HRSA

NHDP Guide to the Management of Hansen’s Disease

National Hansen’s Disease Programs
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I. INTRODUCTION

Treatment has dramatically changed over the past century for people diagnosed with Hansen’s disease (HD or leprosy) in the United States. Confinement at the National Leprosarium in Carville, Louisiana was the only option before 1950. With the discovery of an effective drug protocol and the establishment of Ambulatory Care Programs (ACP) in select cities, patients are now treated in proximity to their homes. Travel to the National Hansen’s Disease Programs (NHDP) headquarters for treatment is no longer necessary unless patients experience complications that cannot be managed by local providers. Currently, in addition to the NHDP and ACP, approximately 375 private physicians are treating one or more HD patients in cities throughout the United States. Since the number of treatment providers for HD has increased, a standardized guide for the care of HD in the U.S. is essential.

A. Objective

The purpose of this manual is to serve as a practical guide for clinicians and healthcare professionals directly involved in the diagnosis and treatment of people with HD in the United States and its territories. It may also be a valuable tool as an overview of HD for medical students, residents and others interested in the disease. Clarification regarding the content of this manual may be requested from the NHDP at 1-800-642-2477 or found on the website: https://www.hrsa.gov/hansens-disease/index.html

B. Mission

The Division of National Hansen's Disease Programs (NHDP) is a medical service within the Department of Health and Human Services (HHS), Health Resources and Services Administration (HRSA), Healthcare Systems Bureau (HSB). NHDP is authorized by Public Law 99-177, Section 2, (a), Section 320 and is guided by DHHS regulations. “The mission of the NHDP is the treatment and eventual eradication of Hansen’s disease through its clinical, rehabilitation, research and training programs. It is the only program in the United States devoted exclusively to the management of HD and the only referral source where all associated complications can be managed.”

C. Eligibility

Individuals living in the United States, or its territories, may receive outpatient medical care for the diagnosis and treatment of HD and its complications. Ideally, individuals are diagnosed and treated locally; however, eligibility for patient care at the NHDP in Baton Rouge, LA is currently based on answers to the following questions:

1. Is the patient’s problem one that is related to HD?
2. Can this problem be solved locally for the patient?
3. Can the NHDP successfully treat this problem if the patient cannot receive local treatment?
4. Does the patient have any additional health complications that would interfere with treatment or require more intensive care than can be provided at the NHDP?

Contacts of known patients are also eligible for HD related services as described by NHDP policy (see “Contact Evaluation” in Section XIV).

II. HISTORY OF HANSEN’S DISEASE

The earliest written description of leprosy (HD) was recorded in India during the sixth millennium BC. The cause, however, remained unknown until 1873 when Dr. Gerhard Armauer Hansen (for whom the disease was renamed) of Norway identified *M. leprae* as the bacteria responsible for the disease. A variety of palliative therapies were tried, but none proved effective and most people were relegated to a life of isolation and abandonment. In 1941, sulfone drugs were first used to treat HD at the National Leprosarium in Carville, Louisiana, and by 1950; dapsone had become the standard treatment. The rapid improvement in patients treated with dapsone therapy led to hopes of the eradication of HD, and the search began in earnest for even more powerful drugs. However, there were no effective animal models at the time and *M. leprae* still cannot be cultivated in vitro. In 1960, Charles Shepard described the limited multiplication of *M. leprae* in the mouse footpad, which made it possible for the first time to screen drugs for anti-leprosy activity and detect drug resistance. Since then, armadillos, nude mice, and a variety of immunosuppressed rodents have been used to obtain higher yields of *M. leprae* for bacteriological and immunological studies. The World Health Organization (WHO) introduced multidrug therapy (MDT) to the global community in 1982.

Today, HD drugs are distributed free of charge around most of the world by the Novartis Foundation. NHDP provides medications at no cost to diagnosed patients living in the US and its territories. Because of these efforts, the global burden of disease has fallen dramatically in recent decades.

III. OVERVIEW OF *M. LEpraE*

A. BACTERIOLOGY

*M. leprae* is a weakly acid-fast rod, 2-8 microns long and 0.3 microns in diameter. It is an obligate intracellular organism, and can be found in tissues singly or in clumps (globi) which may contain hundreds of bacilli. *M. leprae* will survive for a short period in tissue cultures. Limited metabolism (but not multiplication) can take place in some types of special laboratory media. Consistent multiplication occurs following injection of viable bacilli into the footpads of mice, as well as in nine-banded armadillos.

The generally accepted generation time of *M. leprae* is approximately 12 days. This very slow rate of multiplication is consistent with the long (usually 3-5 years) incubation period of HD in humans.

*M. leprae* DNA can be identified in tissue biopsies by nucleic acid amplification techniques such as polymerase chain reaction (PCR). Genotyping of *M. leprae* DNA has demonstrated...
several major genotypes globally, and has revealed that armadillos and humans in the United States share some genotypes. *M. leprae* from biopsies can also be tested for mutations associated with drug resistance.

Since its discovery by Gerhard Armauer Hansen, *M. leprae* was thought to be the only infectious bacterium to cause leprosy. In December 2008, the American Journal of Clinical Pathology published an original article by X. Han, MD, PhD., et. al. documenting the discovery of a “new *Mycobacterium* strain” (Am J Clin Pathol 2008; 130:856-864). The comparison showed this “new strain” to be a new species of *Mycobacterium*. This new species has been named *Mycobacteria lepromatosis*. Cases of *M. lepromatosis* have been reported in Canada, Asia, several provinces of Mexico, South America and Central America (Jessamine 2012, Sotiriou 2016, Han 2014).

*M. lepromatosis*, is genetically distinct from *M. leprae*, but shares striking evolutionary and biologic similarities. *M. lepromatosis* manifests with the same broad spectrum of cutaneous and neurologic findings that are described in HD and is comparable to infection with *M. leprae*. Reactions-Type 1 reversal and Type 2 ENL can occur with both *M. leprae* and *M. lepromatosis*. There is insufficient evidence to determine if immunologic reactions with *M. lepromatosis* are more severe. *M. lepromatosis* could be the causative agent of several cases of DLL (Diffuse Lepromatous Leprosy) with presentations similar to that seen in patients who develop Lucio’s phenomenon (Han 2008). To date, no clear clinical or histopathologic differences between infections with the two species can be recognized, and thus differentiating them is mostly of epidemiologic value. Importantly, multi-drug therapy appears to be effective for both infections, and therefore, one diagnosis does not seem to portend a more ominous disease course than the other.

**B. IMMUNOLOGY**

1. **The Host Response**

   Only 3-5 percent of the world’s population appears to be susceptible to clinically detectable infection with *M. leprae*. Among susceptible individuals, a wide range of clinical manifestations is comparable to the spectrum of histopathological appearance; which is, in turn, based on a spectrum of human cell mediated immune (CMI) responses to *M. leprae*.

   At the Tuberculoid end of the spectrum there is good host CMI response that localizes the disease. At the Lepromatous end, CMI response to *M. leprae* is weak or absent and the disease is generalized, affecting skin, nerves, eyes, lymph nodes, muscle, and internal viscera such as the liver. In the middle of the spectrum, known as Borderline HD, various clinical presentations between the two extremes are seen, reflecting the variation in host responses. (The clinical –pathological classification of HD is described in Section IV.)

   Immunodeficiency due to HIV infection or to medical immunosuppression, such as transplantation or cancer chemotherapy, will render individuals more susceptible.
Similar increases in susceptibility may occur in individuals taking corticosteroids or newer biological agents for arthritis or autoimmune diseases.

2. **The Lepromin Test**

   The Lepromin skin test is not a diagnostic test and the reagent is no longer available. Historical information about this test can be found in the medical literature.

3. **Skin and Nerve Involvement**

   *M. leprae and M. Lepromatosis* have a particular affinity for skin and peripheral nerves. In the skin, they are found in macrophages where, in Lepromatous HD, it is able to avoid digestion and multiply to form large masses of bacilli ("globi"). In nerves, they are found within macrophages and Schwann cells. The presence of *M. leprae* and *M. Lepromatosis* in nerves is pathognomonic of HD.

C. **EPIDEMIOLOGY**

HD is found primarily in tropical and sub-tropical regions. Approximately 25 percent of the world’s population live in areas where they might be exposed to the infection. Because of stigma and other reporting issues, it remains difficult to get an accurate estimate of the global number of HD cases.

The NHDP is the federal agency primarily responsible for care, treatment and control of Hansen’s disease in the United States. The disease has been reported among individuals from every state. About 14,000 cases of HD have been recorded in the U.S. since 1894. Since the 1990’s, an average of 175 new cases are reported annually. The majority of new cases arise among immigrants to the United States from HD endemic countries, or citizens who have worked abroad in endemic areas. However, the single country contributing the most cases is the United States, that is, individuals born in the United States who have not traveled to endemic areas.

**Transmission:**

The exact mode of transmission of *M. leprae* is poorly understood. The evidence suggests that the optimal transmission occurs in a setting of prolonged, close contact, and not from casual contact. It is thought to be transmitted between people primarily via the respiratory route, but there is also some evidence for skin-skin transmission. However, the disease is not highly contagious. Approximately 95-97% of the world’s population appear to be naturally immune to HD.

Although man is considered the primary reservoir for leprosy bacilli, the nine-banded armadillo in the southern United States is also a reservoir for the infection. It is clear that transmission occurs between armadillos and humans, but the exact means of transmission is not known.
IV. CLASSIFICATION AND CLINICAL FEATURES

Extensive differences exist in the pathological features, immunological status, treatment, and types of complications that develop with HD. The aim of HD classification is to define zones in the spectrum of the disease in which these features are similar or different.

Two systems of classification are now in general use: the Ridley-Jopling five-group classification and the WHO two-group classification (see Table 1).

Table 1. The HD spectrum as defined by the Ridley-Jopling and the WHO classifications.

<table>
<thead>
<tr>
<th>Classification</th>
<th>Zones of the HD spectrum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ridley-Jopling</td>
<td>TT BT</td>
</tr>
<tr>
<td>WHO Classification</td>
<td>Paucibacillary</td>
</tr>
<tr>
<td></td>
<td>Multibacillary</td>
</tr>
</tbody>
</table>

A. The World Health Organization (WHO) Classification System

When the WHO introduced Multidrug Therapy (MDT), they recommended a simplified classification for treatment purposes with only two categories, Paucibacillary (PB) and Multibacillary (MB). The current WHO definition of MB disease is "any patient with six or more lesions with or without positive skin smear results". PB disease is defined by the WHO as "any patient who has less than six skin lesions with or without positive skin smear results".

B. Ridley-Jopling Classification System: The Clinical Spectrum of Hansen’s disease

Clinical features of HD cover a wide range, from a single hypopigmented skin macule to generalized disease. The Ridley-Jopling system captures this spectrum in 5 main classifications. The NHDP uses skin biopsy results and the Ridley-Jopling scale to determine the classification for diagnosis and treatment. (Figure 1) The indeterminate class of HD will be described in section C.
1. **Tuberculoid (TT)**

Tuberculoid HD is characterized by limited disease with the presence of a few, well-defined hypopigmented skin lesions with marked sensory loss. Loss of hair in the lesion is common and there is often central healing. In the absence of treatment, the lesions enlarge slowly. This type of HD may be self-healing. (Figure 2)

![Figure 2](image)

Histologically TT lesions reveal a very well organized epithelioid granuloma, with dense foci of lymphocytes. *M. leprae* are rare and hard to find in biopsies. Caseation can occur in nerves (Figure 11).

2. **Borderline Tuberculoid (BT)**

When resistance is high, but not as strong as TT, skin lesions look like TT lesions, but there are too many for the disease to be classified as polar TT.

(Figures 3 and 4)

![Figure 3](image) ![Figure 4](image)
Histologically, BT lesions also show granulomatous inflammation, but not as compactly organized as TT. *M. leprae* are present in low numbers (Figure 11).

3. **Mid-borderline (BB)**

In the mid-range of resistance, the lesions show a mixed appearance, some looking like BT and others like BL lesions. Usually, BB lesions have very clearly defined areas of central healing ("punched-out" areas) with somewhat less well-defined outer edges. This is a unique characteristic of mid-range BB HD, which is considered an immunologically unstable form where such patients tend to shift clinically toward BT or Borderline Lepromatous (BL). (Figure 5)

![Figure 5](image)

Histologically, BB lesions contain poorly organized granulomas as well as collections of foamy histiocytes. The acid fast bacilli (AFB) seen are not numerous but are not difficult to find (Figure 11).

4. **Borderline Lepromatous (BL)**

When resistance is lower, skin lesions look more like Lepromatous (LL) lesions with macules and nodules more sharply defined than in polar LL, and with areas of normal looking skin between them. There is usually some asymmetry of the lesions, whereas in polar LL, they are symmetrical. (Figures 6 and 7).

![Figure 6](image)  ![Figure 7](image)
Histologically, BL lesions reveal poorly organized collections of foamy macrophages and a substantial lymphocytic component. AFB are present in moderate to large numbers and are seen in almost every field, and may include clumps called ‘globi’ (Figure 11).

5. **Lepromatous (LL)**

Lepromatous HD represents the extreme of the spectrum where the patient essentially has no resistance to the infection and the bacilli multiply uncontrollably. The highest concentrations are in skin and nerves, but are not confined to these sites. There may be a bacteremia, even though the patient seldom feels ill. The skin lesions are numerous and may have vague margins. They may present as slightly hypopigmented macules, while in other cases the lesions may present as painless nodules or plaques. There is usually little or no loss of sensation in the skin in the early lesions. There is a generalized infiltration of the skin, usually maximized in the cooler zones of the body such as the extremities or the eyebrows. Extensive anesthesia can develop, but motor nerve function is often well preserved. (Figures 8, 9, 10).

The histological appearance in LL includes totally disorganized collections of foamy histiocytes with a small component of lymphocytes. AFB are present in very large numbers, often with many globi. Bacilli are found in large numbers in nasal mucous membranes, and may be seen in the liver, spleen, lymph nodes, testes, eyes, smooth muscle, and blood vessel walls (Figure 11).
Figure 11 The histological spectrum of HD based on the Ridley-Jopling classification system.


C. Other Types of HD

Other types of HD are sufficiently distinctive to merit a separate description although, actually, they are varieties of the established groups of HD.

1. Pure neural HD

In this type of HD, one or more nerve trunks are damaged and/or enlarged, but no skin lesions are visible. Nerve biopsies show that these patients usually belong to the
tuberculoid end of the spectrum although the entire range of the HD spectrum has been
described in neural HD. This type of HD is more common in India than in the U.S.

2. Indeterminate (I)

There is also an indeterminate form of HD. This is the very earliest stage of the disease in
which histological features and Polymerase Chain Reaction (PCR) testing are
insufficient to make a definitive classification. Clinically, the lesions consist of one or two
vague hypopigmented macules, which may be slightly dry in texture, sweat less readily
than usual, and have minimally impaired sensation. It is difficult to find acid fast bacilli in
the lesion. If it progresses beyond the indeterminate stage, it develops into one of the
established forms of HD.

3. Diffuse Lepromatous Leprosy (DLL)

DLL might be associated with infection with *Mycobacterium lepromatosis*. At times,
these patients may be simultaneously infected with *M. leprae*. Patients will have a
diffuse infiltration of the skin over most of the body, with a smooth shiny appearance
and extensive loss of body hair, including eyebrows and eyelashes. Some of these
patients may develop a severe type of reaction known as Lucio’s phenomenon, which
involves diffuse ulcerations of the skin.

