IN-15 Rabies, leprosy, HIV, hepatitis and the incurable: what can be done honestly

HEALING after the FALL, research library, IN-15

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How to read this report

Four diseases are gathered here because they share one property: a village of 300 people cannot cure any of them, and in three of the four it can still change the outcome a great deal. The report does not repeat two documents already in the library. HY-10 covers the rabies vaccine stockpile, the intradermal dose-sparing schedule, the cold chain and the record of nerve-tissue vaccine manufacture, and concludes that no village should attempt to make rabies vaccine (THRIVING library, HY-10). WO-13 covers the bite itself, the fifteen-minute soap wash, the ten-day observation of a biting dog and the per-site transmission risks (THRIVING library, WO-13). I agree with both and summarise rather than restate them. What I add on rabies is the clinical course, the Milwaukee protocol and its failures, the dying patient's care, and dog control as a village institution. Leprosy, HIV and the hepatitides are new ground.

Three voices are kept separate throughout: what a tradition claims, what the clinical record shows happened, and what modern science infers about mechanism. My own inferences carry [VERIFY]. Ethical choices are marked [OWNER] and left to the author.

Findings by section

1. What "incurable" means after the Fall, and where the leverage actually is

For a village these four diseases divide along a line that is not the line a hospital would draw. Rabies is untreatable once symptoms begin but almost entirely preventable in the first hour after a bite, with soap. Leprosy is curable only with drugs the village cannot make, but its disability is caused by injury to numb limbs and is preventable with instruction, inspection and footwear. HIV is untreatable without antiretrovirals, but its commonest killers in a poor setting are bacterial infections, diarrhoea, malaria and tuberculosis, and one cheap antibiotic cuts deaths by about a third. Hepatitis B is unpreventable after infection but almost wholly preventable at birth and at the needle. Hepatitis A and E are water problems, not liver problems.

So the leverage in every case is upstream of the sickbed: soap, a confined dog, a clean needle, a protected well, a pair of shoes, a daily tablet while it lasts, and a register that says who in the village is at risk. Downstream, where the hospital would act, the village has nursing, food, company and the courage to say a true prognosis. That is not nothing. Several of these diseases killed people at home for most of human history, and the record of how that was done well, and done badly, is the most useful material in this report.

2. Rabies: what happens in the body, and over what hours

Incubation is usually 20 to 60 days after the bite (Warrell, Warrell and Tarantola 2017), with the wider range of one week to a year and a usual two to three months given by WHO (THRIVING library, WO-13). Roughly 80 per cent of human cases take the encephalitic or "furious" form and about 20 per cent the paralytic form. A prodrome of two to ten days brings fever, malaise and, characteristically, tingling, itching or pain at the healed bite site. That symptom at an old bite is the single most useful diagnostic sign a lay healer will ever have for rabies, and it should be taught by itself.

The acute neurological phase then runs about a week. In furious rabies there are periods of agitation, confusion and hallucination alternating with lucid intervals, and the spasms of hydrophobia and aerophobia: an attempt to drink, or a draught of air on the face, triggers violent contraction of the diaphragm and accessory muscles with terror. Many patients die of respiratory or cardiac arrest during a spasm (Warrell 2017). In paralytic rabies, weakness begins in the bitten limb and ascends, hydrophobia is often absent, and untreated patients may survive up to 30 days. Combining the phases gives roughly nine to seventeen days from first symptom to death without intensive care, which is my arithmetic from the published ranges rather than a figure any one source states [VERIFY].

Case fatality once symptoms begin is essentially 100 per cent. The documented recoveries are three or four people in the whole world: two United States children bitten by bats, one of whom had been vaccinated, and one incompletely vaccinated man bitten by a dog in Turkey (Warrell 2017). Warrell and colleagues conclude that intensive care should be considered only for patients who were previously vaccinated, who develop rabies antibody within the first week of illness, or who were infected by an American bat virus. A village meets none of those conditions and has no intensive care. The honest statement to a family is that the disease is certain to kill, and that everything the healer does from here is for comfort.

One fear deserves correcting. There is no laboratory-confirmed case of human-to-human rabies transmission among household contacts, and transmission from patients to health-care staff has not been documented (CDC 2004, in Warrell 2017). Every confirmed person-to-person transmission has been through transplanted tissue: eight corneal recipients in five countries by a 2004 count, plus a cluster of solid-organ recipients in Arkansas in 2004 in which three of four died (CDC 2004). Families can therefore nurse a dying rabies patient. They should wear gloves if gloves exist, avoid saliva on broken skin and mucous membranes, and wash utensils with soap, but they do not need to abandon the bedside.

3. Rabies: the Milwaukee protocol, and why it must not be attempted

What the claim was. In September 2004 Jeanna Giese, aged fifteen, was bitten by a bat in Wisconsin and sought care only after symptoms began about a month later. She was treated at the Medical College of Wisconsin with induced coma plus ribavirin and amantadine, on the theory that sedation would protect the brain while antibody developed, and she survived. Willoughby and colleagues published the case in the New England Journal of Medicine on 16 June 2005, and it became the first documented survival of rabies in an unvaccinated person.

What the record shows. The approach has not replicated. By the time of the early reports it had been attempted in about ten people with no further success. A critical appraisal in the Canadian Journal of Neurological Sciences in 2016 reviewed each component and found that the current literature fails to support an important role for excitotoxicity and cerebral vasospasm in rabies encephalitis, and that none of the therapies, therapeutic coma, ketamine, amantadine or vasospasm management, can be substantiated in rabies or in other acute viral encephalitis. The brief attributes this paper to Zeiler and Jackson, and secondary sources agree, but I could not open the article itself to confirm the author list [VERIFY]. A 2022 preprint by Arslan and Vahaboglu found twelve cases managed with induced coma published after 2014, of which one survived, and noted that the 2015 warning against inducing coma has been ignored (search summary of a non-peer-reviewed preprint). A 2026 review concluded that current evidence does not conclusively support the protocol as an effective, reproducible curative treatment, and that mortality has not been

convincingly reduced beyond what intensive supportive care achieves alone (Quality in Sport 2026). A commentary in Clinical Infectious Diseases argues that the time has come to abandon it. WO-13 gives the failure counts as at least 31 by 2015 and at least 64 by 2025 (THRIVING library, WO-13).

What the village should conclude. The protocol's ingredients are a ventilator, continuous infusions, invasive monitoring and weeks of intensive nursing. None exists after the Fall. Even if they did, the evidence does not support the attempt. Grade **X** for any post-Fall version. The romance of the protocol is itself a hazard, because the energy a district would spend on heroic sedation is energy taken from washing bites and confining dogs, which actually work. Note that the brief's phrase "failed replications" is slightly generous: these were not replications of an experiment but a series of uncontrolled attempts, almost all fatal, and the original case may have involved an unusually attenuated bat virus or an unusually early antibody response.

4. Rabies: the sedated, comforted death

This is the part of rabies care a village can actually do well, and the literature is thin because the disease is rare where journals are written. The best single document is Warrell, Warrell and Tarantola 2017 in Tropical Medicine and Infectious Disease, which I read in full and which exists precisely to tell clinicians in poor settings how to palliate rabies. Its governing sentence is that in the absence of any proven effective antiviral or other treatment, palliative care is an imperative to minimise suffering.

The room. Calm, dimly lit, draught-free, a single room if one can be made. The spasms of hydrophobia and aerophobia are triggered by the sound or sight of water, a draught on the face, bright light and loud noise, so the room is arranged to exclude all four. Relatives should be able to be present in safety and privacy according to their own traditions, with other visitors limited.

Thirst. This is the cruelty of the disease: the patient is desperately thirsty and cannot drink. Warrell's team recommend fluid by a route that avoids the swallowing trigger, intravenous where possible, otherwise intraperitoneal, intraosseous, subcutaneous or intrarectal. A village realistically has subcutaneous and rectal routes. Lips are moistened with a damp sponge. Some patients can suck citrus fruit or eat bananas.

Sedation. Diazepam is the drug a salvaged store is most likely to hold. Adult doses given are 10 mg slow intravenous over three to five minutes repeated one-hourly to four-hourly, 20 mg intramuscular two-hourly, or 10 mg intrarectally one-hourly to four-hourly. Children take 0.1 to 0.3 mg/kg by any of those routes, repeated one-hourly to four-hourly. Midazolam, if present, is given 0.08 to 0.2 mg/kg intramuscularly repeated one-hourly to four-hourly, or 2.5 mg hourly intravenously or subcutaneously, or 10 to 30 mg over 24 hours by subcutaneous infusion. Haloperidol 5 mg hourly until calm, then four-hourly or six-hourly, or chlorpromazine 25 to 50 mg six-hourly to eight-hourly deep intramuscular, help with agitation. A study of 45 patients in Manila found that haloperidol or diazepam relieved anxiety and restlessness, and that combining them helped aggression, disorientation and hallucination (cited in Warrell 2017). Morphine 10 mg four-hourly, or 15 to 30 mg rectally four-hourly, is for pain, not for sedation. Hyoscine hydrobromide 400 micrograms four-hourly subcutaneously dries the drowning secretions. Paracetamol 1 g four-hourly to six-hourly, maximum 4 g in 24 hours, by rectum, is for fever, because fanning and sponging are often intolerable.

