significant degrees of sensory and motor loss.** The patient must be taught how to avoid them by frequent inspections of involved skin, and the use of protective measures such as wearing gloves or special footwear. When an injury does occur in an insensitive area, it must be protected from further damage during healing.

**Drug resistance:**

M. leprae may develop resistance to the drugs. The use of Dapsone began around 1948, the first cases of resistance were suspected in the early 60's and by 1975 it became obvious that dapsone resistance was a major problem. Sulfone resistance develops in perhaps 10% of lepromatous leprosy. It takes an average of 10 to 15 years to develop. It is more likely that patients taking low doses or frequently interrupting the
treatment will develop drug resistance. When rifampin is used alone, resistance may develop within a few years. To date, there is no evidence of developing resistance to clofazimine.

Suspect resistance or non-compliance when:
- The patient's condition does not improve over a period of 1 year, assuming other complications do not develop which could confuse the picture.
- There is no reduction in the patient's Morphological Index (MI) and Bacteriological Index (BI) over 1 or 2 years.

The mouse foot pad studies for the drug resistance allow confirmation of drug resistance. These are performed only in very specialized centers. Send a sterile, dry, 4 to 5 mm punch biopsy of a skin area with a BI of 4+ or more. Do not put any preservative in the sterile tube where the biopsy is. Pack in wet ice if delays in transportation are not too long.

**Leprosy Control**

The main objectives of a leprosy control program are:
1. To interrupt transmission of the infection, reducing the incidence of the disease so that it no longer constitute a public health problem.
2. To find new cases that are symptomatic or before symptoms develop.
3. To follow up all known cases and ascertain that they receive proper medical care and take regular treatment.
4. To prevent the development of associated deformities
5. To educate the patient, his family, the health professional, and the public.

In recent years, leprosy control programs have had to deal with increasing secondary and primary resistance of *M.leprae* to dapsone. The main strategy for control remains early detection of cases and chemotherapy.

**Identify Suspects** is the primary goal of case finding. The health practitioner comes in contact with many persons during the workday. They should be aware of leprosy and always suspect it if working in an endemic area. Suspects should be examined promptly by a person (physician, nurse) experienced in the diagnosis of leprosy.

ALL new cases should be treated, even paucibacillary cases. Although a proportion of these cases can heal without therapy, there is no way to tell which one will heal and which one will get worse.

Following the diagnosis of a new case, it is important to start a Case Investigation. The purpose of this investigation is to provide epidemiological information, baseline clinical information on the patient, and contact information for follow up. Collect information on the patient's identity, address, ethnic group, birth, education and employment. The places of residence should be investigated with great care. The information provided is essential to determine where the patient was contaminated, clinical information at the time of diagnosis. It is important to note the clinical type of leprosy since it will determine the length of treatment.

Programs should promote Self Detection through health education, as self reported patients generally are better compliers. Where multidrug therapy has been successfully implemented, self reporting has been improved.

**Bacteriological Examination** is highly relevant to leprosy control programs. The quality of smears and of microscopy is often the weakest link in most leprosy control programs. It is essential to train personnel in proper collection procedures for smears and to organize an efficient system for processing the smears.
Case Notification

New cases are reportable.

Containment

Isolation

Isolation was used for many centuries as the only way to prevent spread of the disease. It has not proven very efficient. Patients knowing they would be isolated for a long period of time avoided medical care and attempted to hide their disease. Although they were isolated when the diagnosis became obvious, it was usually very late. Most contacts were already contaminated. Infectious cases become noninfectious within a few weeks or months following the initiation of treatment. Isolation of the patient is no longer necessary. Common sense and hygienic precautions with regards to an infectious case are sufficient.

Hospitalization

Following the diagnosis of a new case, hospitalization is not systematically recommended before starting treatment. The major medical indication for hospitalization is in the event of a severe REACTION to drug therapy. Severe reactions may lead to severe disabilities and death. It is to the patient's benefit to be hospitalized when a reaction occurs. Cases that are diagnosed early with minimum signs of leprosy do not need to be hospitalized and may begin treatment as outpatients.

Follow Up Of Known Cases

One must understand the public's prejudices toward leprosy. Persons with leprosy have been unfairly and irrationally ostracized and sequestered for many years. An understandable and common reaction from patients is to refuse any control from anyone. A great deal of tact is necessary in dealing with these cases.

Compliance is a major problem in leprosy control. The old regimen of monotherapy with dapsone was partly unacceptable because of the requirements for extremely long treatment: 3, 5 years or lifetime treatment. With multiple drug therapy the length of treatment have been considerably shortened but are still very long in comparison with treatment for other diseases: 6 months, 2 years or longer.

Contact Examination

Close contacts who have suspect lesions need to be examined. Household contacts are considered to be at highest risk. They are all persons who have lived in a living unit with a patient.

Education

The success of an education program depend on the quality of the communication between the practitioner and the patient. The patient population reflects the cultural diversity of the country. Patients and practitioners employ different cognitive systems for understanding sickness resulting from differences in cultural and educational backgrounds.

Hospital precaution and isolation: Standard precautions