Pregnancy and Lactation

Leprosy frequently affects women of childbearing age. Many will undergo pregnancy and lactation before, during, or after Multi-Drug Therapy (MDT) while still at risk of reactions, or when coping with nerve function impairment (NFI) or eye problems due to leprosy. These vulnerable women require coordinated care from obstetricians/midwives and from leprosy-trained health workers (Figure 1).

Considering its frequency, there is surprisingly little published evidence regarding either the natural history of leprosy during pregnancy and lactation or its impact on fetal outcomes. Even less is known about the psychosocial aspects of childbearing in leprosy-affected women. A systematic review (1) concluded that research was needed to answer questions about the risk of reaction/NFI as well as about pregnancy outcomes, including (i) observational cohort studies of women of childbearing age (both during and after MDT), comparing those who become pregnant with those who do not, and (ii) randomized controlled trials to clarify the effects of drugs used in leprosy on pregnancy and lactation. Nearly two decades later very little more is known on these subjects.

CONCEPTION

While infertility is an observed (though often overlooked) complication among men with a history of borderline lepromatous (BL) leprosy or lepromatous leprosy (LL), infertility due to leprosy does
not appear to be a problem in women (2, 3). In low-resource situations, uninvestigated childlessness in leprosy-affected couples may be due to the male hypogonadism (see Chapter 2.1) caused by leprosy, but women commonly feel shamed by relatives for their “failure to conceive”.

Contraception should be openly addressed at leprosy clinics, since the efficacy of contraceptive drugs may be diminished due to rifampin-associated induction of liver enzymes, though this diminishment is unlikely to be significant if rifampin is used only once monthly. In addition, deferral of pregnancy may be advisable in some circumstances, though termination of pregnancy due to leprosy is not justifiable on medical grounds (Box 1).

RISKS TO FETUS

The congenital transmission of leprosy has never been established, though there are a few reports of *M. leprae* being detected in placental tissue (4, 5). Most reported cases of leprosy in very young infants are explicable by exogenous airborne infection of the newborn from the mother or another untreated leprosy case, and the short incubation period does not prove antenatal infection (6, 7). While a Caesarian section is occasionally performed for mothers because of their leprosy (8), the authors do not believe that this surgery is justified in view of the negligible risk of intrapartum infection.
No teratogenic effects are expected from standard MDT (see Chapter 2.6) with rifampin, clofazimine, and dapsone (9, 10, 11), and any potential small risks of MDT are outweighed by the benefits of treatment (Box 2). Women who need MDT should be specifically advised at each clinic visit to continue treatment during pregnancy (9, 12) and to avoid treatment discontinuation due to maternal anxiety regarding fetal health or inappropriate advice from other health professionals. Patients are recommended to take 5 mg of folic acid daily with dapsone throughout pregnancy because of the potential for a small decrease in folate absorption. Although rare, dapsone has been associated with neonatal hemolysis and methemoglobinaemia (13, 14) and rifampin with neonatal bleeding, both related to their use in the third trimester. Testing for G6PD deficiency (if available) is recommended to identify and avoid severe hemolysis due to dapsone. Accompanied MDT (AMDT; see Chapter 2.6) (15) can be a useful approach if it is difficult for the woman to reach the clinic in the later stages of pregnancy or during the postpartum period, though ongoing close observation is preferred in view of the potential complications of pregnancy.

Women taking MDT (or anti-reaction drugs) should inform their obstetrician/midwife about all other medications being taken. Clofazimine will result in darkened skin color in the baby, noticeable at birth and fading when breastfeeding ceases (or when the mother discontinues clofazimine), but to date no safety concerns for infants have been reported (16, 17, 18).
BOX 2 Drugs commonly used during pregnancy and lactation

Summary recommendations on use during pregnancy and lactation of medications for treatment of leprosy infection and complications of leprosy

Anti-leprosy drugs

Rifampin
Generally safe in pregnancy and lactation. Some association with increased risk of neonatal bleeding when given in 3rd trimester.

Dapsone
Generally safe in pregnancy and lactation, subject to normal considerations of risk of adverse reactions. Some association with neonatal hemolysis and methemoglobinaemia when given in 3rd trimester. Supplementary folic acid is recommended because of theoretical risk of lower folate absorption.

Clofazimine
Safe in pregnancy and lactation. May lead to reversible discoloration of the breast-fed child.

Ofloxacin
Avoid in pregnancy and lactation due to risk of arthropathy (in animal studies).

Minocycline
Teratogenic in pregnancy and can cause infant teeth discoloration and damage through passage in breast milk.

Anti-reaction drugs

Prednisolone
Risk benefit analysis favors use in moderate to severe reactions. Monitor for adverse effects, especially hypertension and hyperglycemia. Small increase in incidence of cleft palate in first trimester, and theoretical risk of hormonal disturbance in the fetus. Safe in lactation, but deferring feeding until four hours after ingestion of drug will reduce infant’s exposure to peak concentration of drug secreted in breast milk.

Thalidomide
Contraindicated in women of childbearing age due to teratogenicity.

Immunosuppressants: i.e., Cyclosporine, Methotrexate, Azathioprine
Contraindicated in pregnancy and lactation.

Prophylactic drugs

Bacillus Calmette-Guerin
Contraindicated in pregnancy and lactation.

Antibiotics for ulcer-related infections:
Weigh up risks and benefits considering safety of specific drug in pregnancy and lactation, known microbiology, and local resistance patterns.

In rare cases in which a woman of childbearing age needs second-line chemotherapy for rifampin-resistant leprosy or other reasons, specialized advice should be sought. Unless reliable contraception can be assured, minocycline should be avoided (because of its teratogenic potential) and ofloxacin should only be used with caution (because of concerns about arthropathy) (9).

BREASTFEEDING

A few cases of leprosy in infants have been reported where infection may have occurred during breastfeeding (6, 7), but these reports date prior to the introduction of MDT, when secondary dapsone resistance was common. Acid-fast bacilli resembling *M. leprae* have been demonstrated using light microscopy in breast milk of lepromatous women not treated with MDT (19), but viability is uncertain, and there is no evidence that *orally ingested* *M. leprae* causes leprosy. The aire-borne infection risk (from the mother’s respiratory tract in the close proximity of breastfeeding) is negligibly low if the mother is on or has completed MDT. There is some secretion of antileprosy drugs in breast milk, but expert opinion is that breastfeeding by women on MDT is safe for infants and may even provide some protective effects (11, 12).