The judgement. Warrell's team make a point a village needs: diazepam given slowly, or rectally, can ease suffering and give a family time to decide whether to take the patient home to die. They also note that if the family wish to take the patient home alive for funeral rites, respiratory failure should be avoided, which means sedating to calm rather than to apnoea. The dose that ends the spasms and the dose that stops breathing are not far apart in a dehydrated, exhausted patient. Deliberately crossing that line is euthanasia, and whether a healer may do it is [OWNER].

If there are no drugs at all. Then the levers are the dark quiet room, no draught, no water in sight or sound, a trusted person holding the patient, padding to prevent injury during spasms, moistened lips, and alcohol by mouth

in the lucid intervals if the patient can swallow it, which many can early on. This is Grade **D**, traditional and plausible, with no trial behind it. It is what people did for centuries and it is better than restraint and fear.

5. Rabies: the village measure, which is dogs

Mass dog vaccination is the most cost-effective way to prevent human rabies and the target coverage is about 70 per cent of the dog population (WHO rabies fact sheet, Coleman and Dye 1996, both via THRIVING library, WO-13). The clearest demonstration is Pemba Island, Tanzania, where island-wide coverage rose from 16.8 per cent in 2011 to 68.2 per cent in 2014, detected cases fell from 42 before the campaigns to 2 in 2014, and no cases were detected after May 2014 until re-introductions in 2016 produced an outbreak that was again eliminated by October 2018 with reinstated vaccination. Modelling for Serengeti and Ngorongoro districts found that an annual campaign reaching 70 per cent would control rabies in both with high certainty. The repeated lesson across Tanzanian studies is that uneven coverage between villages wastes the whole effort, and that cases rise again within a year or two of stopping.

After the Fall there will be no canine vaccine for long. What then survives of this finding is the shape of the policy rather than the vaccine. The village keeps a dog register, limits the number of dogs to those that are owned and fed, ties up or confines any dog that bites unprovoked or changes behaviour, and kills dogs showing rabies signs. Whether culling alone controls rabies is not established, and WO-13 marks it [VERIFY]. I agree. The honest claim is that fewer free-roaming unowned dogs means fewer bites, which is worth doing on bite numbers alone even if it does not interrupt transmission (Grade **C** for culling as rabies control, **B** for reducing bite incidence [VERIFY]).

The ten-day confinement of a biting dog remains the single best decision tool a village has, because virus appears in saliva only shortly before the animal falls ill. It is covered in WO-13 and I add only this: the confinement must be real. A dog loose "in the yard" that then disappears converts a resolvable question into an unresolvable one, and in the absence of vaccine that difference is the family's entire prognosis.

6. Leprosy: what the disease is and what actually disables

Mycobacterium leprae was identified by Gerhard Armauer Hansen in Bergen in 1873, the first bacterium shown to cause human disease. The organism grows very slowly, prefers cool tissue, and attacks peripheral nerves.

Diagnosis. Three cardinal signs, any one of which is sufficient: a hypopigmented or reddish skin patch with definite loss of sensation, a thickened or tender peripheral nerve with loss of sensation or weakness in the muscles it serves, and acid-fast bacilli in a slit-skin smear. Sensation is tested with a wisp of cotton wool while the patient's eyes are closed. Patches may also be dry, because the autonomic nerves to sweat glands fail. Where the first two signs are absent but suspicion is strong, smears are taken from both earlobes and one lesion. Classification for treatment is operational: up to five skin lesions and a negative smear is paucibacillary, more than five lesions or nerve involvement or any positive smear is multibacillary (secondary summaries of WHO criteria. I could not open the WHO 2018 guideline itself [VERIFY]).

A village can do the sensory test and the nerve palpation with hands alone. The smear needs a microscope and Ziehl-Neelsen staining, which IN-10 describes for tuberculosis and which transfers directly (THRIVING library, IN-10).

Transmission is weak. Most exposed people never develop disease. The widely repeated figure that 95 per cent of people are naturally immune comes from public-education pages rather than peer-reviewed work, and a 2020 immunology study says only that the majority of exposed individuals are naturally immune, without a percentage. Treat 95 per cent as an unverified popular estimate [VERIFY]. What is solid is that household contacts of untreated patients with high bacillary loads carry the highest risk, that subclinical infection is common and disease is not, and that the incubation period is years. The COLEP trial gives the hardest number available: among 21,711 close contacts of 1,037 newly diagnosed patients in Bangladesh, 91 of 9,452 in the placebo arm developed leprosy over four years against 59 of 9,417 given a single dose of rifampicin (Moet et al 2008). That is an absolute risk of under one per cent

in four years among the close contacts of untreated cases. A village should hold that number in its head when it is tempted to drive a family out.

What disables. Not the bacillus directly. Paul Brand, working with Robert Cochrane at the Christian Medical College in Vellore from the late 1940s, examined the hands of patients and found that missing digits resulted from chronic bone infection beginning in cuts and abrasions that went unnoticed because the hand could not feel. His conclusion, which is the central practical finding of this whole report, was that meticulous attention to even the slightest injury should prevent the process. At Carville, Louisiana, from 1966 he built a unit to study the complications of insensitive hands and feet, and introduced the idea of moderate repetitive stress as the cause of most neuropathic foot lesions, together with total contact casting for offloading ulcers, a technique he reintroduced from Indian practice and which is now standard in diabetic foot care. The brief dates Clinical Mechanics of the Hand to 1980. The catalogue record gives Mosby, St Louis, 1985, 342 pages, with a third edition in 1999 with Anne Hollister.

7. Leprosy: the record of treatment, and the rundown plan

Chaulmoogra (tradition). The seed oil of *Hydnocarpus wightianus* and related species, used in Indian and Chinese medicine for skin disease and leprosy, made known in the West by Mouat in 1854. In Brazil *Carpotroche brasiliensis* was used as a local source. The active lipophilic compound is hydnocarpic acid, proposed to act as a biotin antagonist. Routes were oral, which was abandoned because of gastrointestinal irritation and because patients found it worse than the disease, topical, which was ineffective, intramuscular or intravenous, and intradermal or intralesional. Muir reported in 1932 that of all methods of administering hydnocarpus oil the intradermal was in his experience the most effective, and that the ethyl esters, being less viscous, infiltrated more easily. Alice Ball at the College of Hawaii developed an ethyl ester method in 1916 but died before publishing, and the work was credited to Arthur Dean. This attribution is disputed and rests on one source here [VERIFY]. Burroughs Wellcome marketed sodium hydnocarpate as Alepol from June 1927.

What the record shows. Bechelli and Rotberg in 1951 found conflicting results across studies and no methodologically adequate evidence confirming efficacy, while acknowledging local benefit (in Araújo 2005). A Carville report of 1950 noted that the tendency to relapse after arrest of lepromatous leprosy by chaulmoogra had been disappointingly high. Hawaiian workers reported that a considerable number of patients became clinically and bacteriologically free of disease but could not attribute the effect to any single fraction. A broad review of the pre-sulfone era concludes that almost all of the many substances tried were worthless. My reading is that chaulmoogra did something locally when injected into lesions, probably enough to produce visible improvement in paucibacillary disease, and did not cure multibacillary disease or prevent relapse. Grade **D**, with **X** for any claim that it cures. Selling it as effective is one of the pitfalls the brief names, and it is a real one: the oil's reputation outlasted its evidence by eighty years.

Sulfones. Guy Faget at the National Leprosarium at Carville injected the first volunteer patients with promin in March 1941, having heard of Cowdry's results in rat leprosy. His 1943 report called it the most encouraging experimental treatment ever undertaken there while stating that they were not in a position to say it had any specific action on Hansen's bacillus. By 1945 the series was 137 patients and 32,000 intravenous injections totalling about 127 kg of drug, starting at 1 g daily and working up towards 5 g. In 1946 Faget stated that promin produced improvement in all major chronic manifestations. Robert Cochrane was the first to use the parent sulphone, dapsone, in patients, and John Lowe in Nigeria from 1947 was the first to publish satisfactory results with it by mouth, after which it became the world standard until multidrug therapy. Sources disagree on who first used dapsone and when, and on the exact routes, so the sequence above is the consensus of secondary accounts rather than a verified chronology [VERIFY].