Experience at NHDP has shown that patients with confirmed infection of *M.
lepromatosis* by PCR studies can exhibit an aggressive form of HD. Such patients develop
multiple skin ulcers and necrotic skin lesions, which can be a challenge to treat.
Intensive wound management and aggressive treatment of reactions when they
develop appear to be necessary for the successful treatment of such patients. These
patients often need inpatient specialized care.

V. NERVE INVOLVEMENT IN HANSEN'S DISEASE

A. Select Nerve Damage

Damage to select cranial and peripheral nerves is the basis of serious HD complications that
can lead to permanent nerve damage and subsequent deformity and disability.

Because *M. leprae* and *M. lepromatosis bacilli* prefer cooler temperatures, the peripheral
nerves susceptible to infection are at sites where the nerves emerge from under the
muscles, surfacing near the skin. When the bacteria enters the nerve, inflammation and
swelling occur, which can lead to permanent damage including sensory loss, muscle
paralysis and sweat gland dysfunction. The vulnerable sites and resulting deformities are
shown in Figure 12.

*M. leprae* and *M.lepromatosis bacilli* can infiltrate the facial and trigeminal cranial nerves.
Damage to the facial nerve can cause muscle paralysis leading to lagophthalmos, while
damage to the trigeminal nerve may result in loss of corneal sensation. If untreated, this
nerve damage can lead to blindness.
Some patients have no signs of reaction or nerve pain, but continue to have progressive sensory or motor loss in hands or feet. Such patients need to be assessed to determine that they are receiving appropriate chemotherapy since noncompliance with medication intake could contribute to this problem. However, some patients take their medications regularly and still have deteriorating nerve function. This idiopathic deterioration may improve in some patients if they receive steroid treatment. If the loss of function has been six months or less, they should be given a course of steroids in the dose range for reactions. If the loss has been present for longer than six months the chance of recovery is diminished. Therefore, it is important to regularly monitor all patients under treatment for any changes in nerve function and treat accordingly.

VI. DIAGNOSIS and DIFFERENTIAL DIAGNOSIS

A. Diagnosis of Hansen’s Disease

1. Cardinal Signs

Identifying these cardinal signs of leprosy can be useful in making a clinical diagnosis.

a. Skin Lesions with Anesthesia

Complete or partial anesthesia occurs almost exclusively in HD skin lesions.
(Note: Pure neural leprosy will not have skin lesions.)

b. Nerve enlargement

Enlarged and/or tender peripheral nerves are palpated at specific anatomical sites shown in Figures 15-21.

2. Presence of Acid-Fast Bacilli

The only laboratory study that can definitively diagnose HD is a skin biopsy. There are no serologic or microbiological tests able to diagnose HD. *M. leprae* and *M. lepromatosis* are non-cultivable.

Polymerase chain reaction (PCR) studies are helpful in some difficult diagnostic cases. PCR studies for *M. leprae* and M. lepromatosis are available commercially and are performed in some research laboratories. Skin biopsies can be processed and interpreted and PCR studies performed by the dermatopathologist at the NHDP in Baton Rouge, LA. (See Appendix A and B)

B. Differential Diagnosis of the Skin Lesions

HD skin lesions can mimic other skin diseases such as those listed below, but HD lesions, almost exclusively, have the cardinal sign of impaired sensation.
Table 2. Mimickers of Leprosy Subtypes

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Type of HD</th>
<th>Differential Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypopigmented patches</td>
<td>Indeterminate and Tuberculoid</td>
<td>Vitiligo, Pityriasis Alba, Post-inflammatory hypopigmentation</td>
</tr>
<tr>
<td>Erythematous macules/patches/plaques</td>
<td>Solitary/few for Tuberculoid or more for Borderline and Lepromatous</td>
<td>Psoriasis, Tinea Corporis, Nummular dermatitis, Syphilis, Mycosis Fungoides, Sarcoidosis</td>
</tr>
<tr>
<td>Annular Plaques</td>
<td>Mid-Borderline</td>
<td>Granuloma Annulare, Tinea Corporis, Psoriasis</td>
</tr>
<tr>
<td>Nodules</td>
<td>Borderline Lepromatous, and Lepromatous</td>
<td>Keloids, Dermatofibromas, Lymphoma, Metastases, Sarcoidosis, Other Mycobacterium, Deep Fungal Infections</td>
</tr>
</tbody>
</table>

Table 3. Mimickers of Leprosy Reactions +/- ulceration

<table>
<thead>
<tr>
<th>Reaction Type</th>
<th>Mimicker</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reversal Reaction (RR - Type I)</td>
<td>Cellulitis, Drug eruption, Lymphoma, Tumid Lupus, Sweets Syndrome</td>
</tr>
<tr>
<td>Erythema Nodosum Leprosum (ENL- Type II)</td>
<td>Erythema Nodosum, Sepsis, Panniculitides, Systemic Connective Tissue Disease flare</td>
</tr>
</tbody>
</table>

C. Mimickers of Leprous Neuritis

Among the neurological conditions that may be confused with HD are:

1. Heritable neuropathies
2. Polyneuropathy
3. Entrapment neuropathy
4. Cervicobrachial and scalenus syndromes
5. Syringomyelia
6. Amyloidosis
7. Neurofibroma

D. Important points to remember include:

1. HD never causes upper motor neuron lesions, and proximal muscles are not involved.
2. Sensory loss in HD is maximized peripherally. There may be islands of preserved sensation on the hands or feet.
3. The tendon reflexes are preserved and position sense is usually intact.
4. Hansen’s disease never involves the central nervous system.

http://www.internationaltextbookofleprosy.org/chapter/differential-diagnosis-leprosy

See the website cited above for a more thorough discussion of the differential diagnosis of HD.

VII. PSYCHOSOCIAL CONSIDERATIONS

The healthcare provider who first informs a patient about a diagnosis of HD sets the tone, which will convey either hope or fear. Fear increases the patient’s anxiety and perpetuates the stigma and misunderstandings about HD. Even if the medical provider does not have experience in treating HD, he/she should convey that medical resources are available for this disease and that the patient can be treated and cured. Acceptance of the patient by all clinical staff is vital since it helps the patient feel confident that HD is like other treatable diseases.

By addressing the issues associated with stigma early during treatment, medical providers can decrease the negative effect on the patient. Moreover, by continuing to address the issue throughout treatment, the provider decreases the likelihood that patients will define themselves as leprosy patients for the rest of their lives.

The way in which patients view their lives and conceptualize the disease are decisive factors for their future mental health. Cultural background should also be considered. Various cultures have differing belief systems that can affect stigma, treatment and compliance. Adherence to the National Standards for Culturally and Linguistically Appropriate Services (CLAS) is vital when caring for patients.

People diagnosed with HD find that stigma can manifest itself in prejudice, discrimination and emotional distress. Educational efforts begin with the patient and medical provider and extend to the family and community. The goal of all interventions associated with HD is that the patient is able to maintain or reclaim physical, mental and social well-being.
A. Stigma/myths
The stigma surrounding HD can be more damaging and enduring than the effect of the infection. Though HD is a treatable bacterial infection, the magnitude of the stigma can affect the patient’s physical status, mental health and interpersonal relationships. Demystifying HD is an essential intervention to help the patient cope with HD and its stigma. In the early stages of educating patients about the medical aspects of HD, it is important to emphasize that it is treatable and curable.

B. Living with the diagnosis
Personal concerns arise for all patients as they interact with the people in their lives:

- Whom to tell?
- What to tell?
- When to tell others?

Signs of deformity, skin rashes, or reaction may force patients to deal with these questions sooner than they had hoped. Medical providers can help a patient decide what works best in his/her particular situation. Information should be given to patients about treatment resources for HD and support resources for coping with the disease, treatment and stigma.

C. Common concerns and questions:
Initial reactions to a diagnosis of HD include:

1. Disbelief – unaware that leprosy still exists
2. Confusion – wondering how and when they caught HD and if it is treatable
3. Fear of contagion – concern about “spreading HD” to family and friends
4. Fear of rejection and social isolation – often associated with images from the Bible, movies, etc. wondering if their appearance will change, if deformities are inevitable
5. Feelings of shame – associated with religious/cultural connotations of punishment, being cursed
6. Feelings of depression or despair – thinking their life as they knew it, is over

Family members and others significant in the patient’s life are likely to have the same reactions, fears and misconceptions. Providing the same education and support to them (with the patient’s permission) helps not only the family and significant others, but also the patient.
D. Psychiatric disorders

This disease carries a number of psychosocial issues, which lead to a higher prevalence of psychiatric disorders among patients with HD than in the general population. Depression is the most common disorder found in HD patients. Early detection and treatment of psychiatric disorders is a powerful psychotherapeutic measure. Integrated behavioral healthcare improves outcomes.

VIII. CLINICAL EVALUATION

A. Patient Interview

1. Do you have the following symptoms?
   a. No pain with injuries such as cuts or burns
   b. Wounds or ulcers that will not heal
   c. Skin rash that has not responded to conventional treatment
   d. Recurrent nosebleeds
   e. Chronic nasal congestion
   f. Burning sensation or loss of sensation on the hands or feet
   g. Painful or tender peripheral nerves
   h. Eye problems (red sclera, poor blink reflex, corneal ulcers, lagophthalmos)
   i. Hair loss in areas of the body, especially the eyebrows and eyelashes
   j. Fever, joint pain, malaise
   k. Enlarged lymph glands
   l. Testicular pain or enlargement
   m. Eye pain with or without redness

2. When did the symptoms first appear and how have they progressed over time?

3. Where have you already sought treatment?

4. Were you diagnosed with HD on his first medical visit or were you initially diagnosed with a different disease?

5. What treatments have already been tried?

6. Do you have a family history of HD?

7. What is your travel history?

B. Examination of the Patient

Every patient with suspected HD should have a careful examination directed to the skin and its appendages (hair and sweat/oil gland activity), eyes, nose, ears, neck, extremities, chest,
abdomen, back, testes, and lymph nodes. The main goal of the examination is to confirm or exclude the cardinal signs listed above (Section VI-A).

4. **Skin:**

   It is important to perform a complete examination of the skin in good light. Hypopigmented or hyper-pigmented flat or raised lesions may be found on the face, ears, trunk, extremities, buttocks, or thighs. Absence of sweating, hair loss, or changes in texture of the skin may also be present.

   Any suspected skin lesion should be examined for light touch, preferably with a sensory monofilament, but a cotton wisp or similar item, which can detect light touch, may suffice for an initial screen. Skin lesions may manifest as macules, papules and/or nodules. There may be skin ulcers, tender red nodules, and edema of the face, ears, and extremities. Anasarca may occur in type I (reversal reaction) but especially occurs in type II (ENL) reactions. Also, examine for loss of hair (alopecia), especially madarosis (loss of eyebrows and eyelashes), dry, cracked, and fissured skin, and dry scaly eruption on the legs.

5. **Eyes: Eye Screen Form (Appendix F)**

   Other than the skin and nerves, the eyes are the most frequently affected organs in Hansen’s disease. HD can affect the eyes by direct invasion or nerve damage.

   a. **Direct Invasion**

   Direct invasion of the anterior part of the eye by *M. leprae* and *M. lepromatosis* causes subsequent inflammation and can result in episcleritis, scleritis and/or iridocyclitis. Long-term oral steroid therapy, used to treat these conditions may cause cataracts and glaucoma.

   b. **Nerve Damage**

   1. **Facial Nerve (7th Cranial Nerve):**

   The Facial Nerve innervates the orbicularis oculi muscles surrounding the eye and is not palpable. Paralysis causes lagophthalmos and subsequent corneal exposure resulting in high risk for injury and blindness. Damage to the seventh cranial nerve can also cause facial deformities due to muscle paresis or paralysis, i.e. seventh cranial nerve palsy.
2. **Trigeminal Nerve** (5th Cranial Nerve):

The trigeminal nerve is not palpable. The sensory distribution includes the cornea of the eye. Damage results in loss of corneal sensation and the patient may lose the eye. This raises risk of eye injury and blindness.

All HD patients should have at least a recording of visual acuity, a simple evaluation for lid closure and evaluation for any evidence of redness or eye pain. These simple examination procedures can prevent further ocular morbidity and blindness. An ophthalmologist should examine patients with any evidence of an eye problem that includes vision loss, lagophthalmos (incomplete eye closure), red eye and cataract. Surgical procedures of the eye, such as tarsorrhaphy temporalis muscle transfer; laser iridectomy, and trabeculoplasty for cases of glaucoma and cataract may be indicated.

3. **Nose**

Look for signs of erosive rhinitis, nasal depression (“saddle nose” deformity), and nasal septum perforation. Inquire about the occurrence of epistaxis. (Figure 14)

4. **Neck: Great Auricular Nerve:**

Located posterior to and over the sternocleidomastoid muscle. It may be enlarged but there is no significant consequence for motor or sensory damage. (Figure 15)
5. **Extremities:**

A. **Upper Extremity**– (Hand Screen Form - Appendix G)

The NHDP Hand Screen is specifically designed to identify issues representative of nerve damage from HD. The hand screen is required at diagnosis for a baseline status, and on a routine basis to monitor the nerve status of the upper extremity for early changes. Evaluation includes sensory testing, muscle testing, skin integrity, nerve palpation, and identification of deformity. Inflamed nerves are at risk of damage causing secondary problems including sensory and motor deficits. The nerves commonly affected are located at sites in the upper extremities where they are superficial and easy to palpate. Palpation of the upper extremity nerves affected by HD is shown in Figures 16-18.

1. **Ulnar Nerve**
   - **Located** proximal to the cubital tunnel of the elbow
   - **Motor function**- intrinsic muscles of the hand-paralysis includes clawing of the 4th and 5th fingers and a weak pinch.
   - **Sensory distribution**- medial palm of the hand and 4th-5th digits

![Figure 16. Ulner Nerve](image1)

2. **Median Nerve**
   - **Located** proximal to the wrist at the carpal tunnel
   - **Motor function**- intrinsic muscles of the hand-paralysis can cause loss of opposition of the thumb and clawing of the 2nd and 3rd fingers of the hand
   - **Sensory distribution**- lateral palm of the hand, thumb and 2nd – 4th digits.

![Figure 17. Median Nerve](image2)

3. **Radial Nerve**
   - **Located** proximal to the anatomical snuffbox on the dorsum of the wrist
   - **Motor function**- wrist and finger extensors - paralysis can cause wrist drop
   - **Sensory distribution**- the dorsum of the hand.

![Figure 18. Radial Cutaneous Nerve](image3)
B. **Lower Extremity** (Foot Screen Form - Appendix I)

The NHDP foot screen is designed to specifically identify issues representative of nerve damage from HD. The foot screen is required at diagnosis for a baseline status, and on a routine basis to monitor the nerve status of the lower extremity for early changes. Evaluation includes sensory testing, muscle testing, skin integrity, nerve palpation, and identification of deformity. Nerves of the lower extremity affected by HD include (Figures 19-21):

1. **Peroneal Nerve** –
   - **Located** posterior/distal to the fibular head at lateral knee
   - **Motor function** – anterior tibialis- dorsiflexion of the foot
   - paralysis can cause drop foot
   - **Sensory distribution** – dorsum of the foot

![Figure 19. Peroneal Nerve](image)

2. **Posterior Tibial Nerve**
   - **Located** at the tarsal tunnel around the medial malleoli
   - **Motor function** – Intrinsic muscles of the foot – paralysis can cause claw toes
   - **Sensory distribution** – plantar foot

![Figure 20. Posterior Tibial Nerve](image)

3. **Sural Nerve**
   - **Located** posterior and proximal to lateral malleoli
   - **Motor function** – no motor component
   - **Sensory distribution** – lateral foot

![Figure 21. Sural Nerve](image)

6. **Joints and Tendons**

   Arthritis and tenosynovitis can occur in non-reactive and reactive disease of borderline lepromatous (BL) and lepromatous leprosy (LL).

