# 14 Chemoprophylactics

*Chemoprophylaxis* refers to the administration of medication to prevent disease or infection. This method was based on the conviction that *hutachiwana hwen'gana*, which *vachena* called *Trypanosoma congolense* (literally, "the trypanosome of Congo," because that is where it was first "discovered" by bench scientists), occurred only within the blood of infected *mombe* (cattle), not outside the vascular system. Although most strains produced low concentrations of *nyongororo* (parasite) in the jugular vein, large numbers of *hutachiwana* could be observed in microcirculation, especially in the brain, heart, and ear veins (Maxie and Loses 1977). In Rhodesia (from 1963 on), chemotherapy was almost as old as white rule itself. By the time that drug prophylactics and other trypanicides were extended to deal with *n'gana* outbreaks in resettled areas cleared of *mhesvi* (but nonetheless prone to reinvasion) in the 1950s, they had been in use since the early twentieth century.

One of the biggest problems that Rhodesia's Branch of Tsetse and Trypanosomiasis Control (BTTC) faced throughout the Rhodesia period was *hutachiwana*'s habit of developing a degree of resistance to the drug deployed to destroy it. The condition that made resistance possible was one in which the "dosage or concentration of drugs [was] too low to kill outright, so that the microbe [had] time to organize its resistance."[1] To develop that capacity, large numbers of *hutachiwana* had to be constantly exposed to a drug circulating within the animal's body. To deal with such resistance, vets across *n'gana*-infected Africa developed "stoppers"—combinations of drugs used when resistant *hutachiwana* were encountered and one drug alone proved inadequate to conquer them. The task for which stoppers were deployed—overcoming drug-resistant trypanosome strains—was called a "challenge."[2]

As early as 1944, skeptics were already warning that the new chemical weapons against pests were "turning out to be double-edged weapons" that "may at the same time destroy both useful and harmful agricultural

insects." Indeed, as Jane Stafford cautioned, "They may rid your dog of fleas, but insidiously … damage his liver or paralyze him through nerve damage. They will rid your home of mosquitoes, flies and vermin, but the price may turn out to be high in human health and life" (Stafford 1944, 90). This is what happened in Rhodesia.

This chapter first gives a historical overview of chemoprophylaxis in Southern Rhodesia, then turns to the problem of drug resistance and photosensitization, which, as noted earlier, is a clinical condition in which the skin's negative exposure and reaction to sunlight is heightened due to phototoxic drugs and chemicals. This photosensitivity occurs when these substances absorb sunlight (ultraviolet radiation), triggering a burning sensation, redness, and swelling. Within twenty-four hours, the skin becomes pigmented and starts peeling off. The animal dies a slow, painful death; the owner endures the pain of seeing his wealth, his cherished cow, ox, or bull, losing its skin piece by piece. *Hutachiwana* attached itself to capillary walls, and trypanicidal drugs worked by disturbing and forcing them off the walls into the general blood circulation system. Any treatment regimen had to work around that to prevent drug resistance; few drugs could.

The chapter ends with a case study of chemoprophylaxis operations in Southern Rhodesia, exploring how the early promises of chemoprophylaxis ended with unforeseen complications that poisoned instead of cured animals of *n'gana*. The argument made is one about pollution of the most intimate kind: within the body of both the animal and *hutachiwana* itself. This chapter will show a general pattern among all the drugs; they worked well initially before *zvipfuyo* (livestock) either relapsed or exhibited signs of drug resistance (Whiteside 1962), prompting the deployment of one drug to cure another.

This chapter must be read in the context of what kinds of prophylactic regimes these new materials were now replacing—specifically, two forms of inoculation already referred to that *vatema* practiced. The difference lies in the toxicity of the new methods. These methods of dealing with pestiferous micromobilities inside their bodies and those of their *zvipfuyo* was discussed earlier in chapter 2. The glossary of all *chidzimbahwe* and other local keywords, found at the back of the book, will aid the reader.