Why monotherapy failed. Relapse with resistant organisms. Indian cohorts treated in 1960 to 1962 and 1968 to 1970 relapsed at 14.3 and 7.9 per cent over seven years, the difference tracking adherence, which was 27.4 and 49.7 per cent regular. In Guadeloupe 118 relapses were seen among multibacillary patients on dapsone for five years or more, an annual incidence of 1.3 per cent, and every relapse biopsy inoculated into mouse footpads yielded dapsone-resistant organisms. In French Polynesia, 36 of 131 multibacillary patients who had become smear-negative relapsed,

the first at four years and the last at 26 years after negativity. In Hong Kong the relapse rate was 8.3 per cent after dapsone monotherapy against 3.2 per cent after multidrug therapy. No source gave a population prevalence of secondary dapsone resistance for the 1960s and 1970s and I could not find one [VERIFY].

Multidrug therapy. WHO recommended it in 1981 and the study group report is dated 1982. Adult regimens, from a 2024 review citing WHO: paucibacillary, rifampicin 600 mg once a month supervised plus dapsone 100 mg daily, for six months. Multibacillary, rifampicin 600 mg monthly supervised, clofazimine 300 mg monthly supervised plus 50 mg daily, and dapsone 100 mg daily, for twelve months. Children 10 to 14 take rifampicin 450 mg monthly, clofazimine 150 mg monthly plus 50 mg daily, dapsone 50 mg daily. Under 10 or under 40 kg: rifampicin 10 mg/kg monthly, clofazimine 6 mg/kg monthly plus 1 mg/kg daily, dapsone 2 mg/kg daily. The 2018 WHO guideline moved to a uniform three-drug set for both classes, six months and twelve months, so that misclassification does less harm. The United States programme has used a longer 24-month course for multibacillary disease. Recurrence on the old 24-dose multibacillary regimen ran about 0.1 per cent per year. About 14 million patients had been treated by 2005. Grade **A** for multidrug therapy.

The rundown. A district with a partial stock faces the same arithmetic as IN-10 sets out for tuberculosis, and the answer is the same: a short full course for one patient beats a long partial course for three. Leprosy is more forgiving than tuberculosis in one respect, since the single monthly supervised rifampicin dose is the most bactericidal element, and more punishing in another, since dapsone monotherapy is exactly what bred resistance for thirty years. If the stock cannot complete a twelve-month multibacillary course, the honest options are to treat paucibacillary patients, who need six months, or to treat the most infectious multibacillary patients for as long as supply allows and accept relapse. Who gets the last course is [OWNER]. The one thing that should not be done is to spread dapsone thinly across everyone, because that reproduces the error of 1950 to 1980 with none of its excuse.

Post-exposure prophylaxis. A single dose of rifampicin given to close contacts cut leprosy incidence by 57 per cent over the first two years, with no difference between two and four years, number needed to treat 297 overall and 265 for the first two years in the accompanying editorial (Moet et al 2008). WHO's 2018 guideline recommends it for contacts aged two and over. It is a real effect, it is not durable, and it costs one dose per contact. If a district holds rifampicin and has a new case, this is a defensible use of a small quantity. Grade **A**.

8. Leprosy: the care that actually prevents disability

This is the heart of the leprosy section, because it is the part that survives the end of drugs entirely.

What is lost. Protective sensation in hands and feet, often sweating as well, and muscle power in the small muscles of the hand and the dorsiflexors of the foot. Brand's insight was that the patient is not destroyed by the bacillus but by ordinary life conducted without pain. A hot pot handle, a nail in a sandal, a thorn, a blister from a new hoe, a pebble worn all day: each one is a wound the patient does not know about, which becomes infected, which reaches bone, which ends in amputation or absorption of the digit.

What replaces pain. Three habits, taught once and practised daily for life.

1. *Looking.* Every evening, in good light, the patient inspects both hands and both feet, including between the toes and the soles, using a mirror or a helper if the eyes are poor. Any red, warm, swollen or blistered area means rest that part until it settles.
2. *Soaking and oiling.* Numb skin is dry skin, because the sweat glands are denervated. Dry skin cracks and cracks become ulcers. Twenty minutes of soaking in plain water, then rubbing off dead skin, then a thin film of any available oil or fat, is standard self-care practice in leprosy programmes.
3. *Offloading.* The commonest cause of neuropathic ulcer is moderate repetitive stress rather than a single injury, which is Brand's finding from his footpad experiments in mice. The practical consequences are cushioned footwear that is never tight, no barefoot walking, rest for any hot swollen foot, and total contact casting or its improvised equivalent for an ulcer that will not heal.

How good is the evidence for footwear? Weaker than the practice deserves. The trial registry for the main randomised study states plainly that evidence for offloading footwear in preventing ulcers is practically non-existent, although offloading clearly helps diabetic ulcers heal. The best comparative study I found is quasi-experimental: Govindasamy and colleagues at two Leprosy Mission Trust India hospitals compared computer-designed customised insoles with standard microcellular rubber footwear plus an orthosis in 178 patients with sensory loss, and at twelve months 10 of 81 (12.3 per cent) in the intervention group and 25 of 97 (25.8 per cent) in the control group had developed an ulcer, relative risk 0.48, 95 per cent confidence interval 0.24 to 0.93. That is a halving of ulcer incidence, but allocation was not random and both arms had footwear and self-care training, so it compares good footwear with better footwear, not footwear with none. Grade **B** for protective footwear plus self-care as a package, **C** for any specific design.

Reactions, which are the emergencies. Two immune events interrupt the slow course and cause most of the nerve damage. Type 1, the reversal reaction, is acute inflammation of skin lesions and nerves with pain, tenderness and sudden loss of function. Type 2, erythema nodosum leprosum, produces crops of painful nodules with fever and can become chronic. Reversal reactions are the commonest cause of nerve damage in leprosy. The treatment is prednisolone, and the window matters: if nerve damage has been present for more than six months, steroids are unlikely to help, which is why nerve function is monitored regularly so that treatment can start at the first sign. Some neuritis is silent, with no pain to announce it, which is why regular testing rather than waiting for complaint is the rule. Thalidomide is the drug of choice for severe type 2 reaction and is both unobtainable and catastrophically teratogenic. Clofazimine and steroids are the fallback and control symptoms incompletely.

For a village the implication is sharp. Any patient with leprosy, treated or not, should have hand and foot sensation and muscle power tested at a fixed interval, monthly while anything is changing and three-monthly otherwise, with the results written down so that loss is detected as change rather than guessed at. That register, and a pair of soft wide shoes, are worth more than any plant in the garden.

9. Leprosy: segregation as cautionary history

The claim. Hansen, appointed Chief Medical Officer for Leprosy in Norway in 1875, drafted legislation enacted in 1877 and more widely in 1885 to isolate patients. Norway's national leprosy register, begun in 1856 with just over 2,850 cases, recorded a steady decline: about 1,800 known cases in 1875 and 575 by 1901. The Norwegian government presented this as proof that isolation worked, and it became instrumental in persuading other countries to legislate.

What the record shows. Meima and Irgens, analysing the same register in 2002, found that from 1856 to 1920 the new case detection rate declined steadily while the age of newly detected cases rose, and concluded that the decline cannot be explained fully by the policy of isolation and that an autonomous decrease in transmission, reflecting improvements in living conditions, must also be assumed. They state that the impact of isolation on interruption of transmission remains uncertain. The decline also began before and continued independently of any effective treatment. Hansen's own superior and father-in-law, Daniel Danielssen, who with Carl Wilhelm Boeck had argued leprosy was a hereditary dyscrasia, disputed the interpretation, and the Boeck and Danielssen position had included a warning that the contagion theory had led to inhumane treatment of the sick in the Middle Ages.

What segregation cost. On 6 January 1866 the schooner Warwick carried the first twelve patients to Kalaupapa on Molokai under a law of King Kamehameha V. About 1,200 people were held there at the settlement's peak, and roughly 8,000 are buried on the peninsula, of whom about 1,300 have marked graves. The isolation law was not repealed until 1969. Father Damien arrived in 1873, served sixteen years, contracted the disease and died there in 1889. Mother Marianne Cope served thirty years and Brother Joseph Dutton fifty-three. John Tayman's The Colony (2006) is the narrative account named in the brief. I did not read it.

The lesson for a village of 300. Leprosy transmits poorly, takes years to develop, and becomes non-infectious

within days of starting rifampicin. Isolating a leprosy patient after the Fall buys an uncertain and probably small reduction in transmission at the cost of the patient's labour, the family's food security and the village's cohesion, and at the cost of driving the next case into hiding, which is the one thing that reliably increases transmission. My reading of the Norwegian data is that removal of patients from crowded damp housing may have mattered less than the general improvement in that housing [VERIFY]. Whether to separate a case is [OWNER], but the evidence does not support compulsion, and the historical record of compulsion is Kalaupapa.

10. HIV: survival without drugs

Natural history. The Collaborative Group on AIDS Incubation and HIV Survival analysed 13,013 people with documented seroconversion dates from 15 countries, published in the Lancet in 2000 (volume 355, pages 1131 to 1137). Age at seroconversion was the most powerful predictor. For those seroconverting at 25 to 34, median time to AIDS was 9.8 years and median survival 10.9 years. At 45 to 54, 7.7 and 7.9 years. At 65 and over, 5.0 and 4.0 years. For 15 to 24 year olds one report gives about 11 years to AIDS. I could not retrieve the paper's overall median, so the brief's "about 10 years" is supported by the age-band figures rather than by a quoted single number [VERIFY].