7. **Breasts**

   In males, gynecomastia can occur in non-reactive disease and mastitis in females with ENL.

8. **Testicles**

   Testicles can be involved in non-reactive disease and as orchitis in reactive disease (ENL.) Ask male patients about pain or swelling of the testicles and examine for erythematous nodules.
9. **Lymph nodes**

Tender lymphadenitis may be seen with ENL reaction.

C. **Laboratory Studies**

1. **Skin Biopsy** (Appendix A)

   Skin biopsy is the only lab test that can diagnose Hansen’s disease.

2. **PCR Assay** (Polymerase Chain Reaction) (Appendix B)

   At NHDP, PCR assay is performed routinely on all samples.

3. **Slit Skin Smears** (Appendix C)

   Results of Fite stained skin smears gives the clinician an assessment of the range and severity of the patient’s infection. It can also be used to assess response to treatment or the degree of bacterial load affecting the severity of the patient’s Type 1 or Type 2 reaction. For reliable results, slit skin smears need to be performed by a properly trained laboratory technician.

D. **Special Considerations**

1. **HD and Pregnancy:**

   A female with HD who is pregnant is uncommon in the U.S., but a few cases occur each year. The majority of these pregnancies are uneventful as far as HD is concerned, but there are a number of potential problems and risks that should be considered when advising female HD patients of childbearing age, and when managing pregnant patients who have HD.

   All female patients of childbearing age should be advised to avoid pregnancy during early stages of the disease, at least until MDT has been completed and preferably, until the disease is completely inactive. The postponement of pregnancy is especially important for patients who have evidence of reaction or neuritis since these problems will be exacerbated during pregnancy and the postpartum period. Additionally, the drugs needed to treat reactions can be teratogenic.

   There are alterations in the immune response during all pregnancies, causing a depression of the cell-mediated immune (CMI) system. This immune suppression during pregnancy and its recovery in the postpartum period appears to play a role in the clinical manifestations of HD in women. It is common for the first symptom of HD in young women to occur during pregnancy or in the postpartum period.

   ENL is more common during pregnancy when the CMI is depressed, while reversal reaction is more common during the postpartum period when the CMI is recovering. The risk of reactions or neuritis during pregnancy will vary considerably with the type of disease and the amount of treatment a patient has received prior to the pregnancy. If a reaction occurs during a pregnancy, it should be managed as in non-pregnant patients.
with the use of prednisone sufficient to control the reaction and prevent nerve damage. Use of Thalidomide is contraindicated and cannot be used under any circumstances.

For patients who are or become pregnant during the early stages of the disease, chemotherapy should generally continue during pregnancy with some modification of the treatment regimen. Dapsone can be continued throughout the pregnancy but avoid the use of rifampin during pregnancy if possible. Clofazimine can be safely given but it can cause hyperpigmentation in the mother and the child.

Patients who have had HD sometime in the past, who have been adequately treated and cured, and whose disease is now completely inactive, can expect to have essentially normal pregnancies. There is no risk of the mother transmitting the disease to infants in such cases.

2. HD and Children:

HD in children is uncommon in the U.S., but when it does occur, it is usually the indeterminate or tuberculoid type disease. It is usually a benign disease with very few deformities reported. Management of the disease is generally the same as for adults except for the adjustment of drug dosages based on the patient's weight to be determined by the physician. Transmission of HD to children or adults should not occur after the patient starts on treatment. Preventive treatment is not generally recommended for child contacts. The presence of new cases in children usually indicates that HD is still being transmitted in the general population.

3. Testicular HD:

Direct invasion of the testicles probably occurs in most cases of Borderline Lepromatous and Lepromatous disease. The testicles are a cool part of the body and are preferentially affected. If HD is not treated early, there is progressive destruction of testicular tissue and eventually testicular atrophy with sterility and a decrease in testosterone production. Acute orchitis may develop during ENL and may be an indication for prednisone therapy. Testicular atrophy is usually permanent. After testicular function is destroyed, the only treatment is testosterone replacement. This does not restore fertility but is helpful in restoring sexual potency. Injectables are the preferred route for replacement therapy. Oral androgens are not recommended for long-term therapy because of potential liver toxicity.

E. HD Surveillance Form (Appendix D)

The HD Surveillance Form is the document used to report leprosy cases to the US NHDP registry. The surveillance form is only required one time, when a patient has been newly diagnosed.

F. Follow-Up Visit

1. Patient Interview
2. Examination of the patient
3. Laboratory monitoring (See Lab Monitoring schedule – Table 5)
4. Annual biopsy is recommended
5. Skin Smears annually (optional)
6. Eye, Hand and Foot Screens (See Frequency of Performance in Section XVI)
7. Patient Education every clinic visit

IX. TREATMENT OF HANSEN’S DISEASE

While the care of an individual diagnosed with HD involves considerably more than prescribing medication, the appropriate drug combination is the most important step toward curing the infection. Health care providers should emphasize to their patient the importance of taking all medications for the duration of treatment. This section will provide an overview of the medications used to treat HD, potential side effects, and alternative medications that may be used if complications arise. Read package inserts for complete information regarding each medication and contact the NHDP for questions not answered in the material below.

A. Chemotherapy: The Anti-Leprosy Drugs

1. Dapsone

Dapsone is bacteriostatic and therefore, must be used in combination with other antibiotics. Dapsone monotherapy may cause drug resistance. Dapsone is available in 25 and 100 mg white tablets. The normal dose is 50 mg or 100 mg daily with or without food.

Side Effects: A mild hemolytic anemia is common. Patients who have a glucose-6-phosphate dehydrogenase deficiency cannot take dapsone and will need an alternate drug. Rare cases of agranulocytosis have been reported.

Dapsone allergy is rare, but if it occurs, it could be mistaken as an HD reaction. Severe cases may develop dermatitis, fever, hepatitis, or even Stevens-Johnson syndrome.

2. Rifampin

Rifampin is available as a 150 mg and 300 mg reddish capsule. It is the most effective bactericidal drug given as a 600mg daily dose. Rifampin is absorbed best when taken on an empty stomach, 1 hour before eating or 2 hours after eating.

Side Effects: Elevated liver function tests (especially ALT, AST). Rifampin should be discontinued if the ALT or AST rises to more than two and one half times normal. Rifampin enhances the toxicity of alcohol. Occasionally patients will develop thrombocytopenia and therefore platelet counts should be monitored.

Patients must be advised that urine and other bodily fluids will turn a reddish color.

Drug Interactions: There are a number of drug interactions with rifampin including steroids, oral contraceptives, warfarin, among many others.
3. **Clofazimine**

Clofazimine is a lipophilic dye that has antibacterial and anti-inflammatory properties. It is available as a brown 50 mg gelatin capsule. The serum half-life of the drug is about 10 days and the tissue half-life may be up to 70 days. The recommended antibacterial dose is 50 mg daily with fatty food.

**Side Effects:** Clofazimine gives the skin a red to blue/black discoloration, more marked in HD skin lesions than in unaffected skin. Sun exposure intensifies the discoloration. The color takes several weeks to develop and may take 2 years or longer to disappear after the drug is discontinued. **NOTE:** Some patients are so distressed by the discoloration that they discontinue the medication without informing the physician. It is important, therefore, to alert the patient in advance that this is a possible side effect. If the patient can agree to take it, and minimize the effect by avoiding the sun, the benefit of Clofazimine will far outweigh the temporary issue of discoloration. Other side effects include dryness and itching of the skin and eyes.

**Special acquisition requirements:**

Clofazimine, used for decades to treat HD around the world, is no longer available in the US commercially. The only way to obtain the drug in the U.S. is by completing an investigational new drug (IND) application through the Food Drug Administration (FDA). The NHDP holds the IND #67.033 for its use in treating HD in the U.S. To obtain the drug for treating HD patients, physicians have to register as an investigator under the NHDP IND. This requires submitting a signed FDA form 1572 and curriculum vitae to the NHDP. A packet of information, including form 1572 and consent forms, etc., will be provided. An Institutional Review Board (IRB) of the Centers for Disease Control (CDC) acts as the central IRB for the use of Clofazimine for Hansen’s disease, so that individual physicians do not need to arrange these themselves. For further information regarding receiving Clofazimine for treatment of HD, or enrolling as an investigator under the IRB held by CDC, please, contact:

National Hansen's Disease Programs  
9181 Interline Avenue Baton Rouge, LA 70809  
Email: HRSANHDPCLINIC@hrsa.gov  
Phone: 1-800-642-2477  
Fax: (225) 756-3819

B. **Protocols**

Multi drug therapy (MDT) has been used by the NHDP to treat patients since the 1970’s. WHO introduced MDT globally in 1982 with a slightly different protocol than NHDP recommends. The U.S. protocols involve a longer duration of treatment with standard guidelines as follows in Table 4.
Table 4

<table>
<thead>
<tr>
<th>MULTIDRUG THERAPY REGIMENS for Newly Diagnosed Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dapsone</td>
</tr>
<tr>
<td>Paucibacillary (TT, BT, I)</td>
</tr>
<tr>
<td>Option 1</td>
</tr>
<tr>
<td>Option 2</td>
</tr>
<tr>
<td>Multibacillary (LL, BL, BB)</td>
</tr>
<tr>
<td>Option 1</td>
</tr>
<tr>
<td>Option 2</td>
</tr>
</tbody>
</table>

In general, NHDP recommends Option 1 protocols that include daily rifampin. Option 2 is used for patients on prednisone. For immunologically compromised or elderly patients, these protocols may be modified. Consultation with the NHDP is advised (800-642-2477).

The recommended duration of treatment is sufficient to kill leprosy bacilli, even though large numbers of dead organisms may remain in the tissues for several years before they are physiologically eliminated. There is no evidence that additional or prolonged treatment hastens the elimination of these dead organisms.

C. Alternative Anti-Microbial Agents

The main indication for the use of alternative anti-microbial agents is intolerance for the recommended drugs or drug-drug interaction.

1. **Minocycline**, 100 mg daily, can be used as a substitute for dapsone in individuals who do not tolerate it. 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. Minocycline causes photosensitivity and sunscreen is highly recommended during use. Minocycline is not to be used during pregnancy.

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

3. **Levofloxacin**, 500 mg monthly, may be used in place of Rifampin, for adults, but is not recommended for children.
D. Special Considerations

1. Treatment for Pregnant Women:

While pregnancy during HD treatment is strongly discouraged, in the event that it occurs, modifications to the medication regime is described in Table 5.

Table 5

<table>
<thead>
<tr>
<th>Pregnant Women Diagnosed with Hansen’s Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dapsone</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Paucibacillary</td>
</tr>
<tr>
<td>Multibacillary</td>
</tr>
</tbody>
</table>

* Rifampin is taken monthly if the patient is on prednisone.

2. Treatment for Children:

In the United States, the occurrence of leprosy in children is fairly low. The general guidelines for HD treatment for children is found in Table 4, however, it is strongly recommended to contact the NHDP for management of leprosy in children.

Table 6

<table>
<thead>
<tr>
<th>CHILDREN - MULTIDRUG THERAPY REGIMENS for Newly Diagnosed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dapsone</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Paucibacillary (TT, I, BT)</td>
</tr>
<tr>
<td>Multibacillary (LL, BL, BB)</td>
</tr>
</tbody>
</table>

*Rifampin is taken monthly if the patient is on prednisone.

3. Testicular HD

If testicular function is destroyed by HD, the only treatment is testosterone replacement. This does not restore fertility but is helpful in restoring sexual potency and general well-being. Injectables are the preferred route for testosterone replacement therapy.


E. Laboratory Monitoring

Table 7 Laboratory Monitoring for Drugs Used to Treat Hansen's Disease

<table>
<thead>
<tr>
<th>Drug</th>
<th>Laboratory Studies</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial studies for all drugs</td>
<td>CBC w/platelets, UA, CMP, G6PD, CRP, Sed rate</td>
<td>Baseline</td>
</tr>
<tr>
<td>Dapsone</td>
<td>CBC</td>
<td>Every 3 months</td>
</tr>
<tr>
<td>Rifampin</td>
<td>CBC w/platelets, CMP</td>
<td>Every 3 months</td>
</tr>
<tr>
<td>Clofazimine</td>
<td>No recommended laboratory studies</td>
<td></td>
</tr>
<tr>
<td>Thalidomide *</td>
<td>CBC with differential</td>
<td>Every 3 months</td>
</tr>
<tr>
<td>Methotrexate *</td>
<td>CBC, CMP</td>
<td>Every 3 months</td>
</tr>
</tbody>
</table>

*Drugs used in managing reactions (see below Section X -Immunological Reactions)

F. PROGNOSIS

Patient isolation is unnecessary since HD is difficult to transmit to another person before treatment and impossible to transmit after 72 hours on treatment. The majority of patients are able to continue normal activities and occupations with little interruption in their daily routines during treatment.

Success of Hansen’s disease treatment is measured by completion of prescribed medication and by absence of deformity and disability due to nerve damage. Most patients in the U.S. have little or no disability at the time of diagnosis. If recent nerve damage or neuritis is present when diagnosed, prompt treatment may improve the patient’s nerve status or at least prevent further damage to the nerves. While the prescribed multidrug regimen minimizes the possibility of nerve damage, 30-40% of patients have recurrent episodes of reaction during treatment with possible nerve damage. Unfortunately, nerves damaged for years prior to diagnosis will not recover, and disabilities are permanent.

G. Follow-Up after Completion of Treatment

Clinical examinations are performed at the following intervals after MDT is complete:

1. Paucibacillary (PB) - Annually up to two years
2. Multibacillary (MB) - Annually up to three years

Annual follow-up forms are completed at each yearly follow-up visit.

Skin biopsies are performed at the doctor’s discretion.

X. IMMUNOLOGICAL REACTIONS

Reports indicate that up to 50% of all HD patients will experience an acute hypersensitivity or immunological reaction to the *M. leprae* organism during the course of their disease. Table 6 identifies the two types of reactions and how they correspond with the
WHO/Ridley-Jopling classification. Table 7 includes a summary of the differences between reversal (Type I) and ENL (Type II) reactions. Unfortunately, there are no predictors to identify which patients will develop a reaction. Patients who do develop reaction should be monitored closely as they are at a higher risk of nerve damage and subsequent disabilities and deformities.

Table 8

<table>
<thead>
<tr>
<th>WHO and Ridley-Jopling classifications with corresponding Reaction types</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paucibacillary</td>
</tr>
<tr>
<td>TT</td>
</tr>
<tr>
<td>No Reaction</td>
</tr>
</tbody>
</table>

A. Reversal Reaction (RR or Type 1 Reaction)

Reversal reaction is recognized by the development of edema and erythema in existing skin lesions. Severe cases are characterized by fever, edema of the hands and feet, and erythema of pre-existing skin lesions, which occasionally ulcerate. Reversal reactions can also involve the nerves. Edema and granuloma formation may lead to sudden nerve damage, with swelling and pain in one or more of the commonly affected nerve trunks.