In conclusion, women should be advised not to withhold breastfeeding, whether for fear of harm to the child from MDT or for fear of infecting the child with leprosy. Alternative methods of infant feeding are unsafe in many leprosy-endemic areas due to the risk of malnutrition and unsanitary water sources (Figure 2).

**FIG 2 Leprosy-Affected Mother Under Treatment, Confidently Breastfeeding Her Child.**
LEPROSY REACTIONAL EPISODES

Most epidemiological data on reaction/neuritis associated with pregnancy predates the MDT era and may not apply to the present. Dapsone monotherapy was typically long term, and the end of treatment was partly determined by a prolonged reaction-free period, which makes comparison with the present MDT era difficult, especially considering dapsone’s immunomodulatory effects. Besides, some episodes of “late reaction” were associated with reactivation of the disease due to unrecognized secondary dapsone resistance. Some cohort studies and case series have been reported, but population-based evidence and more up-to-date studies are needed (Table 1) (1, 20, 21, 22, 23, 24, 25, 26).

Women of childbearing age should be offered preemptive family planning advice, especially if they are at high risk (e.g., MB [multibacillary leprosy] cases with pre-existing NFI) (27). Published evidence to date suggests an increase in the frequency of Type 1 reactions (see Chapter 2.2), mostly in the early postpartum period. During the first and third trimesters, and also postpartum, ENL (erythema nodosum leprosum) may occur more frequently in pregnant than in non-pregnant women, with an earlier onset of NFI (1). This increased susceptibility to reactions/neuritis during and soon after pregnancy is associated with a “Th1-Th2 shift” (see Chapter 6.3) during pregnancy and the rapid restoration of immune capacity after delivery (28). When Lucio’s phenomenon occurs in pregnancy it can be very serious (29), but there is no published evidence on its incidence.

ANTI-REACTION TREATMENT

Health care providers should always ask about pregnancy when treating a woman of childbearing age with anti-reaction drugs. There is no published evidence available on the most effective steroid regimen specifically for pregnant women nor on the outcomes (remission of reaction, recovery of NFI, etc.) of steroid treatment during pregnancy (30). Hence the usual practice is to prescribe the same regimen as that given to other patients with leprosy reactions.

The use of corticosteroids for managing Type 1 reactions, neuritis, or ENL is generally safe during pregnancy, although extra precautions need to be taken to monitor for adverse effects such as steroid-induced hyperglycemia or hypertension. The WHO (9) advises that the commonly used oral steroid, prednisolone, only be used during pregnancy if the risk to the mother of withholding it appears to justify the risk of its adverse effects. Evidence of teratogenicity is meager, namely, a small increase in the incidence of cleft palate if it is given in the first trimester and a theoretical risk of hormonal disturbance in the fetus (31). Prednisolone can be safely used in breastfeeding women, but deferring feeding until four hours after the ingestion of the drug will reduce the infant’s exposure to peak concentration of prednisolone secreted in breast milk (31).

Immunosuppressants such as azathioprine, methotrexate, or cyclosporin are contra-indicated in pregnancy. A high dose of clofazimine has not been shown to be teratogenic (16, 17) and is usually well tolerated during pregnancy. A positive risk-benefit ratio for chloroquine in treating mild reactions in pregnancy has not been established; it is best avoided in the first trimester (32).
Thalidomide (for an ENL reaction) should **never** be used for pregnant women or women at risk of pregnancy since its teratogenic effect occurs early in the first trimester. In some countries, its use in women of childbearing age is permitted provided adequate contraceptive and monitoring precautions are ensured (33).

**PROGNOSIS FOR INDIVIDUAL CASES**

There is insufficient evidence available on risk factors that predict the probability of a first episode of reaction or its likely duration for pregnant women. For women still on MDT, or those who completed MDT within the past two years, best practice would be close observation throughout the pregnancy, continuing for up to 12 months postpartum. Each monthly, or more frequent, visit would include a history, physical examination, nerve function assessment, and visual acuity assessment, in addition to enquiries about the infant’s welfare.

**CHILDCARE ISSUES**

For a mother who is disabled (NFI or visual loss), sick with a reaction, or needing long admissions, childcare presents many practical problems. The baby’s general health and development, as well as that of other children, may be at risk, and the mother may be at an increased risk of postpartum depression. In some cases, abandonment by the husband and relatives is another risk. A referral to social services should be considered, as well as the counseling of family members to encourage and facilitate their support (Figure 3).

**LIVING WITH DISABILITY**

An increase in weight and change of gait may put pregnant women with nerve-damaged feet at an increased risk of trophic ulceration (see Chapter 4.3). The enhanced risk continues after delivery when the mother carries the child. All appropriate POD (prevention of disability) and rehabilitation interventions should be made available (see Chapter 4.3). Any use of antibiotics for foot sepsis should be done cautiously, checking for safety during pregnancy and lactation (Box 2).

**NEW DIAGNOSES/RELAPSE/REACTIVATION**

Older case series (from the pre-MDT period) have reported an increased risk of “reactivation of leprosy”, or relapses during pregnancy and the puerperium (34). Some of these relapses were instances of dapsone-resistance occurring in patients on dapsone monotherapy (21). In some instances, a “late reaction” may have been mistaken for a relapse. Theoretically, a period of relative immune suppression, such as pregnancy, may favor the multiplication of dormant bacilli (persisters). An apparently increased incidence of leprosy during pregnancy (21) might reflect changes in
### TABLE 1 Frequency of reactional episodes during and soon after pregnancy