After AIDS. A European multicentre study of diagnoses from 1979 to 1989 gave a median survival from AIDS diagnosis of 17 months, although zidovudine-treated patients were included. A São Paulo cohort gave 13.4 months for 1988 to 1993 and 22.3 months for 1994 to 1996, and cites an earlier estimate of about five months for 1982 to 1989. A San Francisco editorial covering the eras with no therapy or single and dual nucleosides gives a median survival after the first AIDS-defining opportunistic infection of 2 to 22 months depending on the infection, and surveillance there found only 7 per cent of patients diagnosed with a first opportunistic infection from 1981 to 1986 lived more than five years. The brief's "1 to 2 years after AIDS" is well supported. Note that these are figures from countries with hospitals. After the Fall they will be worse, and the published numbers already include hospital care for pneumocystis, cryptococcus and the rest.

Progression is not uniform. A single-centre cohort of people diagnosed from 1985 to 1994 found 93 of 210 (44.3 per cent) survived more than 25 years. Long-term non-progression is real, it is a minority phenomenon, and nothing a village does will identify in advance who has it.

11. HIV: the day the antiretrovirals stop

This is the scenario the book exists for, and the evidence is indirect because nobody has run the experiment deliberately at scale.

Planned interruption is harmful. The SMART trial randomised 5,472 people with CD4 above 350 either to continuous therapy or to stopping above 350 and restarting at 250. It was halted in January 2006. Interruption raised the risk of opportunistic disease or death from any cause about 2.6-fold, all-cause death about 1.8-fold, and major cardiovascular, renal or hepatic disease about 1.7-fold (figures from conference and news summaries of the NEJM paper, which I could not open [VERIFY]). Follow-up presented in 2008 found that restarting for more than two years did not erase the excess risk. The DART trial in African adults starting at low CD4 counts concluded that fixed-duration interruptions are inferior to continuous treatment.

Rebound and decline are fast. A South African cohort review states that viral load rise and CD4 decline are most pronounced in the first two months after interruption. A real-time adherence monitoring study of 479 participants found the odds of viral rebound rose by about 25 per cent for each day of interruption beyond 48 hours, and in one fixed-dose combination cohort none of 33 who never interrupted beyond 48 hours had drug resistance against 8 of 62 (13 per cent) who did. A clinician interviewed during a South African public service strike claimed resistance can develop within 7 to 10 days of stopping. That is a clinical opinion, not a study result [VERIFY].

The one technical point worth carrying. Efavirenz and nevirapine have plasma half-lives of about 35 to 45 hours, while most nucleoside analogues and protease inhibitors clear in under 10 hours. Stopping everything on the same

day therefore leaves days of effective single-drug therapy with the non-nucleoside alone, which is the ideal condition for breeding resistance to it, and in people with the CYP2B6 516 TT variant efavirenz stayed above the inhibitory threshold for a median of 14 days. The traditional response is to stop the non-nucleoside first and continue the nucleoside backbone for roughly a week. The evidence is genuinely conflicting. A Thai trial at ICAAC 2007 found a 7 to 10 day stagger did minimise non-nucleoside resistance. A 2013 analysis found a median nine-day stagger did not prevent non-nucleoside resistance mutations and increased detection of nucleoside ones. A paediatric PENTA 11 substudy found a stagger of nine days for nevirapine and fourteen for efavirenz was not associated with selection of resistance, while noting more sensitive assays might find low-level resistance.

What this means for a village. If a stock is running down, the worst plan is the one that will happen by default: everyone takes their tablets until the shelf is empty, then everyone stops on the same day. The better plans are two, and both are [OWNER]. Either the stock is concentrated so that a smaller number of people continue uninterrupted for as long as possible, which maximises person-years of suppression and minimises resistance, or it is divided equally and everyone stops together, which is fairer and medically worse. If a non-nucleoside is in the regimen and a stop is unavoidable, stopping it seven days before the rest is a defensible gesture with weak evidence behind it (Grade **C**). What is certain is that stopping is not a return to the untreated baseline: the patient restarts the clock from a lower CD4 count and with whatever resistance the interruption bred, and the SMART data say the harm does not wash out.

12. HIV: what still works when there are no antiretrovirals

Cotrimoxazole is the largest single lever. Anglaret and colleagues randomised HIV-positive adults without major comorbidity in Abidjan to trimethoprim-sulphamethoxazole or placebo and reported a significant reduction in morbidity but no effect on mortality (Lancet 1999, 353:1463 to 1468, via Grimwade and Gilks 2001). The mortality result the brief attributes to Anglaret in fact comes from the companion trial in the same issue: Wiktor and colleagues randomised 771 HIV-positive adults with smear-positive pulmonary tuberculosis to cotrimoxazole or placebo, and deaths were 51 (13.8 per 100 person-years) against 86 (25.4 per 100 person-years), a 46 per cent decrease in risk, 95 per cent confidence interval 23 to 62, p less than 0.001 (Lancet 1999, 353:1469 to 1475). This is a correction to the brief. Mermin and colleagues followed 509 people with HIV in rural Uganda for 18 months on daily prophylaxis and found significant reductions in death, hospital admission, malaria and diarrhoea, slower CD4 decline and slower viral load rise, with a number needed to treat per life-year saved of 2.6 for those with CD4 below 200 and 8.3 overall (Lancet 2004, 364:1428 to 1434). The Cochrane review pooled four trials in 1,476 adults and found, in the three African trials of 1,416 people, a relative risk for death of 0.69, 95 per cent CI 0.55 to 0.87, for morbid events 0.76, 0.64 to 0.90, and for hospitalisation 0.66, 0.48 to 0.92, with no significant excess of adverse effects and similar benefit in early and advanced disease. The WHO dose is 960 mg daily, that is 800 mg sulfamethoxazole with 160 mg trimethoprim, one double-strength or two single-strength tablets. The honest caveat, which the reviewers make themselves, is that Abidjan had unusually low cotrimoxazole resistance among bacterial pathogens, that Senegalese data showed no benefit, and that the effect in a place with high resistance is unknown.

Grade **A**. Roughly a third fewer deaths from one cheap tablet a day. This is the most important sentence in the HIV part of the book.

Tuberculosis is the other. A person with latent tuberculosis and HIV has an annual risk of active disease commonly quoted as 5 to 10 per cent per year, with published ranges of 3 to 16 per cent per year without antiretrovirals, against a lifetime risk of about 5 to 10 per cent for an HIV-negative person. A constant 10 per cent annual risk compounds to about 41 per cent over five years and 65 per cent over ten [VERIFY, my arithmetic]. In treated tuberculosis cohorts, 18.8 per cent of HIV-positive against 3.5 per cent of HIV-negative patients died during treatment (Straetemans 2011, via THRIVING library, IN-10). The village actions are those of IN-10: find and treat cough, separate sputum-positive patients from the household air, and put any HIV-positive person with a cough at the top of the queue for the remaining drugs. If isoniazid alone survives in the stock, preventive therapy for people with HIV is one of the

better uses of it.

Food. Low body mass index at antiretroviral initiation is an independent predictor of early death, and weight loss in the first four weeks predicts death even on treatment. A Malawian randomised trial enrolled 491 adults with BMI below 18.5 and compared a ready-to-use fortified spread with corn-soy blend. Corn-soy blend has been associated with disappointing results in supplementary feeding of adults with HIV, while fortified spreads based on the F-100 formula have done better in malnourished children. I could not retrieve the trial's outcomes [VERIFY]. A 2010-era review found no published controlled trial showing benefit of food supplementation in resource-limited settings, with or without antiretrovirals. So: energy-dense food matters as a prognostic marker beyond doubt and as a treatment on weak evidence. Grade **C**. Feeding the sick is right for other reasons.

Prevention that still works without any drug. Consistent condom use reduces heterosexual transmission by roughly 78 to 83 per cent, derived from serodiscordant couple cohorts in which 100 non-using couples would produce five to seven new infections a year and 100 always-using couples about one (Weller and Davis-Beaty 2002, Cochrane). The estimate is observational, not randomised, and probably flattered by differences between always-users and never-users. Grade **B**. Male circumcision reduced HIV acquisition in men by 60 or 61 per cent at Orange Farm (3,274 men), 53 per cent at Kisumu (2,784 men) and 51 per cent at Rakai (4,996 men), all three trials stopped early. Grade **A** for the effect, with the warning that it is partial, protects men rather than their partners, and is a surgical procedure with its own risks after the Fall. Per-act risks from the CDC, for an infected source and no treatment: receptive anal 1.38 per cent, needle-sharing 0.63 per cent, percutaneous needlestick 0.23 per cent, receptive vaginal 0.08 per cent, insertive vaginal 0.04 per cent.