While reversal reaction generally occurs during the first 6-12 months of treatment, it may occur later, even after drug treatment is complete. Reversal reaction is also seen in untreated HD, and sometimes its onset is the event that makes the patient decide to seek medical attention.

If a patient remains untreated, the reaction might subside after a few weeks. However, a Type I reaction can also persist, with the lesions becoming more extensive.

Reversal reaction is an acute exacerbation of the normal cell mediated immune response to *M. leprae*. It is one of the most clear-cut examples of delayed-type hypersensitivity causing clinical disease. While the precipitating event is unclear, it seems likely that the bacilli residing in Schwann cells are for some reason, suddenly "recognized" by the cell-mediated immune system. As a result, the patient's peripheral and dermal nerves are damaged or destroyed along with the bacilli.

Pathology reports may show edema, epithelioid granuloma formation, and an influx of lymphocytes, but this is not diagnostic of Type I reaction.

B. Erythema Nodosum Leprosum (ENL or Type 2 Reaction)

ENL typically appears as transient red nodules in the skin, which are often tender and painful. The nodules can appear on almost any part of the body, including the face, and will appear on previously normal skin. The nodules resolve in 7-10 days, often leaving a characteristic mottled hyperpigmentation. Patients with ENL often have fever and experience a generalized illness. There may be associated attacks of painful neuritis,
arthritis, lymphadenopathy, uveitis, and orchitis, though it is unusual for all of these to be present at the same time. Edema of the extremities is common.

In severe cases of ENL, the patient may have hepatosplenomegaly, high fever and/or a clinical presentation that resembles Systemic Inflammatory Response Syndrome (SIRS). Skin nodules are usually present.

ENL may be episodic with attacks occurring every month or two. In some cases, these episodic attacks come so frequently that the reaction is considered continuous. In such cases, it often persists for 2 years or more.

If an ENL reaction remains untreated, new crops of nodules will continue to appear. Edema of the hands and feet is common and neuritis can produce extensive nerve damage.

ENL occurs in patients who have large amounts of circulating anti-mycobacterial antibodies, and is proposed to be a clinical manifestation of antigen-antibody immune complex formation. The immune complexes are usually formed extravascularly in sites where *M. Leprae* are present in high concentration, such as skin, nerves, lymph nodes and testes. These complexes elicit an inflammatory response, which results in the clinical manifestations of ENL. It also has been shown that tumor necrosis factor alpha is elevated during ENL and decreases as the reaction subsides. ENL occurs most commonly in patients who are receiving MDT but can occur before treatment or long after treatment is completed. The antigen in this reaction is from dead *M. leprae*.

ENL reaction is characterized by foci of neutrophil infiltration with fragmented *M. leprae* and associated vasculitis. After a few days, as the lesion is resolving, the neutrophils are replaced by loose collections of lymphocytes.
### Table 9

<table>
<thead>
<tr>
<th>Type of reaction</th>
<th>Type 1 (Reversal Reaction)</th>
<th>Type 2 (ENL)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mechanism</strong></td>
<td>Cell-mediated; delayed hypersensitivity reaction</td>
<td>Antigen antibody (immune complex) reaction</td>
</tr>
</tbody>
</table>
| **Skin**         | **Mild:** - red, mildly swollen skin lesions - no lesions on the face - no edema hands, feet or face  
|                   | Severe: - painful, swollen skin lesions - ulceration/threatening ulceration of skin - swollen lesions on the face - edema of hands, feet, face | **Mild:** - minimal pain; no ulcerating skin lesions  
|                   | **Severe:** - red, painful, bullous or ulcerating skin lesions - common on the face, extensor surfaces of arms and legs. - edema of hands and feet |
| **Constitutional symptoms** | Good.  
|                   | Little or no fever or other constitutional symptoms | Mild: - afebrile/only mild fever  
|                   | **Severe:** - febrile systemic illness |
| **Other Organs/Tissues** | Not affected | May be affected:  
|                   | | Joints- arthritis  
|                   | | Testes- orchitis  
|                   | | Kidneys- proteinuria |
| **Nerves**       | **Mild:** - no painful or tender nerves  
|                   | Severe: - nerves close to skin may be enlarged, tender, painful (neuritis) with loss of nerve function, i.e. diminished sensation, muscle weakness of hands/feet. - rapid onset |
| **Eyes**         | **Mild:** - no eye problems  
|                   | Severe: - weak eyelid muscles leading to incomplete closure (lagophthalmos) due to nerve damage  
|                   | - Internal eye disease (uveitis, scleritis) |

### XI. TREATMENT OF REACTIONS AND NEURITIS

Reactions are a major cause of nerve damage; subsequently the focus of management should be on preventing or minimizing nerve damage. Damage to the nerves is caused by inflammation within the nerve due to intraneural *M. leprae*, which is similar to the process seen in the skin. In untreated HD without reaction, nerve damage is more insidious, while in
reaction, nerves may be damaged within days. Skin reactions and acute neuritis often occur together. See Table 8 for a summary of treatment recommendations for reactions.

A. Treatment of Reversal Reaction (Type 1 Reaction)

1. Mild reaction

Mild reaction is characterized by the presence of edema and erythema of existing skin lesions only. There may be low-grade fever and some general discomfort. If there are any signs of neuritis such as nerve enlargement, pain, tenderness or loss of nerve function, the reaction is no longer considered mild and should be managed as a severe reaction. The treatment of mild reaction is symptomatic with analgesics such as NSAIDS. However, such patients must be observed closely for deterioration of nerve function which requires more aggressive treatment. Antibacterial treatment for HD should be continued at full dosage during a mild reaction.

2. Severe reaction

Severe reaction is characterized by the presence of any of the following:

- Nerve pain and/or nerve function impairment (sensory or motor)
- This is usually but not always associated with nerve tenderness.
- Edema of the hands and/or feet
- Fever and systemic symptoms such as malaise
- Joint Pain
- Swollen and tender skin lesions: facial lesions indicate risk of facial nerve damage
- Ulceration of inflamed skin lesions

a. Prednisone

In severe reactions, an overactive immune response causes tissue damage; therefore, treatment involves immunosuppression with corticosteroids. The initial dose of prednisone ranges between 60-80 mg in a daily single morning dose. This dose is tapered to 40-60 mg within 2 weeks. It is gradually tapered until eliminated over a period of 2-6 months, depending on the severity of the reaction and the response to treatment. Some patients require even higher doses than those mentioned. The main objective is to provide sufficient prednisone to relieve the patient's symptoms and prevent nerve damage.

If at any dosage level the clinical signs of reaction persist or recur, the dose is increased 20-30 mg per day until the symptoms resolve, at which time, slow tapering of the dosage can begin again.

Individualized dosing is tailored by clinical response. Patients with co-morbidities, such as diabetes, may need steroid sparing drugs such as methotrexate (see below).
Rapid improvement of nerve function occurs most often in situations when the lesion is of recent onset (less than six months). On the other hand, regeneration and recovery of function following severe nerve damage will take many months or may not occur at all. Therefore, lack of nerve function improvement is not necessarily an indication to increase the dosage of prednisone or prolong the period of steroid treatment.

b. **Clofazimine**

In select cases, a trial of Clofazimine 200-300 mg daily for 6-12 months may be indicated. Clofazimine is not quick acting in regards to its anti-inflammatory effect. The patient may have to take it for 6 weeks or more for the full effect of this drug to be evident.

c. **Surgery**

On a rare occasion, if a nerve trunk remains markedly enlarged and the patient complains of persistent nerve pain in spite of intensive steroid treatment, consider surgical consultation (See Section XIII on Surgical Interventions).

**B. Treatment of Erythema Nodosum Leprosum (ENL - Type 2 Reaction)**

The management of ENL will vary somewhat depending on whether the reaction is mild or severe and whether it is intermittent or continuous.

1. **Mild reaction:**

When the reaction is mild, the patient develops minor attacks of ENL in the skin, which last for several weeks and then resolve. There may be low-grade fever and malaise, and mild nerve pain or tenderness but without loss of function. Mild reactions can be managed symptomatically with analgesics. A patient may then be free of a reaction for several months.

2. **Severe reaction:**

These patients often present to the Emergency Room and are thought to be septic. One or more of the following are present:

- Fever and malaise
- Elevated white blood cell count
- Painful and ulcerating skin lesions
- Nerve pain and/or nerve tenderness with palpation
- Sudden loss of sensation or muscle weakness in hands, feet, or eyes.
- Edema of the hands and/or feet
- Joint pain
- Orchitis
• Red painful eye (Uveitis, Scleritis)
• Headaches

If the physician is inexperienced in treating this reaction, consider contacting the NHDP 1-800-642-2477.

a. Prednisone 40-80 mg daily is initiated as a single morning dose. After the reaction is controlled, a slow tapering of the dose by 5 mg every 1-2 weeks is started. If the reaction recurs, the dose is adjusted to control the reaction.

Severe reactions, especially those with evidence of nerve damage, may require prolonged treatment with corticosteroids. In the U.S., many patients with ENL have a chronic and continuous reaction and therefore require continuous steroid treatment. If steroids are required for 2-3 months, the reaction can be expected to persist for a year or longer. Steroid sparing drugs should be initiated as soon as possible to avoid steroid dependency and side effects.

During reactions, antibacterial treatment should be continued in full dosage. **The one exception to this is patients on daily rifampin who are requiring prednisone to control reactions**. Rifampin induces liver enzymes which results in fast metabolism of prednisone and decreases its clinical benefit. Therefore, such patients will usually benefit from switching the rifampin from 600mg each day to 600 mg monthly dose.

b. Thalidomide is the drug of choice for ENL. It is a TNF alpha inhibitor. Thalidomide is prescribed based on the severity of the symptoms, but a normal dose is 200 mg taken at bedtime as drowsiness is a side effect. Thalidomide is teratogenic causing severe birth defects if taken by women during pregnancy. Women of child bearing age must use two forms of birth control and take a monthly pregnancy test while on therapy. Thalidomide is tightly regulated by Celgene Corporation Risk Evaluation and Mitigation Strategies (REMS) program.

See website: [https://www.celgeneriskmanagement.com](https://www.celgeneriskmanagement.com)

c. Clofazimine can be given in a divided dose of 200 mg daily for several months, and then reduced to 100 mg daily. The addition of Clofazimine at these doses will usually make it possible to reduce the dose of steroids required, but not eliminate them entirely. Clofazimine is not quick acting and it may take 6 weeks or more for the full effect on the reaction to be noted. Patients receiving larger doses of Clofazimine will have more skin pigmentation changes and possibly more frequent gastrointestinal side effects. When the patient has required no steroids for approximately three months, the dosage of Clofazimine can be reduced to 50 mg daily. If the Clofazimine is not required for antibacterial treatment, it can be discontinued when no steroids have been required for an additional three months.
d. Methotrexate

Prior to starting Methotrexate, or before restarting after a rest period, pretreatment assessments should include:

1. Full blood count including differential blood count and platelets
2. Liver function tests including serum albumin
3. Renal function tests
4. Exclude Hepatitis B and C if clinically indicated
5. Exclude TB if clinically indicated.

Methotrexate dosed weekly is used as a steroid sparing agent. The dose can be titrated upward weekly. The maintenance dose is 15-25 mg once weekly. Patients should be closely monitored so that toxic side effects can be detected as early as possible. Baseline assessment should include CBC with differential and platelet count, hepatic enzymes and renal function tests. These should be repeated every 1 to 2 months. It is recommended that 1 mg folic acid should be taken daily except on the day of the weekly dose. The medication should be stopped if mouth sores or diarrhea develops. Methotrexate is contraindicated in pregnancy.

C. Lucio’s Phenomenon

Patients experiencing Lucio’s Phenomenon are acutely ill. In addition to standard antibacterial treatment, these patients are treated similar to those with ENL using a high dose of Clofazimine, steroids and Thalidomide. Close attention should be given to skin ulcerations which can cover the extremities.

D. Patient Education Regarding Reactions

An important part of management of reactions is providing correct information and listening to the concerns of patients and their families. Patients usually fear that treatment has failed, their disease is getting worse and they will suffer permanent disability and disfigurement. In chronic reactions, especially ENL, patients often become depressed. It should be emphasized that a reaction does not indicate a failure of antibacterial treatment or toxicity to drugs. Reactions are due to the immune system reacting to the dead bacteria. Patients can always be reassured that HD and reactions are treatable conditions and that even long standing reactions will eventually end. In the vast majority of cases, the long-term prognosis is good and there should not be any further progression of nerve damage or disability after the initiation of treatment. Patients must understand that discontinuing medication is NOT a good option.
## Table 10

### Summary: Treatment of Reactions

#### Reversal Reaction (Type 1)

<table>
<thead>
<tr>
<th>Mild Symptoms</th>
<th>Severe Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- erythematous, mildly swollen skin lesions</td>
<td>- painful swollen skin lesions</td>
</tr>
<tr>
<td>- no painful or tender nerves</td>
<td>- ulceration or threatening ulceration of skin</td>
</tr>
<tr>
<td>- no lesions of the face</td>
<td>- swollen lesions of the face</td>
</tr>
<tr>
<td>- no edema of the face, hands or feet</td>
<td>- edema of the hands, feet, or face</td>
</tr>
<tr>
<td></td>
<td>- diminished sensation or muscle weakness in hands/feet</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>- analgesics for 1-2 weeks</td>
<td>- Prednisone - 40-80 mg single daily dose 5-7 days then tapered to elimination in 2-6 months</td>
</tr>
<tr>
<td></td>
<td>(Always use prednisone for neuritis)</td>
</tr>
<tr>
<td></td>
<td>- Clofazimine – in selected cases, trial of 300 mg daily up to 3 months.</td>
</tr>
<tr>
<td></td>
<td>If effective, continue at reduced dose 200 mg for 3 months, followed by</td>
</tr>
<tr>
<td></td>
<td>100 mg for additional 6 months</td>
</tr>
</tbody>
</table>

#### Erythema Nodosum Leprosum (Type 2)

<table>
<thead>
<tr>
<th>Mild Symptoms</th>
<th>Severe Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>- afebrile or only mild fever</td>
<td>- febrile systemic illness</td>
</tr>
<tr>
<td>- minimal pain and no ulcerating skin lesions</td>
<td>- painful or ulcerating skin lesions</td>
</tr>
<tr>
<td>- no painful or tender nerves</td>
<td>- painful or tender nerves</td>
</tr>
<tr>
<td>- no eye problems</td>
<td>- diminished sensation or muscle weakness or hands or feet</td>
</tr>
<tr>
<td></td>
<td>- edema of the hands and/or feet</td>
</tr>
<tr>
<td></td>
<td>- uveitis, scleritis, arthritis, orchitis, proteinuria</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>- analgesics for 1-2 weeks</td>
<td>- Prednisone- 40-80 mg daily tapered to lowest dose required to control the reaction</td>
</tr>
<tr>
<td>May be repeated several times if reaction remains mild</td>
<td>(Always use prednisone for neuritis)</td>
</tr>
<tr>
<td></td>
<td>- Thalidomide 300 mg daily in divided doses,</td>
</tr>
<tr>
<td></td>
<td>tapered to 100-200 mg daily within 2 weeks, then given 50-200 mg daily for</td>
</tr>
<tr>
<td></td>
<td>as long as required to control the reaction</td>
</tr>
<tr>
<td></td>
<td>- Clofazimine- 300 mg daily for 6 weeks, 200 mg daily 2-6 months, and 100 mg</td>
</tr>
<tr>
<td></td>
<td>daily for 1-2 years</td>
</tr>
</tbody>
</table>

During the prescribed time of treatment, all antibiotics should be continued during all types of reactions. Rifampin reduces the effectiveness of all steroids, including prednisone, so it is necessary to decrease the dosing of rifampin from a daily dose to a monthly dose to obtain an optimal therapeutic response to prednisone. The dose of prednisone may need to be increased on the day that rifampin is taken.