<table>
<thead>
<tr>
<th>Study Reference</th>
<th>Location and Type of Study</th>
<th>Period of Observations</th>
<th>Number of Pregnancies/Subjects</th>
<th>Type of Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maurus, 1978 (20)</td>
<td>Leprosarium in USA. Retrospective case note review</td>
<td>1957–1975 (Dapsone era)</td>
<td>62 in 26 women, Including 13 American, 13 foreign-born</td>
<td>Active cases: 18 LL cases, 5 borderline, 3 tuberculoid</td>
</tr>
<tr>
<td>Duncan, 1981, 1982A (21, 22)</td>
<td>Ethiopia, clinic at leprosarium. Prospective cohort study</td>
<td>1975–1978 (Era of increasing Dapsone resistance)</td>
<td>Total observed= 156 in 147 women. 119 pregnancies in 114 women with leprosy</td>
<td>89 new and on treatment (including 72 LL/BL), and 25 RFT women and 33 controls.</td>
</tr>
<tr>
<td>Duncan, 1982B (23)</td>
<td>Ethiopia. Prospective cohort study</td>
<td>1975–1978 (Era of increasing Dapsone resistance)</td>
<td>119 pregnancies in 115 women with leprosy</td>
<td>39 women were TT/BT, 44 were BL, 32 were LL</td>
</tr>
<tr>
<td>Duncan, 1984 (24)</td>
<td>Ethiopia, clinic at leprosarium. Prospective cohort study, up to 24 months postpartum</td>
<td>1975–1978 (Era of increasing Dapsone resistance)</td>
<td>79 in 76 women</td>
<td>BL/LL leprosy only</td>
</tr>
<tr>
<td>Lopes &amp; Sarno, 1994 (25)</td>
<td>Brazil. Cohort observation.</td>
<td>3 on Dapsone Rest on MDT</td>
<td>20 pregnancies in 20 women</td>
<td>8L, 7B, 4T, 1I (13 smear positive)</td>
</tr>
<tr>
<td>De Palacios, 2013 (26)</td>
<td>Brazil. Retrospective analysis of national data</td>
<td>2007–2009 (all cases on MDT)</td>
<td>149 pregnancies But for 36 women, no information concerning reaction.</td>
<td>Registered cases, 44% were MB</td>
</tr>
</tbody>
</table>
TABLE 1 Frequency of reactional episodes during and soon after pregnancy (cont’d)

<table>
<thead>
<tr>
<th>Study Reference</th>
<th>Number of Episodes of Reaction or Number of Women Affected</th>
<th>Pregnancy Outcome and Other Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RR</td>
<td>ENL</td>
</tr>
<tr>
<td>Maurus, 1978 (20)</td>
<td>No data</td>
<td>32% had ENL (69% HSP* cases, 12/38 of pregnancies in LL/BL cases)</td>
</tr>
<tr>
<td>Duncan, 1981, 1982A (21,22)</td>
<td>83 episodes in 40 women (24 were postpartum)</td>
<td>28 episodes (mostly in 1st &amp; 3rd trimester but also up to 6 months postpartum)</td>
</tr>
<tr>
<td>Duncan, 1982B (23)</td>
<td>No data</td>
<td>85 episodes of neuritis with improvement in 9/26 who received steroids</td>
</tr>
<tr>
<td>Duncan, 1984 (24)</td>
<td>No data</td>
<td>10/45 BL cases and 20/34 LL cases had ENL at some point.</td>
</tr>
<tr>
<td>Lopes &amp; Sarno, 1994 (25)</td>
<td>2 episodes</td>
<td>8 episodes</td>
</tr>
<tr>
<td>De Palacios, 2013 (26)</td>
<td>8 episodes in 113 women</td>
<td>3 episodes in 113 women</td>
</tr>
</tbody>
</table>

* HSP = highly smear positive; ** DHS = dapsone hypersensitivity syndrome
The widespread convention is to use the term “child case” for a person with leprosy who is under 15 years old at diagnosis (35). However, some authors use different age cutoffs, such as 18 years (reflecting the UNICEF definition) or 12 years. This inconsistency hinders comparison between countries/regions and between published studies.
EPIDEMIOLOGY

A reliance on operational data of variable quality, collected for management purposes, makes it difficult to know the true picture. By analogy with the disability grade 2 indicator recommended by the WHO (13), the new case detection rate could be expressed as new child cases of leprosy/100,000 child population or as number of child cases/100,000 total population, since the total number of children under 15 years old is often not known (36, 37). This approach is more informative than the traditional indicator for “child rate” (child proportion among new cases), which may be distorted by operational factors (different policies determining case finding methods).

Many publications describe case series/cohorts from referral centers (e.g., 38, 39), which may not be representative of the general population of child cases (Table 2A, 2B) (40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62). This issue is well illustrated by a comparison between data from a referral hospital and district data over the same period in Bijapur (Table 2C) (53). According to some studies, “age at onset” has changed over time. Thus, the epidemiological picture is unclear, as only age at diagnosis can be ascertained; however, age at diagnosis is influenced both by methods of case detection and by the unknown rate of self-healing in undiagnosed cases (63, 64). Although very young children may be more susceptible to infection, clinical manifestations of leprosy are rare in children under two years old, presumably because of the lengthy incubation period, especially for MB leprosy. In a recent large series of cases from Delhi (ranging from 2–81 years old), the peak age for PB (paucibacillary leprosy) cases was 10–29 years old (62% of the cases) and the mean age was 27.36, whereas for MB cases, the peak age was 20–39 years old (50% of the cases) and the mean age was 32.60 (a statistically significant difference) (65). (See Chapter 1.2 for a detailed discussion of the epidemiology of leprosy.)

CURRENT WORLD SITUATION

According to national program-derived WHO data (66), global changes over time in the number of child cases follow a similar pattern to that of total cases (Figure 4). Since 2005, the child proportion of cases has been about 9% worldwide, including in India, which has the largest number of child cases (Table 3, Figure 4). However, the huge variation observed between countries has no obvious relationship to other indicators, such as registered prevalence. Routinely collected global disability data does not include separate figures for children.

SCHOOL SURVEYS

The administration of school surveys was previously advocated for leprosy control work in vertical programs (67) and, in some places, they are still used (68). However, this labor-intensive activity is not cost effective in low prevalence situations (69). Cost-effectiveness might be enhanced where combination outreach programs are conducted, for example, in Brazil, where deworming programs have been combined with leprosy screening (70). Different ways of engaging schools to improve case detection (especially where enrollment is almost 100%) range from holding classes on
### TABLE 2A Childhood leprosy in India: recent studies from tertiary care centers