## Chemoprophylactics in Southern Rhodesia: A Historical Overview, 1909–1973

In 1909, entomologist Llewellyn Bevan visited the Pasteur Institute in France and returned with a method of injecting *mombe* with alternating

doses of antimony salt and arsenic to kill *hutachiwana*. Bevan later found that the antimony was the key effective therapeutic agent, so antimony potassium tartrate, rather than arsenic, became the preferred chemotherapy against *n'gana*-struck *mombe* in Southern Rhodesia, alongside antimosan.[3] In 1928, the Division of Entomology even claimed that since 1909, antimony had "saved the lives of thousands of animals,"[4] a conviction that continued to govern *n'gana* policy until 1938[5]—the year that phenanthridine, the compound that eventually replaced antimony potassium tartrate and antimosan, began to be used. One such phenanthridine, dimidium bromide, was being used widely to treat *n'gana* elsewhere in Africa. Early on, homidium bromide, a methyl-substituted analog of dimidium, was shown to be an effective curative against *hutachiwana* that *varungu* called *T. congolense* and *T. vivax*. However, although it was much less toxic to *zvipfuyo* than dimidium, homidium bromide turned out to have limited use as a prophylactic (Dolan 1990). In 1946, the transition was made to intravenous application of dimidium bromide after a rather ineffectual homidium regimen—still with little improvement.[6] This marked the first time that *T. congolense* resistant to drugs had been noticed in the country.

The name *homidium* was changed to *ethidium* in the 1950s, and its derivative, prothidium, was hailed as "one of the more hopeful … newer drugs" on the market. In East Africa, when applied in *mombe* at 2 mg/kg, the drug had a six-month effectiveness.[7] Compare this with studies in Northern Nigeria that had found prothidium to have unusually short periods of protection in *mombe* (Williamson and Desowitz 1956; Stephen 1958; Stephen and Williamson 1958; Williamson and Stephen 1960). However, these negative results were set aside in Southern Rhodesia because the Nigeria studies had used "defective batches containing impurities or too high a dosage rate."[8] By 1960, however, homidium-resistant *hutachiwana* had become a "distinct possibility" (Williamson and Stephen 1960, 366).

By then, antrycide had become the most widely used drug throughout Africa and "the first piece of real progress in the chemical control of trypanosomiasis."[9] Antrycide came in two forms: (1) demethyl sulfate (DMS), which was very soluble, acted rapidly, and passed out of the body equally rapidly (in three to four weeks); and (2) chloride salt, relatively insoluble and forming a deposit in tissues, from which it slowly passed into the body over two to three months. The chloride salt was therefore prophylactic. The methyl sulfate was not; it was viable only for treating or curative purposes. These two drugs were then combined into a "pro-salt" to achieve both a curative and a prophylactic effect. The much cheaper chloride salt could still be used as a prophylactic in situations of sporadic infection by carried

*mhesvi*; pro-salt was, of course, always on standby should the challenge become too difficult.[10]

Antrycide offered a wide range of actions that few other drugs could match against the various *hutachiwana*. The DMS was absorbed rapidly once it entered the body, whereas the pro-salt was slower, the drug "lumping" or "banking" in a kind of "bag" at the injection site from which it was released over three months. The chloride in the "lump" of pro-salt was absorbed into the body system through the intramuscular or subcutaneous route. That way—the vets said—the chemical traveled virtually nontoxically, whereas intravenously inoculated drugs might poison the animal. The chloride was preferred to provide a "bank" of the drug at the site of injection for release over a period of months.[11] Treated *mombe* were easily identified by the orange-sized lump that developed at the site of injection, which often led to huge muscle abscesses. This problem had already led to falling *mombe* prices in Kenya when, in 1955, the meat inspector at Mutare "condemned quite a large proportion of rump steak from carcasses showing lumps" for that reason.[12] It turned out that the risk of a lump could be lessened by simply massaging the site of injection.