A practical guide for the dose of prednisone in neuritis is that the initial doses should be large enough to relieve the pain and tenderness in the nerves in 24-48 hours. The maintenance dose should be large enough to prevent recurrence of the nerve pain. An exception to this would be patients who have had long-standing neuritis with persistent pain probably due to scarring in and around nerves, but whose nerve function status has been stable for a long period. Prednisone is not usually beneficial in such patients.
XII. PREVENTION OF DISABILITY (POD)

Images of deformity and disability still prompt the fear and stigma associated with leprosy (HD). Early diagnosis and treatment, before nerves are damaged, is the best prevention of the deformity and the stigma. If significant nerve damage has occurred, a long-term management program using a team approach is needed. Routine care for insensitive eyes, hands and feet is crucial to minimize or even prevent deformity. The POD team will include some or all of the following professions: physician, nurse, occupational therapist, physical therapist, ophthalmologist, pedorthist, podiatrist, orthotist, social worker, surgeon and pharmacist. Each team member brings specific expertise that collectively provides comprehensive intervention to prevent or minimize deformity. The NHDP developed a five step Prevention of Disability Program that outlines the components of essential preventative care. The following are the five components of the NHDP POD Program.

1. **Eye, Hand and Foot Screening** (Appendix F, G, H)
   Baseline and routine evaluation of the eyes, hands and feet is foundational for objectively measuring the patient’s nerve status. The goal of screening is to identify sensory loss, muscle weakness/paralysis, anatomical deformity, enlarged/tender nerves and autonomic dysfunction. During treatment with MDT, screens are performed quarterly to identify changes in peripheral nerve function. During reaction, screens may be performed as often as monthly to detect early nerve changes for immediate treatment. Based on results of the eye, hand and foot screens, referral to appropriate medical and therapeutic interventions can prevent or minimize nerve damage and subsequent deformity.

2. **Patient Education** (See Appendix I- Healthy Eyes, Hands, and Feet for a Lifetime)
   Patients need to be educated on the autonomic, sensory and motor issues associated with peripheral nerve damage due to HD. They need to understand how to protect inflamed nerves, hydrate dry eyes and skin, protect insensate areas and know who to contact at the first sign of relevant problems. It is critical for patients to understand the concept of “loss of protective sensation” (LOPS). Without protective sensation, the patient can sustain injuries, burns or ulcers from trauma or repetitive pressure without feeling any pain. Patients with LOPS need education on methods to compensate for their lack of pain sensation, such as frequent inspection, protective equipment for the eyes and hands, and appropriate footwear.

3. **Daily Self-Inspection**
   Patients with LOPS, anatomical deformities or current/previous history of ulcers are taught to carefully inspect their eyes, hands and feet on a daily basis. They are to seek immediate treatment if they identify areas of redness, swelling, blisters, callus or wounds. Most patients with LOPS need constant reinforcement to develop and maintain effective self-inspection habits to prevent small issues from becoming major problems. While it is ideal for patients to inspect their own eyes and limbs, a family member or friend may need to assist if the patient is physically unable to perform daily inspection.
4. Management of Problems

Patients with enlarged/tender nerves, LOPS, weakness, deformities, thickened nails/callus and wounds require regular follow-up. A nerve affected by HD may become inflamed and swollen causing tingling, pain or weakness in that particular limb. As a result, a patient may be inclined to move the arm or leg in an attempt to relieve the discomfort or to strengthen the weak limb. This additional movement may cause more damage to the nerve. Advising the patient to limit movement is encouraged, but an inflamed or painful nerve may require splinting to immobilize and protect the nerve from harm.

A. Upper Extremities: Limit repeated movements and heavy lifting; do not sleep with elbows and wrists flexed. An elbow pad, arm splint or wrist splint may be required to impede or prevent movement that would harm the inflamed nerve.

Avoid repetitive motions

B. Lower Extremities: An elastic wrap (cautiously wrapped), compression socks, or a rigid walking boot may help to decrease swelling, immobilize the ankle and relieve discomfort in the leg.

Routine care may be required to manage thick nails and callus to prevent wounds. An open wound becomes a risk for subsequent infection. Infections that go unnoticed due to lack of sensation can eventually penetrate to the bone and result in progressive
shortening of the digits. Offloading pressure is paramount to wound healing in patients with loss of sensation and deformities. Offloading is accomplished using adhesive felt, casts, splints and other devices. In addition to off-loading, wound management consists of appropriate dressings, debridement of devitalized tissue and management of osteomyelitis. Patients with clawing or foot drop may benefit from specific exercises, splints and education to prevent contractures and progressive deformities. These patients may also be eligible for reconstructive surgery. To consult NHDP staff regarding non-healing wounds, surgical candidates and difficult cases (800) 642-2477. (See Appendix J-O for nail/callus care, wound management, basics of wound care, and offloading techniques such as adhesive felt and toe pillows.)

5. Assistive Devices/ Footwear and Orthotics
The patient who lacks sensation can sustain injuries or wounds without noticing, even while performing simple daily activities (i.e. pinching a key tightly in an attempt to open a door, or holding a ceramic mug that has become hot from the microwave or the beverage inside). A combination of modified techniques and assistive/protective devices decrease the risk of injury and maximize independence in the performance of daily functional tasks. Some of these protective devices prevent injury by insulating from the elements or extreme temperature. Other devices prevent injury by increasing the surface area, which decreases the pressure on the limb. Some commonly recommended adaptive and safety devices include the following:

A. Eyes:

Contoured eye mask – worn at night protects the eyes from dryness due to Lagophthalmos.

Wraparound sunglasses protect the eyes from dust, wind and exposure to UV light.

Broad brimmed hat protects the eyes from exposure to UV light.

B. Hands: are susceptible to burns and cuts in the kitchen. Adaptive safety equipment is important for those with LOPS to prevent injuries and subsequent deformity.

Long arm oven mitts

Insulated mugs

Tab grabber
C. Feet: Protective footwear cannot be overemphasized. Proper shoes and orthotics, worn as prescribed, can protect the feet from injury and ulceration. Improper footwear, or going barefoot, may lead to serious harm and cause ulceration. An expert, such as a Pedorthist or Podiatrist, must address the footwear and orthotic needs of a patient with LOPS and deformity.

Patient understanding and compliance with prevention protocols is the most important aspect for prevention of injury and disability. For more information on POD, assistive devices, footwear, orthotics and vendors, see NHDP website: [https://www.hrsa.gov/hansens-disease](https://www.hrsa.gov/hansens-disease).

A patient with decreased corneal sensation in the eyes or LOPS in the hands and feet must be monitored indefinitely - even after being released from medical treatment!

XIII. SURGICAL INTERVENTION

For appropriate candidates, surgery may be an option to expedite wound healing, minimize deformity and maximize function. Depending on the patient’s need and condition, the following surgical procedures may be considered:

A. Hands and Feet

1. **Debridement** – Surgical removal of non-viable tissue including infected bone to promote wound healing.
2. **Skin Graft/Flap** – Once a wound is devoid of all non-viable tissue, a skin graft or flap may expedite wound healing.
3. **Tendon Transfer** – A tendon transfer procedure involves moving or transferring the tendon insertion of a healthy muscle to the insertion site of a weak or paralyzed muscle(s) in order to restore balance and function to the limb. This procedure is most commonly used to correct mobile claw hand deformity and less often to rebalance a drop foot.
4. **Nerve Transposition** – This procedure is indicated to decrease pain and prevent entrapment of the ulnar nerve in the cubital tunnel.
5. **Tendon Release** – A tendon release procedure, such as an Achilles tendon lengthening or toe flexor release may help reduce high-pressure areas that translate onto the foot.
6. **Arthrodesis** – An arthrodesis involves fusing a joint to improve the position of the digit. This procedure is typically used for longstanding paralysis where a contracted digit has resulted in joint subluxation.

7. **Amputation** - An amputation is a last resort procedure, but is required for uncontrollable sepsis, an avascular extremity or when function will be increased by prosthetic usage.

8. **Osteotomy** – This procedure involves removal of a rigid and prominent bony deformity in order to decrease high-pressure areas.

**B. Eyes**

1. **Tarsorrhaphy** – This procedure involves the temporary or permanent joining of select portions of the upper and lower eyelids. It is indicated to protect the cornea from exposure due to facial paralysis. Lateral tarsorrhaphy is preferable for cosmetic and functional reasons.

2. **Laser Iridectomy** – This procedure involves creating a hole in the outer edge of the iris, leading to an opening and widening of the angle to allow for improved fluid outflow. It is intended to preserve vision and prevent glaucoma from occurring or progressing.

3. **Trabeculoplasty** – A trabeculoplasty is used for certain cases of glaucoma to lower intraocular pressure.

4. **Cataract surgery** - Cataract removal is a common procedure and often used with intraocular lens implantation. It may be required for HD patients who have been on long-term steroids.

**XIV. CONTACT EVALUATION**

**A. Contact:**

In the U.S., a Hansen's disease “contact” is defined as a person living in the same household with a new patient in the three-year period prior to the beginning of treatment. Examination of contacts of known cases is the simplest and only practical form of active case finding in low incidence areas such as the United States. This examination can be performed by a physician or public health nurse.

The patient always has a right to privacy and may refuse to notify a contact that they have been diagnosed with HD. In that case, treat the patient as anyone exercising the right to privacy.

If a person becomes aware that they are a contact and wishes to be evaluated, the clinic is obligated to screen the patient. If the contact refuses to be screened, it is not mandatory.

**B. Contact Examinations:**

1. Examine the entire skin surface
2. Nerve function assessment of the peripheral nerves, focusing primarily on the eyes, hands, and feet (See Appendix F,G,H).

C. Contact Follow-Up:
Contacts with a negative initial exam do not need follow-up as long as the patient has been educated about the disease and what symptoms should be reported to the health care provider.

D. Chemoprophylaxis:
The NHDP does NOT recommend chemoprophylaxis for contacts.
There are no known intermediate hosts and *M. leprae* does not survive for long periods outside the body. There is no vaccine available and chemoprophylaxis is impractical in most situations. Thus, at present, there is no practical means of primary prevention, (i.e. the detection and protection of persons at risk). Hansen’s disease control is based on secondary prevention; that is, the early detection and regular treatment of all detected cases existing in an area.

XV. AMBULATORY CARE PROGRAM
Individuals living in the United States and its territories (Puerto Rico, US Virgin Islands, Guam, Northern Mariana Islands, and American Samoa) may receive medical care for the diagnosis and treatment of HD related conditions at one of the federally supported outpatient clinics throughout the USA. Contact the NHDP at 1-800-642-2477, weekdays- 8 a.m. to 4:30 p.m. CST, for referral to one of the Regional Clinics.

A. Services include:
- Confirmation of diagnosis through histopathologic examination of skin biopsies
- PCR testing
- Medical care for Hansen’s disease and its complications
- Medications at no cost to the patient
- Consultation on patients with eye, hand and foot problems for specialized treatment
- Professional and patient education materials

B. Locations:
Additional information for each clinic is located on the NHDP Website under the heading “Ambulatory Care Clinics”:


The Hawaii Department of Health operates a clinic independent of the National Hansen’s Disease Programs.
XVI. PRIVATE PHYSICIANS

HD medications can be provided to patients living in an area not served by an Ambulatory Care HD clinic through a private physician.

A physician can request HD medications from the NHDP at no charge to the patient. Consultation and biopsy processing services are also provided by the NHDP free of charge at the physician’s request.

For patients in the United States and its territories, contact the NHDP at 1-800-642-2477, weekdays 8 a.m. to 4:30 p.m. CST for information on private physicians who have some experience in treating Hansen's disease. In Hawaii, HD patients can call 1-808-733-9831.

XVII. REPORTING REQUIREMENTS

A. Surveillance Form: (APPENDIX D)

The NHDP maintains a National HD Registry for all patients diagnosed with Hansen’s disease in the U.S. A completed HD Surveillance Form is required on all patients diagnosed in the US. The Form can be found in APPENDIX D or on the NHDP website under the “Forms” heading:


The completed surveillance form needs to be sent by secure fax (please do not Email) to the NHDP as soon as the diagnosis is confirmed.

Mail or Fax the HD Surveillance Form to:

NHDP
ATTN: Medical Records
9181 Interline Avenue
Baton Rouge, LA 70809
FAX: 225-756-3706

B. Annual Follow-Up Form: (APPENDIX E)

The Annual follow-up form is required to monitor the treatment and disability status for each patient. Mail or Fax (please do not Email) the Annual Follow-Up Form to:

NHDP
ATTN: Medical Records
9181 Interline Avenue
Baton Rouge, LA 70809
FAX: 225-756-3706
C. Eye, Hand and Foot Screen Forms (Ambulatory Care Clinics) – (APPENDIX F, G, H)

The Ambulatory Care clinics are required to perform Eye, Hand and Foot screens at diagnosis, quarterly while the patient is on medical treatment, and annually for the three year observation period after MDT. Copies of the completed Hand and Foot screen forms need to be sent via secure fax or US Mail on a quarterly basis to:

NHDP
ATTN: Ambulatory Care Program
9181 Interline Avenue
Baton Rouge, LA 70809
FAX: 225-756-3706

XVIII. RESOURCES

A. NHDP Resources for HD include:

1. Consultation on the diagnosis, treatment and management of immunological reactions
2. Consultation regarding treatment for HD related complications including neuropathic limb care, wound care, prevention of disability, and orthopedic procedures
3. Social services and stigma counseling
4. Medications for HD at no cost to the patient
5. Histopathologic examination of skin biopsies, evaluation of Fite stained skin smears and molecular testing (Polymerase Chain Reaction - PCR) at no cost to the patient
6. Educational seminars for Physicians, Nurses, Occupational Therapists, Physical Therapists, Orthotists, and Podiatrists
7. Monofilament kits for sensory testing
8. Online courses for diagnosis and treatment of HD and Lower Extremity Amputation Prevention

Information regarding these services is available from:

National Hansen’s Disease Programs
9181 Interline Avenue
Baton Rouge, LA, 70809
Phone: 800-642-2477
Fax: 225-756-3806
B. Other Resources

1. IDEA
   International Association for Integration Dignity and Economic Advancement
   32 Fall Street, Suite #A
   P.O. Box 651, Seneca Falls, NY 13148 USA
   Telephone: 1-315-568-5838
   Toll free: 1-888-647-4939 (U.S. only)
   Fax: 1-315-568-5891
   Website: http://www.idealeprosydignity.org/index.html

2. Leprosy Mailing List Blog
   http://leprosymailinglist.blogspot.com

3. History of Leprosy
   http://www.leprosyhistory.org

4. The National Hansen’s Disease Museum

5. The International Textbook of Leprosy
   http://www.internationaltextbookofleprosy.org/

6. Up-to-Date

XIX. APPENDICES

A - Skin Biopsies (Consent, Instructions, Form)
B - Protocol for Submitting Specimens for Histological Evaluation to the NHDP
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G - Hand Screen Form
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K – Callus and Skin Care
L - Wound Management
M - Basic Wound Care
N - Off loading – Adhesive Felt Relief
O - Off loading – Toe Pillow Fabrication
APPENDIX A

CONSENT FOR SKIN BIOPSY
National Hansen's Disease Program
Baton Rouge, La.