<table>
<thead>
<tr>
<th>Reference</th>
<th>Period</th>
<th>Location</th>
<th>Age Group (Years)</th>
<th># Child Cases</th>
<th>Child (%)</th>
<th>Prior Family Contact Identified (%)</th>
<th>MB (%)</th>
<th>Smear Positive (%)</th>
<th>Disabled (%)</th>
<th>Reaction Present (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sehgal 1993 (40)</td>
<td>81–91</td>
<td>Urban cl, ND</td>
<td>0–14</td>
<td>161</td>
<td>5.06</td>
<td>8.7</td>
<td>21.7</td>
<td>18</td>
<td>4.3</td>
<td>3.1^b</td>
</tr>
<tr>
<td>Prasad 1998 (41)</td>
<td>91–95</td>
<td>3^rd hosp, TN</td>
<td>0–14</td>
<td>66</td>
<td>7.2</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Selvasekar 1999 (42)</td>
<td>90–95</td>
<td>SLRTC, TN</td>
<td>0–14</td>
<td>794</td>
<td>31.3</td>
<td>30</td>
<td>2</td>
<td>na</td>
<td>0.5</td>
<td>4.03</td>
</tr>
<tr>
<td>Jain 2002 (43)</td>
<td>90–99</td>
<td>Urban cl, AP</td>
<td>0–14</td>
<td>306</td>
<td>9.81</td>
<td>na</td>
<td>9</td>
<td>0</td>
<td>29.7</td>
<td></td>
</tr>
<tr>
<td>Kar &amp; Job 2005 (44)</td>
<td>94–03</td>
<td>Karigiri, TN</td>
<td>0–15</td>
<td>275</td>
<td>4.5</td>
<td>13.4</td>
<td>8.4</td>
<td>10.5</td>
<td>20^c</td>
<td></td>
</tr>
<tr>
<td>Grover 2005 (45)</td>
<td>97–02</td>
<td>Urban cl, ND</td>
<td>0–14</td>
<td>137</td>
<td>7.06</td>
<td>22</td>
<td>29</td>
<td>23</td>
<td>24</td>
<td>2.0^d 10.9^e</td>
</tr>
<tr>
<td>John 2005 (46)</td>
<td>98–03</td>
<td>TLMH, WB</td>
<td>10–20</td>
<td>258</td>
<td>18.0</td>
<td>na</td>
<td>57</td>
<td>5</td>
<td>4.8</td>
<td>14.5</td>
</tr>
<tr>
<td>Sardana 2006 (47)</td>
<td>92–03</td>
<td>3^rd hosp ND</td>
<td>0–15</td>
<td>86</td>
<td>7.71</td>
<td>27</td>
<td>37</td>
<td>28</td>
<td>5.8</td>
<td>8.1 13</td>
</tr>
<tr>
<td>Vara 2006 (48)</td>
<td>99–02</td>
<td>3^rd hosp Guj</td>
<td>0–14</td>
<td>67</td>
<td>8.4</td>
<td>15</td>
<td>&gt;50%</td>
<td>46</td>
<td>10.4</td>
<td>0</td>
</tr>
<tr>
<td>Rao 2009 (49)</td>
<td>04–09</td>
<td>3^rd hosp AP</td>
<td>0–18</td>
<td>32</td>
<td>11.43</td>
<td>18</td>
<td>na</td>
<td>25</td>
<td>3.1</td>
<td>6.24</td>
</tr>
<tr>
<td>Horo 2010 (50)</td>
<td>04–06</td>
<td>TLMH, WB</td>
<td>0–15</td>
<td>151</td>
<td>na</td>
<td>1</td>
<td>33</td>
<td>30</td>
<td>16</td>
<td>11.25</td>
</tr>
</tbody>
</table>
### TABLE 2A Childhood leprosy in India: recent studies from tertiary care centers (cont’d)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Period</th>
<th>Location</th>
<th>Age Group (Years)</th>
<th># Child Cases</th>
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<th>Prior Family Contact Identified (%)</th>
<th>MB (%)</th>
<th>Smear Positive (%)</th>
<th>Disabled (%)</th>
<th>Reaction Present (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sachdeva 2010 (51)</td>
<td>00–09</td>
<td>3rd hosp UP</td>
<td>0–14</td>
<td>219</td>
<td>5.1</td>
<td>35</td>
<td>26</td>
<td>na</td>
<td>na</td>
<td>1.36</td>
</tr>
<tr>
<td>Singal 2011 (52)</td>
<td>00–09</td>
<td>3rd hosp ND</td>
<td>0–14</td>
<td>172</td>
<td>9.6</td>
<td>14.5</td>
<td>52</td>
<td>20</td>
<td>12.8</td>
<td>18.6&lt;sup&gt;f&lt;/sup&gt;</td>
</tr>
<tr>
<td>Palit 2014 (53)</td>
<td>06–13</td>
<td>3rd hosp Karn</td>
<td>0–18</td>
<td>61</td>
<td>19.7</td>
<td>18.2</td>
<td>61</td>
<td>8</td>
<td>5.8</td>
<td>8.2</td>
</tr>
<tr>
<td>Dogra 2014 (39)</td>
<td>01–11</td>
<td>3rd hosp, Chan</td>
<td>0–18</td>
<td>59</td>
<td>4.8</td>
<td>25</td>
<td>29</td>
<td>8</td>
<td>14.5</td>
<td>23</td>
</tr>
<tr>
<td>Kaur 1991 (54)</td>
<td>82–91</td>
<td>3rd hosp, Chan</td>
<td>0–19</td>
<td>132</td>
<td>12.4</td>
<td>20</td>
<td>29.5</td>
<td>17.4</td>
<td>6.8</td>
<td>5.3&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>Kumar 2000 (55)</td>
<td>90–99</td>
<td>3rd hosp, Chan</td>
<td>0–18</td>
<td>61</td>
<td>4.5</td>
<td>na</td>
<td>13.1</td>
<td>11.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>na</td>
<td>1.45&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sasidharanpillai 2014 (56)</td>
<td>03–12</td>
<td>Hosp. Kerala</td>
<td>0–15</td>
<td>138</td>
<td>12.1</td>
<td>12.3</td>
<td>26.8</td>
<td>3.6</td>
<td>5.8</td>
<td>1.45&lt;sup&gt;i&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sethi &amp; Rao 2015 (57)</td>
<td>09–12</td>
<td>TLMH, Delhi</td>
<td>0–15</td>
<td>94</td>
<td>na</td>
<td>3.2</td>
<td>na</td>
<td>14.9</td>
<td>9.6</td>
<td>24.5</td>
</tr>
<tr>
<td>Asia 2016 (58)</td>
<td>10–14</td>
<td>Med coll hosp, MS</td>
<td>0–15</td>
<td>86</td>
<td>13.1, range 8–34</td>
<td>21</td>
<td>29</td>
<td>Not given</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Range in hospital series</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>4.8–31.3</td>
<td>1–37</td>
<td>2–61</td>
<td>5–46</td>
<td>0.5–41</td>
<td>0–29.7</td>
</tr>
</tbody>
</table>