Needles. WHO's 2000 estimates attributed about 5 per cent of new HIV infections, 32 per cent of new hepatitis B and 40 per cent of new hepatitis C in developing and transitional countries to unsafe injection, amounting to roughly 21 to 22 million hepatitis B infections, 2 million hepatitis C and 260,000 HIV in that single year. By 2010 the absolute numbers had fallen 87 per cent for HIV, 83 per cent for hepatitis C and 91 per cent for hepatitis B. The lesson transfers exactly to a village that will be reusing needles: the single most dangerous object in the dispensary is a syringe used twice, and the number it harms is dominated by hepatitis B, not HIV.

Birth and breastfeeding. Without any drugs, transmission is about 15 to 30 per cent during pregnancy and labour, with a further 10 to 20 per cent over prolonged breastfeeding, giving overall figures of 25 to 35 per cent (BMJ Clinical Evidence, a 1999 editorial). UNAIDS gives cumulative breastfeeding transmission without prophylaxis as 11 per cent at 12 months and 22 per cent at 24 months, which works out at roughly 1 per cent per month if the hazard were constant, which it probably is not [VERIFY, my arithmetic]. A mother who acquires HIV during the breastfeeding period transmits in about 30 per cent of cases, because of the high viral load of acute infection. Coutsoudis and colleagues followed 549 HIV-positive women in Durban and found that exclusive breastfeeding carried no excess risk over formula feeding, while mixed feeding roughly doubled the risk (Lancet 1999, 354:471 to 476), confirmed at 15 months in the 2001 follow-up and supported by a Zimbabwean cohort in which mixed-fed infants were more than four times as likely to be infected by six months. After the Fall, formula feeding is not an option, because clean water and a safe substitute will not exist. The recommendation that follows is therefore not a compromise but the best available: exclusive breastfeeding, nothing else by mouth at all, not water, not gruel, not traditional first foods, for six months, then weaning as fast as the food supply allows. Grade **B**.

13. Hepatitis B and C: the carriers already among us

Hepatitis B. Transmission is by blood, by sexual contact, and above all at birth. Age at infection determines everything. The CDC Pink Book gives chronic carriage after infection at about 95 per cent for infants infected at birth, 80 per cent at one to six months, 55 per cent at seven to twelve months, 30 per cent at one to four years and 10 per cent at five years and older. A 2025 ACIP briefing gives 90 per cent for infants and fewer than 5 per cent for those five and older. Without prophylaxis, 70 to 90 per cent of infants born to mothers who are both surface-antigen and e-antigen positive become infected, and about 90 per cent of those become chronic carriers. For e-antigen negative

mothers the figure is 5 to 20 per cent. The vaccine alone prevents about 75 per cent of perinatal transmission, which is why immunoglobulin is normally added, and overall efficacy is about 95 per cent with a range of 80 to 100 per cent, with protection lasting at least 20 years and no routine boosters. Infant antibody response is 16 to 40 per cent after one dose, 80 to 95 per cent after two and 98 to 100 per cent after three.

This is the clearest case in the whole report for spending a dwindling stockpile. A birth dose of hepatitis B vaccine given within 12 hours of delivery to the baby of a known carrier prevents a lifetime of chronic infection in a person who will otherwise probably die of liver disease decades later. One dose gives perhaps a quarter of the protection of three, which is still worth giving. HY-10's stockpile arithmetic applies, and hepatitis B vaccine is among the more heat-tolerant items in the store (THRIVING library, HY-10).

Hepatitis C. Roughly 50 to 85 per cent of acute infections become chronic, with spontaneous clearance perhaps 25 per cent in adults and higher in children. Cirrhosis develops in roughly 10 to 30 per cent over 20 to 30 years, with wide disagreement across cohorts and lower rates in young people. Estimated per-exposure transmission is high for injecting equipment and haemodialysis, about 2 per cent for needlestick and about 2 per cent from mother to child. There is no vaccine and there will be no direct-acting antivirals. The village's hepatitis C policy is therefore entirely a needle and razor and tattoo policy.

What the village does for both. No sharing of needles, razors, toothbrushes or any skin-piercing tool. If a needle must be reused it is reused for the same person only, and boiled between uses. Blood transfusion, if the district ever attempts it, is the single highest-risk act available and is covered elsewhere in the series. Cover open wounds. A known carrier does not need isolation, separate dishes or exclusion from work, and saying so plainly is part of the job.

14. Hepatitis A and E: water, and the pregnant woman

Hepatitis A. Faecal-oral, person to person and through contaminated water and food. Shedding peaks in the two weeks before illness, so the infectious person looks well. In children under six about 70 per cent of infections are asymptomatic and jaundice appears in fewer than 10 per cent, while in older children and adults jaundice appears in more than 70 per cent. Severity rises with age. Case fatality is 0.1 to 0.6 per cent overall and up to 1.8 per cent over 50, with figures of 12.5 per cent reported among hospitalised patients over 60. There is no chronic carriage, although recurrent hepatitis for up to a year occurs in about 15 per cent. Where sanitation is poor almost all children are infected before the age of ten and the adult population is therefore immune, which is the paradox a village must understand: improving water supply without vaccinating raises the average age of infection and therefore the death rate from it. In the United States, 23 of 32 drinking-water-associated hepatitis A outbreaks from 1971 to 2017 involved untreated groundwater. The infectious dose is estimated at 10 to 100 virus particles, which is an estimate rather than a measurement.

Hepatitis E. The same route, with a devastating difference. Case fatality is under 4 per cent in general, and 15 to 25 per cent in pregnant women, especially in the third trimester, in regions where genotypes 1 and 2 circulate. The Darfur outbreak of 2004 gave an incidence fatality ratio of 17.8 per cent overall and 31.1 per cent among pregnant women, in a displaced population with poor sanitation. In an Islamabad waterborne epidemic the attack rate among 162 pregnant women was 21.6 per cent against 10.9 per cent in non-pregnant women of the same ages, and all four adult deaths were third-trimester women, with a case fatality given as 11.4 per cent on an unclear denominator. The brief's 20 per cent figure is a reasonable central estimate and not a constant.

What the village does. Everything in this section is a water and latrine problem and belongs with the hygiene reports. The one clinical instruction that is specific to IN-15 is this: when jaundice appears in more than one household at once, pregnant women must be moved to the cleanest available water and the most careful food handling in the settlement, immediately, before anybody knows which hepatitis it is. That single act is the only intervention with a plausible 20 per cent absolute mortality benefit anywhere in this report [VERIFY, inference from the case fatality figures rather than from a trial].

15. False cures, and why they belong in a medical book

The pitfall the brief names is real and recent. Manto Tshabalala-Msimang, South Africa's health minister, promoted raw garlic, lemon, olive oil, beetroot and the herbal preparation ubhejane while questioning antiretrovirals, and the government restricted donated nevirapine and delayed Global Fund money. Chigwedere and colleagues, using Botswana and Namibia as comparators, estimated more than 330,000 lives lost in South Africa between 2000 and 2005 because a feasible and timely antiretroviral programme was not implemented, plus tens of thousands of preventable infant infections (Journal of Acquired Immune Deficiency Syndromes 2008, 49(4):410 to 415). The Academy of Science of South Africa stated in 2007 that there was no evidence those foods substituted for medicine. The figure is a modelled estimate with conservative assumptions and sensitivity analyses by the authors' account, and attributing deaths to a single policy is inherently uncertain, but the direction is not in doubt.

The point for this book is not that traditional medicine is worthless. It is that a remedy which merely occupies the place of an effective one kills at the rate of the untreated disease, and that the deaths are invisible because they look like the disease. Chaulmoogra oil for leprosy and garlic for HIV sit in the same category, eighty years apart. The test a village should apply is whether a remedy is being offered instead of something that works. Comfort measures offered alongside the truth are a different thing entirely and are most of what this report recommends.

16. The words for a prognosis

Nothing in the literature of these four diseases is as consistently neglected as the sentence the healer has to say. From the palliation literature and the leprosy and HIV record, four rules emerge.

First, separate what is certain from what is not. Rabies after symptoms begin: certain. A dog bite with an unobserved animal: a risk, roughly one in two for face, one in four for hand, one in twenty for leg from a genuinely rabid dog (THRIVING library, WO-13). HIV without drugs: a decade, usually, and then one to two years. Leprosy: not fatal, and the disability is preventable. Hepatitis B carriage: most carriers die of something else.

Second, name the time. "I do not know" is honest about an individual but useless. "Most people in your position live about ten years from infection, and some live much longer" is both honest and usable.

Third, say what will be done. A prognosis without a care plan is abandonment with vocabulary. The plan for rabies is sedation and a dark room and company. The plan for HIV is the tablet, the food, the cough watch and the children's protection. The plan for leprosy is the shoes, the evening inspection and the nerve test.