A skin biopsy is a procedure in which a sample of skin tissue is removed, processed and examined under a microscope.

Obtaining a skin sample depends on the size and location of the abnormal area of skin, called a skin lesion.

You will have a punch biopsy and the technique is described as the following:

NOTIFY PHYSICIAN OF ANY ALLERGIES TO ANESTHESIA

After a local anesthetic is injected, a small, sharp tool that looks like a cookie-cutter (punch) is placed over the lesion, pushed down, and slowly rotated to remove a circular piece of skin. The skin sample is lifted up with a tool called a forceps or a needle and is cut from the tissue below. Stitches may not be needed for a small skin sample. If a large skin sample is taken, one or two stitches may be needed. Pressure is applied to the site until the bleeding stops. The wound is then covered with a bandage or sterile dressing.

You may feel brief stinging pain when the local anesthetic is injected. You will not feel any pain when the skin sample is removed.

Although unlikely, there is a slight risk of infection and a slight risk of persistent bleeding. If you usually form scars after skin injuries or surgery, you could develop a scar at the biopsy site.

Your stitches will be taken out 10 to 14 days after the biopsy, depending on the biopsy site. Adhesive bandages should remain in place until they fall off. This usually takes from 7 to 14 days. The biopsy site may be sore for several days. Call your doctor immediately if you have:

• Excessive bleeding or drainage through the bandage. If excessive bleeding occurs, apply pressure to the biopsy site.
• Increased tenderness, pain, redness or swelling at the biopsy site.
• A fever

I understand that photographs and movies may be taken of this procedure, and that they may be viewed by various personnel undergoing training at this facility. I consent to the taking of such pictures and observation of the operation by authorized personnel, subject to the following conditions:

a. The name of the patient and his/her family is not used to identify said pictures.
b. Said pictures are to be used only for purposed medical study or research.

Signature of Physician Date Signature of Patient Date

Patient Identification
Skin Biopsy in the Diagnosis of Hansen’s Disease

Indications for biopsy:

- Possibility of Hansen’s Disease, including: Cardinal signs of Hansen’s Disease, long-standing skin lesions and painless wounds, not responding to conventional topical treatments or therapies, skin lesions in persons with history of travel/ residence in developing country, etc.

Biopsy:

- Select the leading 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 preferred (4 mm punch is sufficient)

Fixation and processing options:

- If only histopathologic examination is desired, fix the biopsy in 10% neutral buffered formalin
- If PCR testing is desired in addition to histopathologic examination, fix the biopsy in 70% Ethanol. Since formalin damages the biopsy’s DNA, reliable results from PCR testing can only be obtained from specimens fixed in 70% Ethanol.
- Paraffin fixed block (both histopathologic exam and PCR testing can be done)
- 4 unstained slides

Send to NHDP:

- Biopsy in formalin OR ethanol OR paraffin block OR 4 unstained slides
- Documentation: Request form and basic patient information (including DOB or age) and clinical questions: Patient treated? If yes, how long? Use of steroids?
- What is the question you want to ask the pathologist?
- Please include a biopsy report from your lab if examined there
- Send by U.S. mail or courier service (UPS, Fed Ex, DHL)

Address:

Clinical Laboratory
National Hansen’s Disease Programs
9181 Interline Avenue
Baton Rouge, LA 70809
Tel 225-756-3733
Protocol and Form for Submitting Specimens for Histological Evaluation of Hansen's Disease

The following are the requirements needed before sending a biopsy for routine histological evaluation:

1. A biopsy collected with a 4 – 5 mm punch (2 mm if on face) or surgical excision, which should be deep enough to include subcutaneous fat. This depth is important because often the most prominently involved nerves will be found in the upper portion of the subcutaneous fat. As a general rule, the biopsy should be taken entirely within the lesion, preferably from the active margin if there is one.

2. Place in 10% buffered formalin, at least 5 volumes of fixative per volume of tissue. Label container with patient’s name and biopsy site.

3. A brief clinical history including number of lesions, changes in sensation, previous diagnosis and present clinical impressions.

4. The patient’s name, sex, race and social security number if available.

5. The patient’s date of birth.

6. The submitting doctor’s name and the address where the report is to be sent.

7. Send biopsy in leak-proof container.

The following specimens may also be submitted for evaluation (listed in order of preference):

1. Paraffin blocks.

2. Slides of unstained sections - preferably at least 4 slides.

3. Stained slides to include H&E and Fite.

Specimens should be placed in protected mailing containers to prevent damage such as screw-cap cardboard cylinders or padded mailing envelopes.

Specimens are then sent to:
National Hansen’s Disease Program
Clinical Lab
1770 Physicians Park Drive
Baton Rouge, LA 70816
Phone 225-756-3733
PATHOLOGY CONSULTATION REQUEST FORM

Submiting Provider/Pathologist Information:
Pathologist Name: __________________________
Phone No: __________________________
Fax No: __________________________
Email: __________________________

TREATING Physician Information:
Physician Name: __________________________
Phone No: __________________________
Fax No: __________________________
Email: __________________________

***Consultation results will be faxed to physicians listed above.

Return Materials To:

Patient Information:
Name (Last): __________________________, (First): __________________________
DOB: __________  Age: __________
Sex: __________ Country of Birth: __________________________
Race: __________________________
Accession #: __________________________ Collection Date: __________
Biopsy Site: __________________________

Brief History: (please check all that apply)
1. Number of lesions: □ 5 or LESS lesions  □ MORE than 5 lesions
2. Does patient experience decreased sensation around biopsy site? □ YES  □ NO
3. Was this condition ever previously treated? □ YES  □ NO  If yes, where? __________________________
4. Has patient been prescribed any of the following?
   * Prednisone □ YES  □ NO  □ CURRENTLY TAKING, DOSAGE: __________
   * Methotrexate □ YES  □ NO  □ CURRENTLY TAKING, DOSAGE: __________
   * Biologics □ YES  □ NO  □ CURRENTLY TAKING, DOSAGE: __________
5. Additional Information:

Mail Pathology Materials To:
National Hansen’s Disease Program
Attn: Clinical Lab-Nurah Al-Ahmed
1770 Physicians Park Drive
Baton Rouge, LA 70816
Phone: 225-756-3733/Fax: 225-756-3734

***Please include a copy of this form, as well as the biopsy report with submission.
APPENDIX C

Preparation and Examination of Skin Smears

The skin smear is a valuable, cost-effective tool in the routine management of the patient with Hansen’s disease. The smear is a means of estimating the number of acid-fast bacteria present, reported as the Bacterial Index (BI), and is important in determining the type and severity of disease as well as assessing the response to treatment.

- Initial skin smears are usually taken from 6 “routine sites” (both earlobes, elbows, and knees) as well as several typical lesions from the patient.
- The time interval between repeat smears to evaluate response to treatment is determined by the physician, but in general, annual smears are adequate for monitoring response to treatment and during the following-up period to detect any evidence of relapse.
- All microscopic slides on which skin smears are made should be pre-cleaned in 70% alcohol, acetone, or alcohol-acetone to remove amorphous debris. The slides are wiped dry with a clean hand towel. Razor blades or scalpels that are used in smear taking are likewise cleaned.
- Slides should be air-dried and NEVER heat fixed.
- They may be sent in protective mailers to:
  National Hansen’s Disease Programs Attention: Clinical Laboratory – Skin Smears
  9181 Interline Avenue, Baton Rouge, LA 70809, Phone: (225) 756-3734

Procedure for Obtaining Smears

1. Universal precautions should be observed in obtaining skin smears.

2. The skin is cleansed with 70% alcohol and air-dried or wiped dry with cotton. (Zephiran tends to make the skin too slippery and is not recommended.)

3. A fold of skin is made relatively avascular by pinching or mild clamping. If the skin cannot be grasped by pinching, it can be compressed. A surgeon’s glove may aid in grasping.

4. Local anesthesia is generally unnecessary. (If there is not adequate decrease in sensation, obtain local anesthesia with 1% Xylocaine or Ethyl Chloride spray.) The compression of the skin by pinching aids in the anesthesia.

5. An incision 3-5 mm long and 2-3 mm deep is made with an alcohol cleansed, single-edged razor blade. A scalpel with a #15 Bard-Parker blade may also be used. Mild pressure to maintain relative avascularity is continuously applied to the area until an adequate smear has been obtained.
6. A small amount of blood does not interfere with the reading, but large amounts should be avoided and can usually be controlled by the amount of pressure of the pinch. If excessive bleeding occurs, it can be wiped away with a cotton swab.

7. After the incision is made, and before the blade is withdrawn, the inner surface of the wound is scraped with the blade held at a right angle to the incision. Upon scraping, tissue fluid and dermal tissue are obtained.

8. The material is transferred to the cleaned microscope slide. A moderately thick smear, with a visible uniform opacity is made. The smear is made in a circular manner on the slide, no larger than a pencil eraser (5-7 mm), beginning peripherally and ending in the center, leaving a central “button” (2-4 mm) which can be easily focused upon with the microscope. Slides should be properly labeled as shown for 3 routine sites.

9. A Band-Aid is generally sufficient to protect the smear site.

10. A single technician takes all smears to insure more uniform and consistent results.

11. The smears are then sent to the National Hansen's Disease Programs for reading.

12. A chart to diagram sites of the skin smears is found in Appendix A, page 3.

**Staining of Skin Smears**

1. Dry the slide with smear at room temperature. **DO NOT HEAT FIX**.

2. Place slides on a staining rack and flood with 10% formalin for 15 minutes for fixation.

3. Gently rinse well with tap water. All formalin must be removed to prevent the formation of precipitates.

4. Flood slides with Ziehl-Neelsen carbol-fuchsin for twenty minutes. The carbol-fuchsin must be filtered before each use. Filtering can be accomplished by placing pre-cut filter paper strips on the slide prior to the addition of stain and left in place for the full twenty minutes.

5. After removing and discarding filter paper strips, gently rinse slides well with tap water to remove excess stain.

6. Decolorize with 2% acid alcohol for 1 minute. This is best accomplished by placing slide into a two-slide plastic slide mailer filled with acid alcohol. Occasional up and down movement of the slide in the acid alcohol should remove all excess carbol fuchsin.

7. Gently rinse slides **thoroughly** with tap water.

8. Counterstain with alkaline methylene blue for 30 seconds to 1 minute.

9. Gently rinse well with tap water and air dry.
NOTE: Positive & negative control slides must be used each day for quality control purposes.

Z-N Carbol Fuchsin Stain:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic fuchsin</td>
<td>1.0 gm.</td>
</tr>
<tr>
<td>Phenol crystals (melted)</td>
<td>5.0 mls.</td>
</tr>
<tr>
<td>95% ethanol</td>
<td>10.0 mls.</td>
</tr>
<tr>
<td>Water, to make</td>
<td>100.0 mls</td>
</tr>
</tbody>
</table>

Dissolve stain in alcohol, and then add phenol/water mixture. Let stand overnight before use. Store in dark brown bottle. Stable for 1 year.

Alkaline Methylene Blue:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>KOH (10%)</td>
<td>0.10 mls.</td>
</tr>
<tr>
<td>Methylene blue</td>
<td>0.35 gms.</td>
</tr>
<tr>
<td>95% ethanol</td>
<td>30.0 mls.</td>
</tr>
<tr>
<td>Water, to make</td>
<td>100.0 mls.</td>
</tr>
</tbody>
</table>

Dissolve the stain in the alcohol, then add the KOH and water mixture and allow to sit overnight.
Filter before use.

Microscopic Examination of Skin Smears

The stained smears are examined with a quality microscope using the oil immersion objective (x100) to determine the total number of bacilli. The same individual should read all smears for the purpose of consistency. The smear will have similar numbers of bacilli throughout. However, four separate quadrants of the smear are examined and averaged to establish the Bacterial Index.

Reporting the Bacterial Index

The results are reported on a 0 to 6+ semi-logarithmic scale using a descriptive phrase or numerical code. This is an indicator of the total bacillary load of the patient. It falls about 1 point per year during effective treatment as dead bacilli undergo lysis and are absorbed.
<table>
<thead>
<tr>
<th>Category</th>
<th>Value</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very Numerous</td>
<td>(+6)</td>
<td>over 1000 bacilli per oil immersion field.</td>
</tr>
<tr>
<td>Numerous</td>
<td>(+5)</td>
<td>100 to 1000 bacilli per oil immersion field.</td>
</tr>
<tr>
<td>Moderate</td>
<td>(+4)</td>
<td>10 to 100 bacilli per oil immersion field.</td>
</tr>
<tr>
<td>Few</td>
<td>(+3)</td>
<td>1 to 10 bacilli per oil immersion field.</td>
</tr>
<tr>
<td>Very few</td>
<td>(+2)</td>
<td>1 to 10 bacilli per 10 fields.</td>
</tr>
<tr>
<td>Rare</td>
<td>(+1)</td>
<td>1 to 10 bacilli per 100 fields.</td>
</tr>
<tr>
<td>None found</td>
<td>(NF)</td>
<td>No AFB seen on entire site.</td>
</tr>
</tbody>
</table>

None found

<table>
<thead>
<tr>
<th>Category</th>
<th>Value</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very Numerous</td>
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<td>Numerous</td>
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<tr>
<td>Very few</td>
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<td>1 to 10 bacilli per 10 fields.</td>
</tr>
<tr>
<td>Rare</td>
<td>(+1)</td>
<td>1 to 10 bacilli per 100 fields.</td>
</tr>
<tr>
<td>None found</td>
<td>(NF)</td>
<td>No AFB seen on entire site.</td>
</tr>
</tbody>
</table>
APPENDIX D

Instructions for Completing the Hansen’s Disease (Leprosy) Surveillance Form

The Hansen’s Disease or Leprosy Surveillance Form (LSF) is the document used to report leprosy cases to the U.S. National Hansen’s Disease Registry. These data are used for epidemiological, clinical, and basic research studies throughout the National Hansen’s Disease Program (NHDP), and are the official source for information on leprosy cases in the U.S. Please report this case to your state health department. The NHDP does not report to state health departments.

The information requested on the LSF is used by many clinicians and researchers, and collection of all information is highly desirable. However, the fields that are boldfaced on the form and in the instructions below are considered to be the minimal information needed to register a patient. Failure to provide this information will result in the form being returned which creates additional work and may cause delays in obtaining program services for the patient.

1. Reporting State: Use the abbreviation of the state from which the report is being sent. This is usually the state of the clinic’s office and not necessarily the patient’s resident state.

2. Date of Report: This is date of the initial LSF completion. If patient was previously reported and has relapsed, write the word “RELAPSE” next to the date.

3. Social Security Number: Optional; (xxx-xx-xxxx)

4. Patient Name: (Last name, First name, Middle name)

5. Present Address: Please include the county and zip code which are used to geographically cluster patients.

6. Place of Birth: Include state and city, if born in the U.S., or the country, if foreign born.

7. Date of Birth/Sex: (mm/dd/yyyy)

8. Race/Ethnicity: This information should be voluntarily provided by the patient. If the patient refuses or indicates a race/ethnicity category not listed, check the “Not Specified” box.