<sup>a</sup> AFB +ve on biopsy not on SSS; <sup>b</sup> at diagnosis or during MDT; <sup>c</sup> at diagnosis (54 cases RR, 1 case ENL); <sup>d</sup> at diagnosis; <sup>e</sup> during MDT; <sup>f</sup> during disease (29 cases RR, 3 cases ENL); <sup>g</sup> during disease (14 cases RR, 6 cases ENL); <sup>h</sup> 6 cases RR, 1 case ENL; <sup>i</sup> 2 cases RR, 0 cases ENL; <sup>j</sup> all 6 cases RR
### TABLE 2B Population based studies

<table>
<thead>
<tr>
<th>Reference</th>
<th>Period</th>
<th>Location</th>
<th>Age Group</th>
<th># Child Cases</th>
<th># Child Cases (%)</th>
<th>Prior Family Contact (%)</th>
<th>MB (%)</th>
<th>Smear +ve (%)</th>
<th>Disabled (%)</th>
<th>Reaction</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ekeke 2014 (59)</td>
<td>02–12</td>
<td>S Nigeria</td>
<td>0–14</td>
<td>64–110 pa</td>
<td>7.0</td>
<td>50.0</td>
<td>80.5</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>Langbin 2015 (60)</td>
<td>05–09</td>
<td>All China</td>
<td>0–15</td>
<td>191†</td>
<td>2.42</td>
<td>80</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>11</td>
<td>11%† National program data</td>
</tr>
<tr>
<td>Shetty 2013 (61)</td>
<td>2007</td>
<td>Maharashtra State, India</td>
<td>0–14</td>
<td>68</td>
<td>34.1</td>
<td>36</td>
<td>25</td>
<td>7.3</td>
<td>4.4</td>
<td>8.8</td>
<td>13.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>MS-rural</td>
<td>0–14</td>
<td>32</td>
<td>35.5</td>
<td>47</td>
<td>34</td>
<td>6</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>MS-urban</td>
<td>0–14</td>
<td>36</td>
<td>33.0</td>
<td>19</td>
<td>19</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Then Lwin Tun 2014 (62)</td>
<td>2012</td>
<td>Myanmar</td>
<td>0–15</td>
<td>155</td>
<td>5.14</td>
<td>49b</td>
<td>63.3b</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>Arpana Patil 2014 (53)</td>
<td>06–13</td>
<td>Community LCP (Dt)</td>
<td>0–18</td>
<td>223</td>
<td>21.25</td>
<td>na</td>
<td>30.5</td>
<td>na</td>
<td>na</td>
<td>5.82</td>
<td>4.93% 11 RR, 0 ENL National program data</td>
</tr>
</tbody>
</table>

† 165 analysed; ‡ disabled children; † at diagnosis
the early signs of leprosy for pupils or staff to involving schoolchildren in the active examination of family members (71, 72). School populations have also been studied to assess the prevalence of sub-clinical leprosy, as indicated by immunological parameters (73) and PCR studies.

**CONTACT SURVEILLANCE**

Contact tracing is probably the case detection method with the best evidence base (74). Residents in the same household as an index case have a higher risk of developing leprosy (75), and this correlation holds true for children. Most national programs, following WHO advice (15, 35), recommend examining all household contacts at least once soon after the detection of a new case. Regarding children, this recommendation fulfills two purposes: (i) when the index case is a child, to identify the source of infection (and thus to protect other children in the household) and (ii) when the index case is an adult, to identify any secondary cases among children early. Household contact examination will include pre-school-age children and usually includes an element of health education to encourage prompt voluntary reporting of suggestive signs subsequently. Innovative approaches to contact surveillance include using family motivation techniques (76). The proportion of new child cases with a known prior case in the household varies widely in different publications (Table 2A). Household contact examinations are vital when post-exposure prophylaxis (PEP) is considered (see below), as it is essential to exclude overt cases from PEP.

### TABLE 2C Comparison of referral center data with community data, in same area, over same period

<table>
<thead>
<tr>
<th>Source</th>
<th>Community Clinics</th>
<th>Hospital Series</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Cases</strong></td>
<td>1049</td>
<td>309</td>
</tr>
<tr>
<td><strong>Child Cases (#, %)</strong></td>
<td>223 (21.25%)</td>
<td>61 (19.7%)</td>
</tr>
<tr>
<td><strong>Sex (#, %)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>134 (60%)</td>
<td>29 (47.5%)</td>
</tr>
<tr>
<td>Female</td>
<td>89 (40%)</td>
<td>32 (52.5%)</td>
</tr>
<tr>
<td><strong>Age Group (years)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-5</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>6-15</td>
<td>164</td>
<td>39</td>
</tr>
<tr>
<td>16-18</td>
<td>56</td>
<td>20</td>
</tr>
<tr>
<td><strong>Treatment Group (#, %)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PB</td>
<td>155 (69.5%)</td>
<td>24 (39.34%)</td>
</tr>
<tr>
<td>MB</td>
<td>68 (30.49%)</td>
<td>37 (60.65%)</td>
</tr>
<tr>
<td><strong>Reaction (#,%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>11 (4.93%)</td>
<td>5 (8.19%)</td>
</tr>
<tr>
<td>Type 2</td>
<td>0</td>
<td>5 (8.19%)</td>
</tr>
<tr>
<td><strong>Disability (#, %)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>13 (5.82%)</td>
<td>5 (8.19%)</td>
</tr>
</tbody>
</table>

Source: Palit 2014 (53), Bijapur, India
The distribution across the Ridley-Jopling spectrum (see Chapter 2.4) for children differs from that for adults, reflecting both differences in the methods of case detection and the longer incubation period for the BL/LL forms of leprosy as compared with other forms. In general, the proportion of child cases classified as Indeterminate is higher, as is the proportion classified as PB (Tables 2A, 2B) (36, 52, 57, 62).