Fourth, do not require the patient to accept it today. Warrell's team make this point about rabies specifically, that the drug given slowly buys the family time to decide. The same applies to a diagnosis of HIV in a village of 300, where the social consequences arrive faster than the medical ones.

Evidence grades

Remedy or practice	Tradition says	Record shows	Grade
Soap wash 15 min after bite	cleansing	guinea-pig protection with repeated swabbing, no human trial, WHO first-line	B
Ten-day confinement of biting dog	folk practice	no US case from an animal well at day 10	B
Mass dog vaccination to 70 per cent	n/a	Pemba 42 cases to 0, modelling confirms	A

Remedy or practice	Tradition says	Record shows	Grade
Culling free-roaming dogs for rabies control	n/a	unproven as control, reduces bites	C
Milwaukee protocol	n/a	1 survivor 2004, near-universal failure since	X
Palliative diazepam/midazolam/morphine in rabies	n/a	expert consensus, one 45-patient series	C
Dark quiet room, no draught, no water in sight	folk nursing	mechanism clear, no trial	D
Chaulmoogra oil, intralesional	cures leprosy	local improvement, high relapse, no adequate evidence	D (X for cure)
Dapsone monotherapy	n/a	worked then bred resistance, 8.3 per cent relapse	B then X
WHO multidrug therapy	n/a	14 million treated, 0.1 per cent per year relapse	A
Single-dose rifampicin for contacts	n/a	57 per cent at 2 years, NNT 297 (Moet 2008)	A
Prednisolone for leprosy reactions	n/a	consensus, window under 6 months	B
Daily inspection, soaking, oiling of numb limbs	n/a	Brand's mechanism, programme standard	B
Protective footwear plus self-care	n/a	ulcers 12.3 vs 25.8 per cent, RR 0.48, non-randomised	B
Segregation of leprosy patients	prevents spread	Norwegian decline not explained by it	D
Cotrimoxazole 960 mg daily in HIV	n/a	RR death 0.69 (Cochrane), 46 per cent in TB co-infection	A
Energy-dense food in HIV wasting	n/a	BMI predicts death, no controlled benefit shown	C
Exclusive breastfeeding vs mixed	n/a	mixed roughly doubles transmission (Coutsoudis 1999)	B
Consistent condom use	n/a	78 to 83 per cent reduction, observational	B
Male circumcision	n/a	51 to 60 per cent in three RCTs	A
Hepatitis B birth dose within 12 h	n/a	about 75 per cent of perinatal transmission prevented	A
One needle, one person, boiled between	n/a	32 and 40 per cent of HBV and HCV from reuse	B

Remedy or practice	Tradition says	Record shows	Grade
Moving pregnant women to clean water in a jaundice outbreak	n/a	HEV 15 to 25 per cent CFR in pregnancy	C
Staggered stop of a non-nucleoside	n/a	conflicting trials	C
Garlic, beetroot, ubhejane for HIV	cures	no evidence, displacement deaths	X

Means ledger

Rung	Rabies	Leprosy	HIV	Hepatitis
Hands and knowledge	15-min wash with water, dark quiet room, company, honest words, confine the dog	sensory testing with cotton wool, nerve palpation, evening inspection, no barefoot walking, teaching	abstinence and partner reduction, no shared blades, exclusive breastfeeding, cough watch	handwashing, latrine siting, no shared razors
Hearth	soap for the wash, boiled cloth, fluid by rectum, alcohol by mouth in lucid intervals	soaking and oiling, soft wide footwear from scrap, rest for a hot foot, dressings	food, boiled water, clean dressings, nursing through diarrhoea	boiled drinking water, separate the jaundiced person's stool
Garden and still	ethanol for wound antisepsis after soap, beeswax and fat for ulcer dressings	moulded leather or rubber sandal, crutches, splints for a dropped foot	nothing that treats HIV, better food, honey dressings	nothing specific
Village dispensary (300, lay healer, microscope)	salvaged vaccine intradermally 0.1 mL x2 on days 0, 3, 7, immunoglobulin into the wound, dog register, bite register	slit-skin smear, nerve function register, dapsone or MDT while stock lasts, prednisolone for reactions	cotrimoxazole 960 mg daily to every known case, tuberculosis smears, birth plan for positive mothers	hepatitis B birth dose from stock, needle discipline, outbreak water response
District infirmary (3,000, nurse, lab)	sedation by infusion, intravenous fluid, isolation room, pre-exposure vaccination of carers	MDT courses with supervised monthly dose, total contact casting, tendon transfer if a surgeon exists	partial antiretroviral stock with a written rationing rule, isoniazid prophylaxis, infant feeding counselling	serology if any test strips survive, vaccination programme from stock
Hospital before the Fall	PEP within hours, intensive care, intravenous immunoglobulin	MDT, reconstructive surgery, thalidomide	lifelong suppressive triple therapy, viral load, resistance testing	antivirals, interferon-free cure for HCV, transplant

What each rung changes. For rabies, the step from hands to hearth is the whole game: soap in the first minute is worth more than everything above it, and no rung above the dispensary adds anything once symptoms start. For

leprosy, the step that matters is dispensary to district, because a complete drug course cures and a partial one breeds resistance, but the disability outcome is set at the hands-and-knowledge rung by inspection and footwear. For HIV, the dispensary rung with cotrimoxazole is worth roughly a third of the deaths and the district rung without a full drug supply adds little. For hepatitis, everything above the hearth is marginal compared with water and needles, except the birth dose.

Harm test

Rabies palliation. Benzodiazepines and opioids depress breathing. In a dehydrated, exhausted patient the sedating dose and the apnoeic dose converge. Diazepam intramuscular is painful and absorbed erratically, and the rectal route is more reliable in an agitated patient. Morphine is for pain and is a poor sedative, and palliative-care guidance specifically advises against using opioids as the sedating agent because their sedating effect is unpredictable. Signs that it is going wrong: respiratory rate falling below about 8, deep cyanosis, loss of all response. The response is to stop further doses and support the airway, not to add more. The family must be told before the first dose what the drug can and cannot do, and that deep sedation may shorten life. Deliberate overdose is euthanasia [OWNER].

Rabies prophylaxis. The harms here are omissions. Indian case series document deaths in people who received proper wound care and at least three intramuscular vaccine doses but no immunoglobulin because none was available. A 2005 Indian Pediatrics letter describes a child who died after vaccine and immunoglobulin where only about half the immunoglobulin was infiltrated locally and the wound was sutured, the authors citing short incubation, incomplete local infiltration and suturing as contributors. A pooled analysis of 30 Indian studies found post-exposure prophylaxis compliance of 57 per cent. Suturing a rabies-prone wound is the specific error to name. So is a dog "confined" that escapes.

Leprosy drugs. Dapsone causes haemolysis, severe in glucose-6-phosphate dehydrogenase deficiency, and a rare hypersensitivity syndrome with fever, rash and hepatitis in the first weeks that can kill. Rifampicin turns urine, sweat and tears orange, which must be explained or the patient stops, and intermittent dosing after an interruption has caused flu-like reactions and, rarely, renal failure and haemolysis. Clofazimine darkens the skin, sometimes for years, which is itself a cause of stopping treatment in a village where everyone can see it. Prednisolone for reactions unmasks tuberculosis, raises blood sugar, and causes gastric bleeding. It must not be started in someone with an undiagnosed cough. Thalidomide must never be given to a woman who could become pregnant.

Leprosy care. The hazards of self-care are soaking in water that is too hot, which a numb limb cannot report, and footwear that is too tight, which produces exactly the ulcer it was meant to prevent. Test water temperature with an elbow or a thermometer, never a numb hand. Shoes must be fitted while the foot is swollen at the end of the day. Aggressive paring of callus with a knife in a numb foot is how a healer creates an ulcer.

Cotrimoxazole. Sulphonamide hypersensitivity, including Stevens-Johnson syndrome, which is more common in HIV than in the general population. Rash in the first weeks is the warning. A rash with mucosal involvement, blistering or fever means stop permanently and never rechallenge. It also causes marrow suppression, raises potassium and interacts with folate. It should not be given with methotrexate. In pregnancy WHO still recommends it for women with advanced disease, balancing the risks, but folate supplementation matters. Resistance rises with widespread use, and in a place with high background cotrimoxazole resistance the benefit may be absent.

Stopping antiretrovirals. Stopping everything at once with a non-nucleoside in the regimen creates functional monotherapy and resistance. Stopping and restarting repeatedly is worse than either continuing or stopping once. SMART showed the harm persists after restarting.

Isolation. The harm of segregation is historical, large and documented: Kalaupapa. The medical harm is that visible compulsion drives the next case into hiding, which increases transmission, and that an isolated patient in a village of 300 loses the labour that feeds them.

False cures. Chigwedere's estimate of 330,000 deaths is the harm figure for a remedy offered instead of one that works.

Red flags

These are the signs that mean death or disability is coming and the village cannot stop it. In each case there is still something to do.