The complications of pro-salt also led to a revised formula (RF) for antrycide treatment, reducing the chloride element that did not dissolve while maintaining the sulfate radical element that did. This two parts chloride and three parts sulfate solution immunized *mombe* for over one hundred days (among exotic breeds on *mapurazi*), or even longer (over five months among indigenous breeds). Not only did it offer better prophylaxis, but RF also was cheaper than ordinary formula (OF) and induced smaller local reactions that disappeared quickly, unlike OF-induced lumps that remained even after three years. To avoid lumps and reduce incidents of "condemned meat" even further, *mombe* requiring a few inoculations could be injected on the caudal fold site rather than the neck.[13]

However, antrycide turned out to be poisonous to *mombe*. Among the toxic symptoms were increased salivation, sweating tremors, and collapse—even death—due to overstimulation of the parasympathetic nervous system (PSNS).[14] The PSNS is one of two key divisions of the autonomic nervous system (ANS) that regulates internal organs and glands unconsciously. When the body is in repose, it stimulates rest-and-digest activities, such as salivation, lacrimation, sexual arousal, defecation, digestion, and urination. The PSNS complements the sympathetic nervous system (SNS), which stimulates fight-or-flight responses. A photosensitized animal exhibited poor health, exhaustion, overheating, fright, undue exertion, and dehydration before succumbing to death. Postmortems normally revealed hemorrhagic

gastroenteritis with or without zebra markings of the large intestine, pulmonary edema, excessive fluid in the thoracic and cardiac cavities, and evidence of serious kidney damage.[15]

With antrycide's problems of photosensitization mounting, the Veterinary Department switched to samorin (isometamidium), a product of Samorin in the United Kingdom. It went on to become one of the most widely used drugs of its time against *n'gana* in *mhesvi*-infested areas of Africa. The beauty of samorin was its flexibility of deployment, as both a prophylactic and a curative. By contrast, drugs like homidium and berenil (diminazene aceturate) were solely curative. Samorin and berenil would be used as a sanative combination successfully in *n'gana* control in the 1960s (Moloo and Kutuza 1990).

By 1966, reports of drug-resistant trypanosome populations were increasing, casting serious doubts on the hopes of a drug-based solution to the *n'gana* problem. The first response to the samorin-resistant *hutachiwana* was to search for a better method of administering treatment. Vets decided to inoculate the drug intravenously instead of intramuscularly, thereby achieving an initial concentration of the drug in the blood that was significantly higher than that achieved through intramuscular administration. Under experiment, all the *hutachiwana* present in the host were eliminated (Whitelaw et al. 1991; Kinabo and Bogan 1988; Dowler, Schillinger, and Connor 1989).

A second problem was the relapse of all *zvipfuyo* treated with samorin, which demonstrated that *hutachiwana* infections had not been eliminated, nor had the demonstrable presence of *nyongororo* in the blood (parasitemia) been delayed to any significant degree. The experiments had shown that only a dosage of 2.0 mg/kg or higher offered the best opportunity for eliminating infections with *hutachiwana* resistant to samorin. Finally, it was found that the trypanicidal efficacy of samorin depended not only on the concentration of the drug to which *hutachiwana* were exposed but also on the length of exposure (Sutherland et al. 1991; Moloo and Kutuza 1990.

Berenil (diminazene) entered the Rhodesian chemotherapeutic scene as a stopper with an entirely different chemical composition from its predecessors: a diamidine used in the form of an aceturate (N-acetylglycinate). Berenil was a product of German pharmaceutical company Farbwerke Hoechst AG and by far the most commonly used treatment for *n'gana* throughout Africa. In the 1968–1969 agricultural season alone, 47,577 doses were given in Rhodesia, with not a single case of toxicity reported. Years earlier, only one beast had suffered an allergic reaction. It was discovered, however,

that berenil fatalities occurred only when it was dispensed right after samorin, as a result of changes in liver fat.[16]

The problem with berenil early on was that it was rapidly eliminated from blood through urinary excretion, even though biologically active quantities remained in *mombe* for up to three weeks after treatment. In 1977, it was discovered that berenil did not kill *hutachiwana* directly, but could instead make them available for destruction by the "big eaters" of the immune system, the white blood cells called *macrophages* (Maxie and Losos 1977, 280–281).