Parents may voluntarily present children with skin lesions, or less often with impairments, to health services. If health workers are alert to the possibility of leprosy, such cases are promptly referred to a leprosy specialist for confirmation. Where parents first present their children to traditional healers or unqualified practitioners, diagnosis may be delayed. Very early case detection (as a result of efficient regular household contact surveillance) can lead to the treatment of children whose disease would have self-healed spontaneously, though it remains impossible to distinguish reliably which cases would regress if left untreated. If health workers who see children for routine health care, including immunizations, practiced passive surveillance (noticing leprosy

### TABLE 3 Child cases in countries with larger leprosy load, 2015

<table>
<thead>
<tr>
<th>Country</th>
<th>All new cases</th>
<th>Child cases</th>
<th>Child proportion (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bangladesh</td>
<td>3,976</td>
<td>327</td>
<td>8.22</td>
</tr>
<tr>
<td>Brazil</td>
<td>26,395</td>
<td>1,942</td>
<td>7.35</td>
</tr>
<tr>
<td>Congo</td>
<td>4,237</td>
<td>486</td>
<td>11.47</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>3,970</td>
<td>583</td>
<td>14.68</td>
</tr>
<tr>
<td>India</td>
<td>127,326</td>
<td>11,389</td>
<td>8.94</td>
</tr>
<tr>
<td>Indonesia</td>
<td>17,202</td>
<td>1,930</td>
<td>11.22</td>
</tr>
<tr>
<td>Madagascar</td>
<td>1,487</td>
<td>151</td>
<td>10.15</td>
</tr>
<tr>
<td>Myanmar</td>
<td>2,571</td>
<td>101</td>
<td>3.93</td>
</tr>
<tr>
<td>Nepal</td>
<td>1,751</td>
<td>197</td>
<td>11.25</td>
</tr>
<tr>
<td>Nigeria</td>
<td>2,892</td>
<td>264</td>
<td>9.12</td>
</tr>
<tr>
<td>Philippines</td>
<td>1,617</td>
<td>131</td>
<td>8.10</td>
</tr>
<tr>
<td>Sri Lanka</td>
<td>1,977</td>
<td>223</td>
<td>11.28</td>
</tr>
<tr>
<td>Mozambique</td>
<td>1,335</td>
<td>116</td>
<td>8.69</td>
</tr>
<tr>
<td>Tanzania</td>
<td>2,256</td>
<td>87</td>
<td>3.86</td>
</tr>
<tr>
<td>Total</td>
<td>199,992</td>
<td>17,927</td>
<td>8.96 (94.9% global total)</td>
</tr>
<tr>
<td></td>
<td>(95.4% of global total)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global total</td>
<td>210,758</td>
<td>18,796</td>
<td>8.92 (range 3.86–14.68)</td>
</tr>
</tbody>
</table>

This data refers to new cases as true incidence rates cannot be reported by national programs.


**DIAGNOSIS IN CHILDREN**

The distribution across the Ridley-Jopling spectrum (see Chapter 2.4) for children differs from that for adults, reflecting both differences in the methods of case detection and the longer incubation period for the BL/LL forms of leprosy as compared with other forms. In general, the proportion of child cases classified as Indeterminate is higher, as is the proportion classified as PB (Tables 2A, 2B) (36, 52, 57, 62).

Parents may voluntarily present children with skin lesions, or less often with impairments, to health services. If health workers are alert to the possibility of leprosy, such cases are promptly referred to a leprosy specialist for confirmation. Where parents first present their children to traditional healers or unqualified practitioners, diagnosis may be delayed. Very early case detection (as a result of efficient regular household contact surveillance) can lead to the treatment of children whose disease would have self-healed spontaneously, though it remains impossible to distinguish reliably which cases would regress if left untreated. If health workers who see children for routine health care, including immunizations, practiced passive surveillance (noticing leprosy
lesions during consultations for other conditions), more child cases would be detected before impairment occurs.

### DIAGNOSTIC PROBLEMS

Dermatological differential diagnosis (see Chapter 2.3) in children is similar to adults, and the same diagnostic criteria (see Chapter 2.1) apply. However, the examination of patches should be adapted for the child’s age and understanding (Box 3). Much patience and a calm atmosphere is essential, but, even in children as young as 12 months, patch sensation can be tested by observing the child’s reflex responses to an unseen touch. Sometimes, if shown how to test sensation, a parent will more easily gain the child’s cooperation. An observation period (with no medication and a planned review at 1–2 months) is usually safe in uncertain cases and preferable to a biopsy. In such cases, a detailed description, including the measurements of any skin lesions, must be

---

**FIG 4** Graph of Child Rates Around the World in the Recent Decade.
recorded for comparison with the signs at a later date (36). Considering the seriousness of the diagnosis in a child and the practical difficulties inherent in confirming it, every new case (and preferably every child suspect) should be examined by a qualified medical practitioner experienced with leprosy.

**BOX 3 Quotation about diagnosis**

“The demonstration of sensory impairment can be undertaken in children as young as three or four years, provided that their confidence is gained and that they know what you want. Take a minute or two to become friends; sit him on your knee, play with a feather or a blade of grass and tickle him. When he is chuckling, get him to point with the index finger to the place you touched. Then play the same game with the eyes shut.

Impairment of sweating function is usually simple to recognise in field work in the tropics, when a short romp together in the sun induces sweating in healthy skin.”

Foreword by Stanley Browne, in Leprosy in Children, Noussitou (66)

**NERVE FUNCTION ASSESSMENTS**

Despite the difficulties, conducting and recording a full assessment of nerve function in every new case is necessary. According to the child’s risk level, a plan for future monitoring can be made. Some children present suspected consequences of neurological impairment, such as trophic ulceration. NFI *per se* is not an adequate ground for the diagnosis of leprosy, since spina bifida, congenital indifference to pain, and inherited or other acquired peripheral neuropathies should also be considered. (See Chapter 2.5 for a detailed discussion of the neurological manifestations of leprosy.)

**ROLE OF SKIN SMEARS AND BIOPSIES**

Slit-skin smears are highly desirable (though not necessarily at a first visit) for children with the suggestive signs/symptoms of leprosy, where simple clinical examination of the skin and nerves does not confirm the diagnosis. This approach is especially desirable in children with suspected MB disease (e.g., presenting with ENL-like lesions or “hazy non-anaesthetic hypopigmented patches”) (67). A slit-skin smear can easily be collected locally by a clinician or laboratory technician, then sent to a specialist center for staining and reporting. A positive smear confirming leprosy obviates the need for a biopsy. A skin biopsy for histology is traumatic for a young child and only exceptionally justified in cases of diagnostic difficulty in which neither an observation period nor a second opinion resolves the uncertainty. In early indeterminate leprosy, the histological findings are often as non-specific as the clinical features. Several case series in child suspects demonstrate that a biopsy rarely clarifies the diagnosis (62, 77, 78). (See Chapter 2.4 for a detailed discussion of the pathology of leprosy.)
Chemotherapy

EDUCATION OF PARENTS AND CHILDREN

Parents should be made aware of the diagnosis of leprosy as soon as it is confirmed, and the process of educating them about the disease and its management begun early, to ensure maximum support and engagement (Box 4) (79). The task of informing children needs a sensitive, age-appropriate approach. Younger children may learn better from their parents rather than directly from health workers, while adolescents may resent being excluded from conversations about their health (46, 80).