1. **Tingling, itching or pain at a healed bite site, with fever.** Rabies has begun. Nothing will cure it. Begin palliation, prepare the family, offer pre-exposure vaccination to carers if any vaccine exists, and do not attempt intensive measures.
2. **Hydrophobia or aerophobia.** The same, confirmed. Dark room, no draught, sedation, company.
3. **Sudden loss of power or sensation in a leprosy patient, with pain or tenderness over a nerve.** A type 1 reaction. Without steroids within weeks the loss is permanent. Splint the limb in a functional position, rest it absolutely, and give prednisolone if any exists. After six months of established loss, steroids will not help.
4. **A painless deep ulcer over a pressure point in a numb foot, with probing to bone.** Osteomyelitis. Without antibiotics and offloading this ends in amputation or absorption. Offload completely, immobilise, dress, and give whatever antibiotic exists.
5. **A person with HIV and persistent cough, fever and weight loss.** Tuberculosis until proved otherwise. Without drugs this is usually fatal within months. Smear if possible, give the drugs ahead of others, and separate the sputum from the household air.
6. **Headache, neck stiffness and confusion in a person with HIV.** Cryptococcal or tuberculous meningitis. Both are untreatable in a village and both kill. Analgesia, dark room, fluid, honest words.
7. **CD4 unknown but oral thrush, chronic diarrhoea and wasting together.** Advanced disease. Expect one to two years at most, and less without hospital. Cotrimoxazole, food, and planning for the children.
8. **Jaundice in a pregnant woman in an outbreak of jaundice.** Hepatitis E is likely and the risk of death is 15 to 25 per cent. There is no treatment. Clean water, rest, careful attention to bleeding after delivery, and a skilled attendant at the birth.
9. **A newborn of a hepatitis B carrier with no vaccine available.** Nine chances in ten of lifelong carriage. Record it, and give any vaccine that arrives later.
10. **Drowsiness, confusion or bleeding in anyone with jaundice.** Fulminant hepatitis of any cause. Mortality approaches 80 per cent and nothing in a village changes it. Comfort.

The case against

Against the whole frame. The strongest case against this report is that it is a counsel of resignation dressed as realism. Jean Willoughby's position on the Milwaukee protocol, maintained against his critics, is that a disease thought universally fatal produced a survivor once, and that the correct response to one survivor is to keep trying rather than to declare the matter closed. Alan Jackson's position, which this report follows, is that trying has killed dozens of patients in intensive care units while teaching nothing, and that the protocol's elements have no mechanistic support. The honest reading is that Jackson is right about the evidence and Willoughby is right about the attitude, and that a village has no way to act on the attitude.

Against the cotrimoxazole recommendation. Grimwade and Gilks pointed out that Abidjan had atypically low cotrimoxazole resistance among bacterial pathogens, that Senegalese data showed no benefit, and that extrapolating is unsafe. After the Fall, with decades of uncontrolled antibiotic use behind it, a community's resistance profile is unknown and may be bad. The counter-argument is that malaria and diarrhoea contribute much of the benefit in the Ugandan data, and that the Cochrane pooled estimate held across early and advanced disease. I keep the recommendation and flag the uncertainty.

Against the leprosy footwear claim. The registry of the main randomised trial states that the evidence for offload-

ing footwear in preventing ulcers is practically non-existent. The Govindasamy comparison is non-randomised and compares two kinds of footwear, not footwear with none. A sceptic can say that the whole self-care edifice rests on Brand's mechanism and on programme consensus rather than on trials. That is accurate. The reply is that the mechanism is as close to certain as mechanisms get, that the intervention is cheap and reversible, and that nobody will ever randomise patients to bare feet.

Against the advice on isolation. A public health officer could argue that Meima and Irgens showed only that isolation was not the whole explanation for Norway's decline, not that it did nothing, and that a village with no drugs at all has fewer tools than Norway had. That is fair. My position is that the evidence for benefit is weak and uncertain, the evidence for harm is a century of documented cruelty, and a weak uncertain benefit does not justify compulsion. Others may weigh it differently [OWNER].

Against the advice on exclusive breastfeeding. Coutsoudis herself called for replication, and the Guttmacher commentary argued that recommendations may be most usefully made at the level of the individual mother. In a setting with no safe alternative the argument is somewhat academic, but a critic is right that the mechanism by which exclusive feeding is safer than mixed feeding is not known.

Against writing any of this down. The deepest objection is that a book which tells a village that rabies is always fatal, that HIV gives about ten years, and that leprosy drugs will run out, removes hope and therefore removes effort. The answer is the structure of this report: every one of those sentences is followed by a list of things that still work. The alternative, a book that implies the village can cure what it cannot, produces the South African decade.

10-line summary

1. Rabies is near-certainly fatal once symptoms begin, and washing a bite with soap for fifteen minutes in the first hour is the largest single lever a village has over any of these diseases.
2. The Milwaukee protocol produced one survivor in 2004 and has failed essentially every time since. It requires intensive care a village does not have and should not be attempted.
3. A rabies death can be made bearable with a dark quiet draught-free room, sedation by any available benzodiazepine, fluid by a non-oral route, and the family present, and there is no documented household transmission to fear.
4. Mass dog vaccination to 70 per cent coverage eliminates human rabies where it is done. Without vaccine, a dog register and real ten-day confinement of biters are what remains.
5. Leprosy transmits poorly, takes years, and does not cause the deformity directly. Injury to numb hands and feet does, and Brand showed that meticulous attention to small injuries prevents it.
6. Chaulmoogra oil was never shown to cure leprosy. Dapsone alone bred resistance for thirty years. Multidrug therapy from 1982 cures, and a partial stock should complete courses for a few rather than be spread thinly.
7. Untreated HIV gives a median of about ten years from infection to death, with age the strongest predictor, and one to two years once AIDS begins.
8. Cotrimoxazole 960 mg daily reduces death in HIV by about a third, and by 46 per cent in those with tuberculosis, and is the single most valuable drug a village can hold for HIV.
9. Hepatitis B is 90 per cent chronic when acquired at birth and under 5 per cent in adults, so a birth dose of vaccine and one needle per person are worth more than any treatment.
10. Hepatitis E kills 15 to 25 per cent of pregnant women in the third trimester, so moving pregnant women to the cleanest water at the first sign of a jaundice outbreak is a life-saving act requiring no drug.

Open questions

- Does washing alone, without vaccine, measurably reduce human rabies deaths? No human study shows it, and the often-quoted 90 per cent reduction could not be traced in WO-13 or here.

- What proportion of people are genuinely resistant to *M. leprae*? The 95 per cent figure is popular, not peer-reviewed.
- How long after a household case does untreated leprosy transmission continue, and does separating sleeping quarters without social exclusion help?
- Does a staggered stop of a non-nucleoside actually prevent resistance? The trials conflict.
- What is the real survival of untreated HIV in a setting with no hospital at all? Every published figure includes some care.
- Does cotrimoxazole prophylaxis retain its benefit in a population with high background resistance?
- Does food supplementation reduce HIV mortality independently of antiretrovirals? No controlled trial has shown it.
- Does culling free-roaming dogs control rabies, or only reduce bite numbers?
- What is the per-month hazard of breastfeeding transmission, and does it fall with time? The cumulative figures do not decompose cleanly.
- Who gets the last course of multidrug therapy, the last antiretroviral, the last vaccine dose? [OWNER]
- Is deep sedation that shortens a rabies death permissible? [OWNER]

Sources to read in full

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At the bedside after the Fall

A household. The minimum is a bar of soap kept where a bitten child can reach it, a clock or a counted rhythm for fifteen minutes, and the knowledge that the dog must be tied up and watched for ten days rather than killed. For a numb hand or foot in the house, the minimum is an evening inspection in good light and shoes that are never tight. For a person with HIV, the minimum is food, boiled water and a daily tablet while any remains. For a newborn, the

minimum is exclusive breastfeeding for six months if the mother is positive, and a hepatitis B birth dose within twelve hours if any vaccine exists. **Needs to start:** soap, a lamp, a pair of wide soft shoes, a rope for the dog. **First failure to expect:** the wash will be done for two minutes, not fifteen, because fifteen minutes with a frightened child feels impossibly long. Time it out loud.

A village of 300. The minimum is three registers kept by one named person: a bite register, a dog register and a register of people with long illnesses, recording for each leprosy patient the result of the monthly nerve and sensation test. Add the fifteen-minute wash as a trained drill, confinement of biting dogs as a rule everyone knows, cotrimoxazole to every known HIV case while the stock lasts, one needle per person, and a decision made in advance about who holds the last doses of anything. **Needs to start:** a literate keeper of registers, a measured container of soap, somewhere to confine a dog, a microscope if one survives, and a settled public position that people with leprosy, HIV and hepatitis stay in the village. **First failure to expect:** the first leprosy case will be hidden, because the family will have heard the old stories. The counter is to announce the policy before the first case, not after.