9. Date Entered the U.S.: For patients who have immigrated to the U.S., provide the month and year of entry.

10. Date of Onset of Symptoms: This information is usually the patient’s recollection of when classic leprosy symptoms (rash, nodule formation, paresthesia, decreased peripheral sensation, etc.) were first noticed.

11. Date Leprosy First Diagnosed: Provide the month and year a diagnosis was made. This usually coincides with a biopsy date if one was performed.

12. How many healthcare providers has the patient seen for this problem? This will be based primarily on the patient’s recollection. Include the physician reporting the case.

13. Initial Diagnosis: Was the patient diagnosed in the U.S. or outside the U.S.

14. Type of Leprosy: Classify the diagnosis based on one of the ICD-10-CM diagnosis codes. (NHDP Clinic physicians: Please circle specific classification, if possible)

   A30.5 Lepromatous Leprosy (macular, diffuse, infiltrated, nodular, neuritic – includes Ridley-Jopling [RJ], Lepromatous [LL] and A30.4 Borderline lepromatous [BL]): A form marked by erythematous macules, generalized papular and nodular lesions, and variously by upper respiratory infiltration, nodules on conjunctiva or sclera, and motor loss.

   A30.1 Tuberculoid Leprosy (macular, maculoaesthetic, major, minor, neuritic – includes RJ Tuberculoid [TT] and A30.2 Borderline tuberculoid [BT]): A form marked by usually one lesion with well-defined margins with scaly surface and local tenderness or peripheral nerves.

   A30.0 Indeterminate (uncharacteristic, macular, neuritic): A form marked by one or more macular lesions, which may have slight erythema.

   A30.3 Borderline (dimorphous, infiltrated, neuritic – includes RJ Borderline [BB] or true mid disease only): A form marked by early nerve involvement and lesions of varying stages.

   A30.8 Other Specified Leprosy: Use this code when the diagnosis is specified as “leprosy” but is not listed above (A30.0-A30.3), including “pure neural” disease.

   A30.9 Leprosy, Unspecified: Use this code when the diagnosis is identified as “leprosy” but inactive.

15. Diagnosis of Disease: Reaction? Y if steroids required. Enter INITIAL biopsy and skin smear dates and results.

16. Residence (Pre-diagnosis): List all cities, counties, and states in the U.S. and all foreign countries a patient resided in BEFORE leprosy was diagnosed. This information is used to map all places where U.S. leprosy cases have resided.

17. Disability: Eye, Hand & Foot. For each eye, hand and foot check Yes or No. [Normal always = No]

   Loss of any sensation in hands or feet, for Eyes, is blinking abnormal (very infrequent?). Normal = No

   Visible deformity (muscle wasting, clawing of fingers or toes, ulcers or other abnormality of the hands or feet. For Eyes, lagophthalmos or reduced vision (e.g. cataract). Normal = No


19. Current Treatment for Leprosy: Date that treatment started and indicate all drugs used for initial treatment.
HANSEN'S DISEASE (LEPROSY) SURVEILLANCE FORM
NATIONAL HANSEN'S DISEASE PROGRAM
1770 PHYSICIANS PARK DRIVE
BATON ROUGE, LA 70816
1-800-642-2477

1 Reporting State: ___________________________ 2 Date of Report: ___________________________
3 Social Security Number (optional): ___________________________

4 Patient Name: ___________________________ (Last) ___________________________ (First) ___________________________ (Middle)

5 Present Address: Street ___________________________ City ___________________________
County ___________________________ State ___________________________ Zip ___________________________

6 Place of Birth: State ___________________________ City ___________________________
Country ___________________________ 7 Date of Birth: Sex: Male ☐ Female ☐

8 Race/Ethnicity: ☐ White, Not Hispanic ☐ White, Hispanic ☐ American Indian, Alaska Native ☐ Indian, Middle Easterner
☐ Black, Not Hispanic ☐ Black, Hispanic ☐ Asian ☐ Native Pacific Islander ☐ Not Specified

9 Date Entered U.S.: Mo. _____ Yr. _____ 10 Date of Onset of Symptoms: Mo. _____ Yr. _____
11 Date Leprosy First Diagnosed: Mo. _____ Yr. _____ 12 How many doctors have you seen for this problem? ☐ In U.S. ☐ Outside U.S.

13 Initial Diagnosis: ☐ T.B. ☐ M.L. ☐ Other ☐

14 Type of Leprosy: (ICD-10-CM Code) (NHDP Clinic physicians: Please circle specific classification, if possible)
☐ Lepromatous Leprosy (A30.5 - LL) ☐ Borderline Tuberculoid (A30.1 - BB)
☐ Borderline Lepromatous (A30.4 - BL) ☐ Indeterminate (A30.0 - IN)
☐ Tuberculoid (A30.1 - TT) ☐ Borderline (A30.2 - BT)
☐ Other Specified Leprosy (A30.8) ☐ Leprosy, Unspecified (A30.9)

15 Diagnosis of Disease:
Leprae reaction at diagnosis? ☐ Yes ☐ No
Was biopsy performed in U.S.? ☐ Yes ☐ No
Date _____ / _____ / _____
Result ☐ Yes ☐ No
Skin Smear? ☐ Yes ☐ No Date _____ / _____ / _____
Bl: Positive ☐ Negative ☐

16 List all places in the U.S.A. and all foreign countries a PATIENT resided (Including Military Service) BEFORE leprosy was diagnosed:

<table>
<thead>
<tr>
<th>TOWN</th>
<th>COUNTY</th>
<th>STATE</th>
<th>COUNTRY</th>
</tr>
</thead>
</table>

17 Disability:

<table>
<thead>
<tr>
<th>Hands</th>
<th>Feet</th>
<th>Eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of Sensation?</td>
<td>Right</td>
<td>Yes ☐ No ☐</td>
</tr>
<tr>
<td>Visible deformity?</td>
<td>Right</td>
<td>Yes ☐ No ☐</td>
</tr>
</tbody>
</table>

18 Current Household Contacts: Name/Relationship

1. ___________________________
2. ___________________________
3. ___________________________
4. ___________________________

19 Current Treatment for Leprosy: (check all that apply)

Date Treatment Started: Mo. _____ Yr. _____
☐ Dapsone ☐ Rifampin ☐ Clofazimine
☐ Other (list) ___________________________

20 Name and Address of Physician: ___________________________

Investigator: ___________________________

This form may be FAXED to NHDP at (225) 766-3708
A copy of this form should be sent to your local or state health department.
APPENDIX E

NHDP ANNUAL FOLLOW UP FORM

Date of Exam ______ / ______ / ______

Name: _______________________________ Gender: ______ Date of Birth ______ / ______ / ______

NHDP Clinic OR City / State: ____________________________________________________________

Treating Physician: __________________________ Telephone or E-mail: ______________________

<table>
<thead>
<tr>
<th>Loss of Sensation?</th>
<th>Hands</th>
<th>Feet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Visible deformity?</th>
<th>Hands</th>
<th>Feet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Blink abnormal? Visible abnormality? (see Instructions below)

| Yes | No | Yes | No |

Was patient treated for leprosy reaction (e.g. prednisone required) during the last year? [ ] Yes [ ] No

Status regarding completion of minimum treatment of HD (check one)

1. Continuing in first year of MDT. [ ]
2. Continuing in second year of MDT. [ ]
3. Completed *minimum course of MDT. [ ]
4. Left to follow up [ ]
5. Deceased [ ]
6. Other (re-treatment after relapse, etc.) [ ]

Month and year: ____________________________

*Minimum=1 yr. for PB disease, 2 yrs. for MB disease

INSTRUCTIONS:

Disability: Eyes, Hands & Feet:

For each eye, hand and foot, check Yes or No for:

Loss of sensation:

Hands & Feet: Y = loss of sensation at 2 points

Eyes: Y = blinking is abnormal (very infrequent)

Normal eyes = No

Visible deformity:

Hands & Feet: Y = Muscle wasting, clawing of fingers, wounds or ulcers

Eyes: Y = Lagophthalmos, Reduced vision, Uveitis, etc.

Leprosy reaction during the last year: Y = ANY reaction requiring corticosteroids

This form may be Faxed or Mailed to:

NATIONAL HANSEN'S DISEASE PROGRAM
MEDICAL DEPARTMENT
1770 PHYSICIANS PARK DRIVE
BATON ROUGE, LA 70816
ATTENTION: BERYL ASBERRY, MHR, MISM, BS
FAX (225) 756-3706

NHDP FORM 208 REV/AUG 2015
NHDP FOLLOW UP FORM
APPENDIX F

EYE EVALUATION

Patient's Name (Last, First, Middle): DOB: Pt. File No.: Initial __ F/U __

Complaints/Changes:

Section I. SENSORY TESTING (Trigeminal Nerve)
Sensation in the eye is determined by examining the patient for delayed or absent BLINK
Record: S or L according to sensory findings (observation):
S = Sensation intact (normal, symmetrical blink)
L = Loss of Sensation (delayed or absent blink)
BLINK
Right ___ Left ___

Section II. MUSCLE TESTING (Facial Nerve)
Muscle strength in the eye is tested by having the patient hold both eyes tightly closed against resistance
Record: S, W or P in the box according to muscle test findings
S = Strong-patient can hold position against full resistance
W = Weak-patient can close eyes, but not able to hold closed against resistance
P = Paralyzed-patient cannot fully close eyes

Section III. HD DEFORMITY: (Check if present)

Section IV. ADDITIONAL COMPLICATIONS: (Check if present and known to be HD related)

Section IV. VISUAL ACUTITY: is tested using an eye chart
- Test each eye separately
- 20/200 or worse is considered "decreased"

VISUAL ACUTITY
Right ___ Left ___

Section VI. WHO Grade: check WHO grade level for each eye according to screen results

<table>
<thead>
<tr>
<th>WHO Grade</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal blink</td>
</tr>
<tr>
<td>1</td>
<td>Loss of protective sensation (delayed or absent blink) No HD deformity and Vision better than 20/200</td>
</tr>
<tr>
<td>2</td>
<td>Loss of protective sensation (delayed or absent blink) + HD related deformity or decreased visual acuity (worse than 20/200)</td>
</tr>
</tbody>
</table>

Examined by: __________ Date: __________
Entered by: __________ Date: __________
APPENDIX G

HAND EVALUATION

<table>
<thead>
<tr>
<th>Patient’s Name (Last, First, Middle):</th>
<th>DOB:</th>
<th>Pt. File No.:</th>
<th>Initial <em>F/U</em></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Complaints/Changes:

Section I. SENSORY TESTING: Begin with green filament. Mark filament number on corresponding line for each positive response. If no response, use the next heaviest filament until all sites are scored.

<table>
<thead>
<tr>
<th>FILAMENT NUMBER</th>
<th>FORCE (gms)</th>
<th>INTERPRETATION</th>
<th>SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.83 (Green)</td>
<td>0.05</td>
<td>Normal Sensation</td>
<td>5</td>
</tr>
<tr>
<td>3.61 (Blue)</td>
<td>0.20</td>
<td>Residual Texture Sensation</td>
<td>4</td>
</tr>
<tr>
<td>4.31 (Purple)</td>
<td>2.00</td>
<td>Protective Sensation</td>
<td>3</td>
</tr>
<tr>
<td>4.65 (Red)</td>
<td>4.00</td>
<td>Loss of Protective Sensation</td>
<td>2</td>
</tr>
<tr>
<td>6.65 (Red Line)</td>
<td>300.00</td>
<td>Deep Pressure Sensation</td>
<td>1</td>
</tr>
<tr>
<td>6.65 (Red Line)</td>
<td>No</td>
<td>Insured Deep Pressure Sensation</td>
<td>0</td>
</tr>
</tbody>
</table>

Dark: N/A Missing or Inaccessible

Section II. SKIN INSPECTION: Describe skin condition in space provided below:
W-Wound, C-Callos, S-Swelling, R-Redness, D-Dryness, T-Temperature, M-Missing, J-Contracture, O-Other

Section III. MUSCLE TESTING: Mark: S-Strong, W-Weak, P-Paralyzed (or Grade 5-0)

<table>
<thead>
<tr>
<th>(Ulnar Nerve)</th>
<th>(Median Nerve)</th>
<th>(Radial Nerve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R_ L_</td>
<td>R_ L_</td>
<td>R_ L_</td>
</tr>
<tr>
<td>1) Index finger Abduction (FDI)</td>
<td>2) Little Finger MP Joint Flex. (L)</td>
<td>3) Thumb Abduction Out of Palm (APB)</td>
</tr>
<tr>
<td>4) Thumb to Little Finger (OP)</td>
<td>5) Radial Wrist Extension (ECR)</td>
<td></td>
</tr>
</tbody>
</table>

Section IV. NERVE PALPATION:

<table>
<thead>
<tr>
<th>Ulnar (at Cubital Tunnel)</th>
<th>Median (at Carpal Tunnel)</th>
<th>Radial Cut. (Proximal to snuff box)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R_ L_</td>
<td>R_ L_</td>
<td>R_ L_</td>
</tr>
<tr>
<td>Enlarged</td>
<td>Tender</td>
<td>Tenderness</td>
</tr>
</tbody>
</table>

Section V. DEFORMITY: (Check if present)

<table>
<thead>
<tr>
<th>Open Wounds</th>
<th>Clawed but mobile hand</th>
<th>Contracted or stiff joints</th>
</tr>
</thead>
<tbody>
<tr>
<td>R_ L_</td>
<td>R_ L_</td>
<td>R_ L_</td>
</tr>
<tr>
<td>Amputation/Absorption</td>
<td>Wrist Drop</td>
<td>Other</td>
</tr>
</tbody>
</table>

Section VI. W.H.O. GRADE

<table>
<thead>
<tr>
<th>WHO Grade</th>
<th>Description</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Protective sensation: Can feel 4.31 (2g) filament or better at all test sites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Loss of protective sensation: Does NOT feel 4.31 (2g) filament and NO AD damage/deformity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Loss of protective sensation and HD damage/deformity: Does NOT feel 4.31 (2g) filament &amp; has HD related damage/deformity</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Examined by: ______________________  Date: ______________________
Entered by: ______________________  Date: ______________________

NHSF FORM 130 HAND EVALUATION REV OCT 2017
APPENDIX H

FOOT EVALUATION

| PROGRAM NAME: |
| Patient's Name (Last, First, Middle): | DOB: | Pt. File No.: | Initial F/U |

Complaints/Changes:

Section I. SENSORY TESTING: Begin with 1 gm filament. Mark SCORE on corresponding line for each positive response. If no response, use the next heaviest filament until all sites are scored.