BOX 4 Parental education before starting MDT for child

Parents and young patients need to understand the following:

- MDT rapidly renders a case non-infectious, so there is no need for segregation.
- Isolating the child and his/her things is unnecessary; normal domestic hygiene suffices.
- How to recognize the common and serious adverse effects of MDT and what to do
- Which effects to anticipate (i.e., do not expect a rapid disappearance of skin lesions)
- Where and when MDT is available
- What costs are associated with treatment
- Need for safe storage of the medication at home
- Leprosy reactions may occur despite taking MDT correctly.
- How to recognize leprosy reactions and what to do

STANDARD MDT

The criteria for classifying cases as PB/MB are the same as those for adults (see Chapter 2.1) and so are the basic WHO regimens (see Chapter 2.6), that is: rifampin and dapsone for PB for 6 months; rifampin, dapsone, and clofazimine for MB for 12 months minimum (Box 5). In endemic countries, medication will usually be provided in standard blister calendar packs (BCP) as supplied through the WHO, and health workers will frequently encounter problems of dosages for small children. Dosages should be decided by body weight, not purely by age. With only BCPs available, it is difficult to dispense correct doses for children who weigh less than 15 kg (Box 5) (81). (See Chapter 2.6 for detailed information on the treatment of leprosy.)
ALTERNATIVE REGIMENS

Children with dapsone hypersensitivity syndrome (see Chapter 2.6) are usually offered a modified WHO MDT regimen consisting of rifampin monthly and clofazimine daily at the usual doses and same duration. In children with “single skin lesion leprosy” (smear negative), the single dose rifampin, ofloxacin, and minocycline combination (ROM) (82) has been used. However, ROM is...
Reactions and Neuritis

INCIDENCE OF REACTIONS IN CHILDREN

Overall, both reversal and ENL reactions appear to be less common in children than in adults (85) (possibly because of the smaller proportion of MB cases), but there is a paucity of data on incidence rates or the timing of reactions in children. Many published case series mention the “number of reaction cases” (or percentage), but do not distinguish between reactions occurring (i) before diagnosis, (ii) during chemotherapy, (iii) after completing MDT, or (iv) in children referred for reaction after being diagnosed and starting MDT elsewhere (Table 2A). Detailed observational studies of cohorts that are followed for 2–3 years are essential to understand whether risk factors for children are similar to those for adults and to make it possible to estimate the probability of reaction (and neuritis) in any individual case.

MONITORING FOR NEW NFI

Monitoring for nerve function impairment (NFI) should be planned at the first visit to ensure that any new primary impairments will be detected early, when there is a greater chance of reversal with treatment, and before the onset of secondary impairments. Since there is little evidence on the outcomes of new NFI in children, deciding whether to prescribe steroids in a particular situation entails balancing individual risks and benefits. In the absence of better evidence, the clinical prediction rule (27), which has not been validated specifically for children, can be utilized in determining the frequency of formal assessments in clinic (Box 6). Monitoring might include home testing, by the parent or the child, to ensure the prompt recognition (and management) of any new impairment. (See Chapter 2.5 for a discussion of the neurological manifestations of leprosy.)

NERVE ABSCESSES

Nerve abscesses (due to intense immune reactions causing tissue damage) may occur in children (86). They need urgent incision and drainage by a trained surgeon. General anesthesia is preferable if the patient is a young child.
BOX 6 Clinical prediction rule for probability of new nerve function impairment

Risk of New NFI Over the First Two Years From Diagnosis

<table>
<thead>
<tr>
<th></th>
<th>PB cases</th>
<th>MB cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>No nerve function impairment at diagnosis</td>
<td>1% (Low risk)</td>
<td>16% (Moderate risk)</td>
</tr>
<tr>
<td>With nerve function impairment at diagnosis</td>
<td>16% (Moderate risk)</td>
<td>67% (Moderate risk)</td>
</tr>
</tbody>
</table>

Proposed Use of the Prediction Rule To Prioritize the Use of Resources

After initial nerve function assessment (NFA):

- Those at lowest risk could be educated about reporting promptly if any deficit is noticed, then re-assessed every 6 months for two years.
- Those at moderate risk should be seen for an NFA every three months, for at least 24 months.
- Those at highest risk should be seen for an NFA monthly for 12 months, then quarterly for up to 24 months.

USE OF CORTICOSTEROIDS IN CHILDREN

In view of the lack of evidence on steroid regimens for children, the usual practice is to prescribe based on recommended adult regimens (see Chapter 2.2) modified for the child’s weight (and age) (Box 7). The special risks of steroids in children (effects on skeletal growth and puberty) must be borne in mind in addition to general adverse effects, including immunosuppression, hyperglycemia, osteoporosis, and adrenal suppression. Monitoring weekly for adverse effects and for response is desirable. Pediatric guidance should be sought if the child needs more than three months of treatment or if doses above 1 mg/kg are considered (87). Alternate day regimens are sometimes recommended to reduce adrenal suppression. Parents should be warned about the risk of adrenal crisis following sudden cessation and also about the risks of administering unprescribed doses of steroids to their children. Medicines should be stored safely out of the reach of young children. Anti-helminthics, especially those targeting strongyloides infection, should be given at the start of a steroid course, when helminth infections are common.

OTHER DRUGS FOR REACTIONAL EPISODES

“High dose clofazimine” for chronic ENL can be used in children (16), who often tolerate the color change well. But the family should be aware of the risk of acute abdominal pain associated with an overload of clofazimine. Since little is known about the pharmacokinetics of clofazimine (see
BOX 7 Use of steroids in children

As an example,

For a child of about 30-35 kg body weight, having silent neuritis with definite new NFI and no specific contra-indications to steroids, give a 12-week course beginning at 30mg prednisolone per day and tapering rapidly to 20mg, then more slowly.

i.e. 30mg once daily x 7 days,
25mg once daily x 7 days,
then 20mg once daily x 28 days,
then 15mg once daily x 14 days,
then 10mg once daily x 14 days,
then 5mg once daily x 14 days.

Chapter 2.6), and accumulation depends on the quantity of subcutaneous fat, choosing the regimen is a matter of judgment for an experienced clinician. The daily dose should be divided, given with food to reduce gastrointestinal effects, and withheld for a few days in the event of acute gastroenteritis (Box 8).

BOX 8 Using high dose clofazimine in children

As a rough guide, 1.5–2mg/kg three times daily for one month, then reducing by one dose per day each month. Maximum: 300mg daily.