A district of 3,000. The minimum adds a written rationing rule for every scarce drug, agreed in advance and in public, so that it is not decided at the bedside by whoever is loudest, a nurse or healer trained in intradermal injection so that rabies vaccine stretches six-fold, the capacity to make a sedated death decent, meaning a quiet room and a small reserved stock of diazepam, and smear microscopy for leprosy and tuberculosis. **Needs to start:** a lockable store, a scale, a microscope and stain, someone who can teach the nerve examination, and an agreed rule. **First failure to expect:** the rationing rule will be broken for the first sympathetic case, and once broken it will not be re-established. Write it down and name who may override it.

Protocols

P1. Bite by any mammal (summary, full version in WO-13 and HY-10)

1. Wash with soap and running water for a full fifteen minutes, timed. Flush deep punctures. Water alone if no soap.
2. Apply 70 per cent alcohol or iodine afterwards if either exists.
3. Do not close the wound. Do not suture. Delay any closure about four days.
4. Confine the biting dog or cat with food and water. Watch it for ten days. Well at day ten means no rabies from that bite.
5. If vaccine exists: 0.1 mL intradermally at two sites on days 0, 3 and 7. If immunoglobulin exists, infiltrate as much as anatomically fits into and around the wound, once.
6. Record animal, owner, site, washing duration and every dose.

P2. Rabies has begun (symptoms present)

1. Confirm by history: a bite or lick on broken skin in the past week to one year, plus tingling, itching or pain at that site, plus fever, agitation or fear of water or draughts.
2. Tell the family plainly that this will kill, that it usually takes about one to two weeks, and that everything from now is comfort.
3. Prepare one room: dark or dimly lit, no draught, no running water audible, no loud noise, one or two trusted people at a time.
4. Sedate. Diazepam adult 10 mg slow intravenous over 3 to 5 minutes, repeat every 1 to 4 hours as needed, or 20 mg intramuscular every 2 hours, or 10 mg rectally every 1 to 4 hours. Child 0.1 to 0.3 mg/kg by any of those routes every 1 to 4 hours. Titrate to a calm patient who still breathes at 10 or more breaths a minute.
5. If agitation persists, add haloperidol 5 mg hourly until calm then 4 to 6 hourly, or chlorpromazine 25 to 50 mg deep intramuscular every 6 to 8 hours.
6. Fluid by a route that is not the mouth: subcutaneous or rectal, about 1 litre over 6 to 8 hours for an adult, adjusted to urine output. Moisten lips with a damp sponge.

7. Fever: paracetamol 1 g rectally every 4 to 6 hours, maximum 4 g in 24 hours. Child 125 to 500 mg rectally every 4 to 6 hours.
8. Secretions: hyoscine hydrobromide 400 micrograms subcutaneously every 4 hours if available.
9. Pain: morphine 10 mg every 4 hours, or 15 to 30 mg rectally every 4 hours. Not as a sedative.
10. Carers: gloves and a cloth over the face if available, no saliva on broken skin, wash utensils with soap. Offer vaccine to carers if any exists. Do not exclude the family.
11. Do not attempt induced coma, ribavirin, amantadine or any heroic measure.

P3. New leprosy case in the village

1. Examine: count skin lesions, test each for sensation with cotton wool with eyes closed, palpate ulnar, median, radial cutaneous, common peroneal, posterior tibial and great auricular nerves for thickening and tenderness.
2. Record power: little finger abduction, thumb abduction, wrist and foot dorsiflexion, eye closure. Score each as normal, weak or absent.
3. Classify: 5 or fewer lesions and no nerve involvement is paucibacillary. More than 5, or nerve involvement, or a positive smear, is multibacillary.
4. If drugs exist, start the full course. Paucibacillary: rifampicin 600 mg once monthly supervised plus dapsone 100 mg daily, six months. Multibacillary: rifampicin 600 mg monthly supervised, clofazimine 300 mg monthly supervised plus 50 mg daily, dapsone 100 mg daily, twelve months. Child 10 to 14: rifampicin 450 mg, clofazimine 150 mg monthly plus 50 mg daily, dapsone 50 mg daily. Under 10 or under 40 kg: rifampicin 10 mg/kg monthly, clofazimine 6 mg/kg monthly plus 1 mg/kg daily, dapsone 2 mg/kg daily.
5. Warn about orange urine and darkening skin before the first dose, or the patient will stop.
6. If rifampicin exists but not a full course, give household contacts aged two and over a single 600 mg dose once (adult), 10 to 15 mg/kg for children.
7. Repeat the nerve and sensation examination monthly for the first year, then three-monthly for life, and write it in the register each time.
8. Say in public that the patient stays in the village and keeps working.

P4. Care of a numb hand or foot, every day for life

1. Evening inspection in good light: palms, backs, between fingers and toes, soles, heels. Use a mirror or helper if the eyes are poor.
2. Any area red, warm or swollen: rest that part completely until it is cool and the same colour as the rest, usually three to seven days.
3. Soak 20 minutes in plain water tested with an elbow, never a numb hand. Rub off dead skin with a rough cloth. Apply a thin film of oil or fat.
4. Never walk barefoot. Footwear wide enough that a finger slides in at the heel, fitted at the end of the day when the foot is largest, soft under the ball of the foot.
5. For any tool with a handle, pad the handle. For cooking, use a cloth or tongs, never a bare numb hand near fire.
6. An ulcer: clean, dress, and offload completely. Complete offloading means no weight at all on that foot, by crutches or a cast, until it closes. Partial offloading does not work.
7. Measure and record ulcer size weekly. No reduction in two weeks means the offloading is not complete.

P5. HIV without antiretrovirals

1. Cotrimoxazole 960 mg (800/160) once daily, every day, for every known case. Child dosing by weight, halved and quartered tablets.
2. Stop permanently and never rechallenge if a rash appears with fever, blistering or mouth or eye involvement. A mild rash alone may be watched closely.

3. Weigh monthly. Record. Falling weight is the prognostic signal that matters most.
4. Feed: the most energy-dense food available, plus salt and oil. Treat diarrhoea with oral rehydration solution as in the relevant report.
5. Ask about cough at every contact. Cough over two weeks with fever, night sweats or weight loss goes to the front of the tuberculosis queue.
6. Pregnancy: exclusive breastfeeding for six months, nothing else by mouth, then wean as fast as the food supply allows. Do not mix feed.
7. Keep the diagnosis in a register, not in public. Say in public that people with HIV eat, work and sleep in the village.

P6. Running down an antiretroviral stock

1. Count the stock in person-months, by regimen, before announcing anything.
2. Decide and write the rule before the shortage bites: either concentrate supply in fewer patients for longer, or divide equally and stop together. Name who may override it [OWNER].
3. If a non-nucleoside (efavirenz or nevirapine) is in the regimen and a stop is unavoidable, stop the non-nucleoside seven days before the rest, continuing the backbone alone for those seven days.
4. Never stop and restart repeatedly. One planned stop is less harmful than several unplanned ones.
5. On the day of stopping, start or continue cotrimoxazole for everyone who stops.
6. Record the date each person stopped, so that any future supply can be allocated by CD4 proxy and by time off treatment rather than by argument.

P7. Jaundice outbreak

1. Count cases by household and by water source. Map them.
2. Move every pregnant woman to the cleanest water source and the most careful food handling available, that day, before any diagnosis is made.
3. Boil or chlorinate all drinking water. Inspect latrines upstream of every well and spring.
4. Treat the jaundiced with rest, fluid and food. There is no specific treatment for A or E.
5. Watch for drowsiness, confusion or bleeding. These mean fulminant disease and near-certain death. Give comfort.
6. Assume a jaundiced person is infectious from two weeks before the jaundice appeared, and trace accordingly.

Numbers to carry

1. **Fifteen minutes.** The soap wash after any mammal bite, timed by a clock, not by feel.
2. **Ten days.** A dog or cat alive and well ten days after it bit was not shedding rabies virus when it bit.
3. **Seventy per cent.** The dog vaccination coverage that eliminates human rabies where vaccine exists.
4. **One to two weeks.** From first rabies symptom to death, and therefore the length of the vigil.
5. **Six and twelve months.** The leprosy multidrug course for paucibacillary and multibacillary disease. Below that, expect relapse.
6. **Fifty-seven per cent, number needed to treat 297.** Single-dose rifampicin for leprosy contacts, protection lasting two years.
7. **Ten years, then one to two.** Median survival from HIV infection, and from the onset of AIDS, without drugs.
8. **Nine hundred and sixty milligrams.** Daily cotrimoxazole in HIV, which cuts deaths by about a third and by 46 per cent in those with tuberculosis.
9. **Ninety per cent and five per cent.** Chronic hepatitis B carriage after infection at birth and after infection as an adult. That gap is the whole argument for the birth dose.

10. **Fifteen to twenty-five per cent.** The death rate of hepatitis E in pregnant women in the third trimester, which is why they are moved first.

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