Interestingly, *vachena*'s experimentation with these drugs did not end with *zvipfuyo*; in East Africa, white doctors injected black patients suffering from *gopé* with a dose of berenil as a primer before treatment with melarsoprol. It was believed that the berenil would reduce the incidence of the reactive encephalopathy that often followed the use of melarsoprol alone. The physicians considered oral berenil a nonirritant, with no significant side effects even after prolonged administration. Hopes were high in 1968 that the drug could be used as chemotherapy for *gopé* as well. An experiment duly conducted on *vatema* infected with *gopé* confirmed its effectiveness (Bailey 1968, 122). I have not yet found evidence of berenil use on *vatema* in Rhodesia, but that does not mean it was not used that way.

Curiously, berenil was applied to cure the photosensitization and drug-resistant *hutachiwana* that samorin had created, only for berenil itself to induce diminazene-sensitive and diminazene-resistant trypanosome infections in domestic *zvipfuyo* later (Aliu, Mamman, and Peregrine 1993). A study in 1981 then found berenil to be highly toxic to camels, with the main signs of poisoning being hyperesthesia, salivation, intermittent convulsions, frequent urination and defecation, itching, and sweating. It also found damage to the liver and concentrations of ammonia and decreases in the concentrations of calcium and magnesium in serum (Homeida et al. 1981). Indeed, berenil summarizes the double role of the trypanicidal drugs as *mushonga* (medicine) and *chepfu* (poison) all at once—even more so when *mhesvi* drew blood containing such drug-resistant *hutachiwana* and spread them!

## Experiments in Drug Resistance

The experiments that determined the "facts" about these drugs were conducted at three stations. The first research started at Lusulu Field Research Station after the discovery of antrycide- and berenil-resistant *hutachiwana*

in 1963. The early research tested for effective trypanicidal drugs and the mechanical modes of transmission of *hutachiwana*, including and excluding an animal medium.[17] It focused on stock responses to drug treatment "under conditions of high trypanosomiasis risk"[18] and the development of drug resistance.

Observations during 1965 confirmed that administering samorin to all stock for one year "successfully eliminated" the antrycide-fast strain of *hutachiwana* that was now firmly established in *mombe* "and which was evidently being transmitted by the tsetse fly." Further trials showed that the drug, administered at 1 mg/kg dosage, could "completely control" *n'gana*. Even a 0.5 mg/kg dose at the start of the disease had been shown to offer a two-month protection.[19]

The early optimism evaporated in 1966 when further trials showed samorin to have "unpleasant side effects," described as follows in the director's 1966 annual report:

> It produces extensive muscle destruction with replacement fibrosis. Injections are given into the neck of the animal on each side. The contraction of the scar tissue has produced in some animals a condition resembling opisthotonus. Two animals have to kneel to crop short grass, but despite the disfigurement they are in satisfactory condition. In the animals that have received a prolonged treatment the replacement fibrosis is rendering it increasingly difficult to find suitable sites for the intra-muscular injections. After two or three treatments into the neck muscles, the extent of the replacement tissue renders the neck valueless as meat. Apart from this drawback, the drug at 1.0 mpk (milligrams per kilogram) and 2.0 mpk at two and three monthly intervals has successfully maintained cattle at Lusulu, where they have been exposed to an estimated 4.5 infected bites per hour.[20]

The experiment also showed that 56 percent of the infections happened one hundred or more days after treatment—"an unexpectedly prolonged period of protection."[21] Very heavy doses of 2.0 mpk at three monthly intervals in *zvipfuyo* carrying *hutachiwana* highly resistant to antrycide had proved "quite successful": 223 days of protection from 33 to 506 days after dosing. However, the trials were stopped rather abruptly when samorin-fast strains of *hutachiwana* developed. Twenty-two *mombe* died of samorin treatment, displaying distinctive lesions.[22]

By contrast, berenil at 3.5 mpk at two-month intervals kept *zvipfuyo* healthy for three years and was declared successful, albeit causing parasitaemia in the fourth to fifth week after routine treatment, which then vanished for a while after the next treatment. The 1967 experiments were conducted to determine the toxicity of samorin and berenil following illness and mortalities at the station among experimental *mombe* undergoing

long-term trials with the two trypanicidal drugs.[23] As we will discuss later, the drug-resistance problem would persist in the two chemotherapies, leading to a search for alternatives.