For example, for a child of 25 to 35 kg, a suitable regimen is 50 mg three times daily for month 1, 50 mg twice daily for month 2, then 50 mg once daily for month 3.

For comparison: adults of 50–70 kg usually tolerate a total dose of 18 grams over 3 months equivalent to 100 mg twice daily for 90 days (or 100 mg three times per day x 30 days, 100 mg twice a day x 30 days, 100 mg once a day x 30 days).

Divided doses are better tolerated.

In general, the expected morbidity and mortality of leprosy reaction would not justify the risks of immunosuppressants in children, but in some individual cases—where facilities and expertise permit—supervised, off-label use of drugs such as methotrexate (as steroid-sparing agents) might be considered. Nevertheless, there is scant trial data to support the use of methotrexate even for adults, and azathioprine has not been demonstrated to be effective. Thalidomide is not recommended for children below 12 years old, due to the lack of safety information, but it might be used—if legally available—in adolescent boys. (See Chapter 2.2 for a discussion of reactions.)
Puberty

As with pregnancy, the immunological disturbances of puberty might increase the risk of relapse or reactivation of inadequately treated leprosy. Sasidharanpillai (56) identified 10/138 children registered for MDT who were diagnosed as relapse cases over 10 years. Eight (8) who had received PB MDT and were RFT (released from treatment) only 3 months to 2 years before, might have been originally incorrectly classified. Of the total group in this age group, 8/10 were 13–15 years old (cf 34.8%) and 8/10 were girls (cf 38% of all 138, p=0.004).

Some experts have an impression there is an apparent increase in the incidence and severity of reactions around the time of puberty (88), though we are not aware of any epidemiological evidence to confirm an increased risk of reaction during puberty. There are particular problems with long-term use of steroids in this age group because of the effects on the onset of puberty and interruption of growth (87). When no alternative therapy is available for chronic reaction, the priority is usually to keep the steroid dose as low as possible, consistent with control of reactional episodes. An uncontrolled reaction has a huge impact on a child’s quality of life and future prospects through long admissions (missing education at a critical stage), onset of new disability, and pain suffered. A pediatrician should be consulted in complex cases. There is an urgent need for evidence-based clinical management guidelines and for multicenter RCTs and cohort studies to establish which steroid regimes are the safest and most effective for this age group.

MANAGING DISABILITY IN CHILDREN

While the goals and principles of disability management are the same for children as they are for adults (see Chapter 4.3), a modified approach is needed for a child with a physical impairment, who is especially at risk because of his or her dependence on a variety of adults. Because children are less able to weigh future consequences and adolescents often have a false sense of invulnerability, it is difficult to motivate children to consistently undertake self-care of neurologically impaired limbs. Age-appropriate educational materials are needed for self-care training. Protective footwear (including something suitable for play) should be regularly replaced as the child’s feet grow. When trophic ulcers occur, off-loading solutions such as the use of POP (a double rocker shoe/BK cast with Bohler iron) are preferable to bed rest. Adequate nutrition for wound healing is particularly important in a growing child. Reconstructive surgery (see Chapter 4.2) should not be denied on the grounds of age alone (Figure 5) (89). Clinicians may need to advise schools about special needs, depending on the specific impairments present (e.g., extra time for examination writing, avoidance of some sports, provision of low vision aids). No one doubts that developing an irreversible disability is a tragedy for a child (88), but the progression from impairment to secondary disability after diagnosis can be halted. In addition, clinicians should audit the long-term outcomes of their management (58, 61).
PSYCHOLOGICAL IMPACT OF LEPROSY

The psychological effects of physical impairment (60, 61, 90), including the risk of bullying or social isolation, on a child’s life need attention. Very little research has been conducted in this area on leprosy-affected children specifically, but findings from studies of other chronic diseases in children or leprosy in adults may be applicable. Enhancing the self-efficacy of a patient is as important as imparting information (91, 92). Teenagers may also have different opinions about the care needed from their parents (80), and both parents and children may need guidance regarding future career plans.

FIG 5 Pre- and post-operative photos of Nepali child.

“Healthy” children of disabled leprosy-affected parents and siblings of children with leprosy may experience adjustment problems, deprivation, or impaired mental health (Figure 6). The research evidence is sparse, but clinicians dealing with “leprosy-affected families” need to be alert to such issues as the welfare of “young carers” on whom disabled adults depend for the activities of daily living. (See Chapter 4.6 for a detailed discussion of the stigma of leprosy.)

SERVICES FOR CHILDREN

In clinics, efforts should be made to cater for children’s special needs, e.g., by limiting waiting times, making child-friendly spaces, and protecting the children from more unpleasant sights. When in-patient care is unavoidable, admission to a pediatric ward is preferable (67) for age-appropriate nursing care and a child-friendly environment. Complicated cases should preferably be seen by a pediatrician or family medicine specialist.

Inclusive education should be the norm and absences from school for health care minimized (Figure 7). Any necessary aids including protective footwear should ideally follow designs approved by children. Finally, educational materials about leprosy should feature language and illustrations attractive to children.
FIG 6 Father with hands damaged by leprosy takes his child to school.

FIG 7 Child goes to school unembarrassed by the leprosy patch on his face.
PREVENTION

A hygienic and uncrowded environment at home and at school will reduce a child’s risk of being infected by *M. leprae* (4). Resistance to an *M. leprae* infection is enhanced by *Bacillus Calmette–Guérin* (BCG) vaccination (93) (see Chapter 6.4), probably for a limited period, and children immunized with BCG are more likely to have PB rather than MB leprosy. Prompt treatment of infectious cases to which the child might be exposed and contact examinations to ensure early diagnosis before the disease is advanced (74) are of great importance. In children, as in adults, delayed diagnosis is associated with more disability (47, 64, 94). For each late diagnosis of leprosy in children, a review of all recent health services contacts is recommended to identify failures in case detection (81). Although it cannot yet be identified as such, many children have a subclinical infection (95) and might be “cured” before the development of the clinical disease by a single dose rifampin, although this possibility has not been confirmed by evidence. Post-Exposure Prophylaxis (PEP) with anti-mycobacterial drugs is being studied for contacts of newly diagnosed cases but is not routinely available and may be less effective in children of infected adults, since this highest-risk group had the least benefit in one major study (96, 97). As children under five were excluded from that study, evidence for them is lacking. A booster dose of BCG for contacts is routinely offered in Brazil (98). The effectiveness of combined chemo-immunoprophylaxis is currently being investigated (99, 100) in children as well as adults.

References


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