<table>
<thead>
<tr>
<th>FILAMENT NUMBER</th>
<th>FORCE</th>
<th>INTERPRETATION</th>
<th>SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.17 (Green)</td>
<td>1 gm</td>
<td>Normal Sensation</td>
<td>3</td>
</tr>
<tr>
<td>5.07 (Purple)</td>
<td>10 gm</td>
<td>Protective Sensation</td>
<td>2</td>
</tr>
<tr>
<td>6.10 (Red)</td>
<td>71 gm</td>
<td>Loss of Protective Sensation</td>
<td>1</td>
</tr>
<tr>
<td>6.10 (Red Line)</td>
<td>No Response</td>
<td>Impaired Deep Pressure Sensation</td>
<td>0</td>
</tr>
<tr>
<td>Black</td>
<td>N/A</td>
<td>Missing or Inaccessible</td>
<td></td>
</tr>
</tbody>
</table>

Section II. SKIN INSPECTION: Describe skin condition in space provided below:

W-Wound, C-Callus, S-Swelling, R-Redness, D-Dryness, T-Temperature, M-Missing, J-Joint Contracture, O-Other

Section III. MUSCLE TESTING: Mark: S-Strong, W-Weak, P-Paralyzed (or Grade 0-5)

1) Ankle Dorsiflexion
   Tibialis Anterior Muscle (Peroneal Nerve)

2) Spread Toes
   Intrinsic muscles (Tibial Nerve)

Section IV. NERVE PALPATION:

Common Peroneal (at Fibular Head)
Posterior Tibial (at Med. Malleolus)
Sural Sensory (at Lat. Lower Leg)

Section V. DEFORMITY: (Check if present)

Open Wounds
Claw Toes
Equinus

FOOTWEAR:

Is footwear appropriate for Risk Category?

Yes ___ No ___

Other:

Section VI.

W.H.O. GRADE

RISK Category

<table>
<thead>
<tr>
<th>WHO Grade</th>
<th>R</th>
<th>L</th>
<th>Description</th>
<th>RISK Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td></td>
<td></td>
<td>Protective sensation (Can feel 10 gm filament at all test sites)</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td>Loss of protective sensation (Does NOT feel 10 gm filament and NO HD deformity)</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td>Loss of protective sensation and HD related deformity (Does NOT feel 10 gm filament and has HD related deformity)</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td>History of Plantar Ulcer</td>
<td></td>
</tr>
</tbody>
</table>

Examine by: ___________________________ Date: ___________________________

Entered by: ___________________________ Date: ___________________________

NHDP FORM 133 FOOT EVALUATION REV OCT 2017

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APPENDIX I

HEALTHY EYES, HANDS AND FEET FOR A LIFETIME

National Hansen’s Disease Programs
Baton Rouge, Louisiana
Phone: 1-800-642-2477
https://www.hrsa.gov/hansens-disease
NERVES

Nerves are like electrical wires in the body that carry information to and from the brain and other body parts. Without treatment, Hansen’s disease can damage some of the nerves in the face, hands and feet. This booklet was made to help you understand what signs to look for to help you protect your nerves and prevent deformity.

The nerves of your eyes, hands, and feet control the following:

1. **Mucous Membranes** that affect your tears which keep your eyes moist.
   **Sweat Glands** that keep the skin on your hands and feet soft and moist.

2. **Muscle Balance and Strength** that enable you to blink and close your eyes; and allow you to use your hands and feet in normal daily activities.

3. **Sensibility** allows you to feel, and is your warning system to protect your eyes, hands, and feet from injury.

The arrows on the next page point to the places where Hansen's disease usually affect the nerves. At these places, the nerves are just under the skin and are a cooler temperature than those buried deep under muscle. Damage to the nerve at these sites can cause some typical problems. These problems can be prevented if the symptoms are caught and treated as early as possible.

*****************************************************************************
Please see your doctor if you notice any discolored areas, tingling, pain, or swelling at the places shown by the arrows; or if you have any changes in the feeling or movement of your eyes, hands, or feet.
*****************************************************************************
You can find early changes in your nerves before anyone else if you look for the signs and symptoms listed above and ask yourself the questions on the following three pages.

Finding the early changes and reporting them to your doctor is the best way to prevent injury and deformity. The Eye, Hand, and Foot Screen in this booklet are for you to test yourself for signs and symptoms of nerve damage.

(You may need the help of a family member or friend; or a mirror to answer the questions about your eyes, hands, and feet.)
EYE SCREEN

1. Can I close my eyes like I'm sleeping?

2. Can I squeeze my eyes shut tight?

3. Does the pupil (black center) of my eye look round?

4. If something gets in my eye, do I feel it?
   If you answered "NO" to any of the questions 1-4; Please see your doctor to report your eye problems.

5. Are my eyelids sticky in the morning?

6. Are my eyes red, swollen, painful, or watery?
   If you answered "YES" to question 5 or 6; Please see your doctor to report your eye problems.
HAND SCREEN

1. Are there any places on my hands that are tingling or feel numb?
2. Are there any places on my hands or arms that get a sharp, shooting pain?
3. Are there sores, cuts or blisters on my hands that I cannot feel?
4. Is the skin on my hands dry or cracked?
5. Do I drop things easily?

If you answered **YES** to any of the above questions, see your doctor.

6. Am I able to put my hands together with my palms and fingers flat against each other?

If you answered **NO** to the last question, see your doctor.
FOOT SCREEN

1. Are there any places on my feet that are tingling or feel numb?

2. Are there any places on my feet or legs that get a sharp, shooting pain?

3. Are there sores, cuts or blisters on my feet that I cannot feel?

4. Is the skin on my feet dry or cracked?

If you answered **YES** to any of the above questions, see your doctor.

5. Can I stand up on my toes and on my heels?

6. Are my toes straight?

If you answered **NO** to either of the last two questions, see your doctor.
**TREATMENT**

**Protection of Nerves:**
It is important to rest and protect an inflamed nerve. Movement may cause more damage to the nerve. Some ways to protect an inflamed nerve from further damage include:

**Immobilization:**
- **Eyes:** Artificial tears or ointment plus a mask or tape to hold the eyelids shut at night.
- **Hands:** Use of an elbow pad, arm splint, or hand splint may be provided to prevent movement that would harm your inflamed nerve.
- **Feet:** A compressive sock or elastic wrap may be used to decrease the swelling in your leg.

**Medication:**
Prednisone or another medication may be prescribed by your doctor to treat an inflamed nerve. This medicine will decrease the swelling of the nerve and may improve your sensibility and muscle strength if they have been affected. This medication comes in pill form to take by mouth, or as drops/ointment for the eyes.
TREATMENT

Positioning:

DO NOT sleep with elbows or wrists fully bent. Use a pillow or elbow pads to help keep them straight.

DO NOT sit with legs fully bent as in a squatting position or cross-legged.

Daily Activities, Work, and Exercise:

LIMIT repeated movements and lifting if nerves in the arms are inflamed.

LIMIT walking if nerves in the legs are inflamed.

Wear sunglasses and a wide brimmed hat to protect your eyes from light and dust.
TREATMENT

Skin Care:
If your skin is dry from nerve damage affecting the sweat glands, there are a few simple things that you can do to care for your skin and prevent further problems:

- Carefully inspect the skin on your hands, feet and between your toes everyday. Look for dryness, callus, blisters, and wounds.

- Wash your hands and feet with soap and warm (NOT HOT) water when you bathe and any other time it is needed for cleansing. (Safe water temperature is below 110 degrees)

- It is not recommended to soak your feet. In fact, prolonged soaking can actually cause more dryness to the skin.

- After bathing, dry your feet well especially between your toes.

- When your feet are dry, apply lotion that does not contain perfume or alcohol in the ingredients. Do not apply lotion between your toes as it can cause the skin to breakdown.

- Your fingernails and toenails need attention on a regular basis. Use a nail clipper and nail file to cut your nails and NOT a knife, scissors, or razor blades. Your toenails should be cut straight across to prevent ingrown nails.

- If you do notice a callus or find a wound, notify your doctor for help to resolve the problem.
PROGRESSION TO DEFORMITY

It is important to follow the instructions in this booklet so you can find and report early nerve changes in your eyes, hands and feet.

Without treatment, nerve damage can lead to permanent damage, deformity and blindness as shown below.

<table>
<thead>
<tr>
<th>SENSIBILITY</th>
<th>MUSCLES</th>
<th>MUCOUS/SWEAT GLANDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numbness</td>
<td>Weakness</td>
<td>Dry skin/Dry eyes</td>
</tr>
<tr>
<td>Tingling/Eye Irritation</td>
<td>Paralysis</td>
<td>Skin cracks/fissures</td>
</tr>
<tr>
<td>Decreased feeling/vision</td>
<td>Deformation</td>
<td>Eye ulceration</td>
</tr>
<tr>
<td>Wounds/Infection</td>
<td></td>
<td>Infection</td>
</tr>
<tr>
<td>Deformity/Blindness</td>
<td>Amputation/Blindness</td>
<td>Deformity/Blindness</td>
</tr>
</tbody>
</table>

Deformity from Hansen’s Disease
RESOURCES

Help is available for those with Hansen’s disease
The following resources can provide you with information and assistance to evaluate, treat, and fully understand your disease.

The National Hansen’s Disease Programs includes:

*Headquarters in Baton Rouge, Louisiana
*Ambulatory Care Regional Clinics in the following cities: Los Angeles, Miami, New York, multiple sites in Texas, and Puerto Rico
*Designated Private Physicians throughout the country

Medical, Rehabilitation, and Psychosocial needs:
Contact: 1-800-642-2477
(ask for a physician, therapist, or social worker)

Website: https://www.hrsa.gov/hansens-disease

******************************************************************************

IDEA (International Association for Integration Dignity and Economic Advancement for people with Hansen’s disease).
Providing emotional support for people with Hansen’s disease.

32 Fall Street, Suite #A
P.O. Box 651
Seneca Falls, NY 13148
Phone: 1-315-568-5838 or 1-888-647-4939
Email: alaw@idealeprosydignity.org
Website: www.hansensdisease.org
Support Group: www.idealeprosydignity.org
Contact Person: Nicole Holmes 1-866-637-1525
Email: nholmes@hansensdisease.org
**Nail Care**

**Nail Care Supplies**
- Nail Clippers
- Dust mask
- Vacuum
- Dremmel tool
- Sanding discs
- Nail files or fine grit sandpaper

**Most people require their nails to be trimmed every 4-6 weeks**

**Cut nails straight across with corners visible.**

**Trim nails from one side to the other in small nips to avoid splitting.**

**Smooth out rough edges with a Dremmel tool, nail file, or sand paper.**

**Use sanding surface only. Do NOT use disc edge or center point. Constant motion of tool will prevent burn to skin or nails.**

**Sand down thick nails to prevent pressure.**

**Short, Curved, V-shape**

**Improper way to cut nails**

Do NOT cut nails too short, curved or v-shaped. This can cause ingrown nails.

**Ingrown nails**

**Long nails can snag on clothing or bedding.**

**Thick nails can cause pressure and damage to underlying skin.**
Callus & Skin Care

Supplies
- #10 or #15 blade scalpels
- Dremmel tool & sanding discs
- Vacuum
- Dust mask
- Callus files or fine grit sandpaper
- Products to hydrate the skin

Calluses are thick, hard layers of skin that develop as the skin tries to protect itself against friction and pressure. Callus needs to be trimmed regularly to prevent ulcers. Hold the scalpel parallel to the skin surface. Carefully remove callus down to soft tissue. Remove remaining callus and smooth rough skin with a Dremmel tool, callus file or fine grit sandpaper. Note the pre-ulcerative sites that were underneath the callus. If the callus had not been removed, an ulcer could have developed.

Some callus can be prevented by protective gloves, footwear & orthotics. Nerve damage can cause loss of sweat glands which leads to dry skin, cracks, and fissures. Dry scaly skin on hands/feet can be removed by daily washing. Use an oil based product like Vaseline to seal in moisture. DON'T use lotions with perfume.
Wound Management

Hypergranulation Tissue
When healthy tissue rises above the wound edges, it can prevent normal healing.
Treatment: Apply Silver Nitrate

Infection
Signs of infection: Redness & swelling, odor, increased drainage, pus formation, pain. Alert the doctor of these changes.
Treatment: Antibiotics

Neuropathic Wound
These ulcers occur when the patient cannot feel their hands and feet.
Treatment: Pressure relief & offloading with casting and splinting.

Venous Insufficiency
When the veins no longer function properly, chronic swelling can cause ulcers on the lower leg and ankle.
Treatment: Local wound care combined with compression.

Arterial Insufficiency
Poor circulation will prevent ulcers from healing.
Treatment: Surgery to improve blood flow

Maceration
When wound drainage is not controlled, the edges of the wound stay wet and prevent healing.
Treatment: Change the dressing more often or use a more absorbent bandage.

Undermining
When pressure is not relieved, the wound edge can detach from the wound bed and prevent healing.
Treatment: Pressure relief & offloading with casting and splinting.

Squamous cell carcinoma
Wounds that do not heal with proper treatment or wounds that have been present for a long period of time may contain cancer cells. Alert the doctor.
Treatment: Surgical debridement
Basic Wound Care

Black Wound
- The black wound may require aggressive debridement
- Schedule an appointment with the doctor
- Keep wound covered with gauze

Yellow Wound
- Clean the wound to remove yellow tissue by gently scrubbing with gauze or lightly scraping with a scalpel
- Keep wound moist
- Change gauze dressing as often as necessary to absorb drainage

Mixed Wound
- Pick the color that is most prominent within the wound and treat accordingly
- Gently scrub yellow and black areas with gauze or scrape with scalpel
- Rinse with saline or clean water and dress with gauze

Red Wound
- A red wound is a healthy wound
- Protect the red wound
- Gently clean but NO scrubbing
- Avoid bleeding
- Keep wound moist
- Change the dressing as little as possible

Proper Dressing Change Technique

1. Wash your Hands
Wash your hands with soap and water for 15-20 seconds. Wash between fingers and under nails. Rinse and dry thoroughly. Use hand sanitizers if soap and water are not available.

2. Remove Old Dressing
Put on clean protective gloves. Gently remove the old dressing and turn it inside-out over the old dressing and throw it in the garbage. Put on new gloves again before cleaning and dressing the wound. Do not touch the wound with your fingers.

3. Clean the Wound
Rinse and clean the wound with saline or clean water according to the wound color. If scrubbing or wiping the wound, clean from the center of the wound outward. Use a clean gauze to dry the surrounding skin.

4. Dress the Wound
Hold the edge of the gauze when placing it on the wound to prevent contamination. Secure gauze with tape or wrap the gauze in place. Do not let the dressing get wet. When you see the drainage on the outside of the dressing, it is time to change the gauze.
Adhesive Felt Relief

- Cover wound with opsite or other transparent dressing
- Mark exact wound location with lipstick
- Trace foot from base of heel to end of toes on adhesive side of felt pad
- Lipstick should transfer onto felt pad
- Trim outline of foot and around the wound location
- Bevel the felt pad to prevent edge pressure
- Remove adhesive back from felt pad
- Adhere felt pad to plantar foot
- Apply gauze dressing directly over wound

* Make 2-3 felt pads in case it becomes soiled or wet
* Use skin prep to remove any oil or dirt from the skin to improve adherence
* Instruct the patient not to get the felt pad wet to avoid skin breakdown
Toe Pillow Fabrication

1. 5" x 2.5" piece of moleskin and 2x2 gauze pad
2. Peel adhesive back off moleskin
3. Roll up gauze pad and place in center of moleskin
4. Reflect moleskin over the gauze pad and try to avoid wrinkles
5. Trim moleskin

- This is the desired shape
- Cut small hole just above gauze pad large enough for toe
- Slip over involved toe and check for fit
- Check fit in shoe or sandal

* Make 2-3 extra toe pillows in case it becomes soiled or wet
* You can make the pillow wider to incorporate multiple toes
* Recommend taping the pillow in place to prevent shifting