By the end of 1964, veterinary staff at Rekomichi Field Station had started conducting trials with three groups of *zvipfuyo*, divided according to the drug they were being administered—that is, antrycide, ethidium, and berenil groups. The first group was on antrycide pro-salt RF and berenil, monitored for drug resistance. The second group bore a homidium-resistant (i.e., ethidium-resistant) *hutachiwana* strain that broke out in June that year, leading to the immediate termination of ethidium and the switch to berenil 7 mpk every fourteen days. The third group was divided into two, for immediate and delayed treatment with berenil, both administered to adult *mombe* and calves. Besides *mombe*, *nguruve*, *mibhemhe*, and *makwayi* were also experimented on.[24]

The 1965 trial sought to prove whether "the extended period of aparasitaemia which develops after prolonged treatment of cattle with a prophylactic drug is due to immunity and not to an accumulation of the drug in the tissues or body fluids."[25] In February, the Food and Agriculture Organization (FAO) immunologist M. A. Bolton examined the immune response in *mombe* to a number of drug treatments, including ethidium and berenil.[26] The trial was slated for eighteen months to allow immunity to develop in *mombe* under drug administration; afterward, the treatment would be stopped and the *mombe* removed to join others. Their differential behavior relative to continued exposure to high *mhesvi* risk would prove whether immunity had developed. Ethidium was chosen because berenil was the sanative (restorative drug) in the event ethidium-resistant *hutachiwana* developed. Because berenil was already being used, there was no risk of interfering with results in other ongoing experiments at Rekomichi, as there might be with a new drug.

The following year, the experiment was continued with two groups of five *mombe* each maintained at Gwebi Research Station and two similar groups at Rekomichi. At both stations, one group was kept on berenil, the other on ethidium, each at similar dosage rates, and each subjected to repeated infections. The results from the berenil group were consistent with the hypothesis that "repeated infections confer a form of immunity." The ethidium group showed that "the prolonged use of ethidium under conditions of repeated infection" did not result in any sustained immunity after withdrawal of treatment as antrycide did.[27]

Like at Lusulu, Rekomichi Research Station also conducted tests on the toxicity of samorin when used in combination with or after berenil. Another herd of bulls and slaughter cattle was also maintained under ethidium

prophylaxis and berenil. At Rekomichi, the experiments also extended to the pathology and immunology of *makwayi* and *mbudzi*, the latter an indigenous flock maintained since 1965. It was divided into two: one receiving no prophylactic treatment, another berenil therapy. Just four of the ten untreated survived, forcing the staff to dose them with berenil.[28] Donkeys, by contrast, were maintained in perfect condition under samorin. *Imbwa* responded adversely to berenil, with many succumbing to side effects; the use of the drug became "to say the least a somewhat controversial subject," even though the work continued because of the ethics of subjecting *imbwa* to certain death.[29]

### Case Study: The Eastern Districts and Drug-Resistant Chemoprophylactics

The Mkota communal land is in the Mudzi district, right on Zimbabwe's northeastern border with Mozambique. It was in that area that the first major trial of dimidium bromide, involving block inoculation of 4,723 head of *mombe*, was conducted in 1948. After four months, just five head were discovered to have died, and just two had contracted *n'gana*. The conclusion was obvious: a huge success without adverse effects.

The following year, the drug was extended to Chikwizo, also along the border but south, almost on the boundary of Nyanga District. Here, 11,300 *mombe* were block inoculated, "mortality being immediately arrested and no ill effects … observed." Further outbreaks in 1951 were dealt with decisively using dimidium bromide, with 20,000 *mombe* block inoculated, even though "doubt was being expressed as to the future efficacy of the block inoculation in view of so little being done to prevent the tsetse fly itself becoming fully established in the areas concerned."[30] In both cases, *mhesvi* was coming from Mozambique, constituting the western edge of a long *mhesvi* belt that extended into Catandika, Gorongosa, and all the way to Mtarara.

The infections resurfaced in Mkota in 1951. This time, when vets treated *mombe* with dimidium bromide as before, the *mombe* did not respond; in fact, one hundred *zvipfuyo* were dying every month.[31] A new problem arose. Twenty cases of photosensitization were recorded in 1952, but inoculations of dimidium bromide continued. In March 1953, as the casualties mounted, the government decided that the remaining 250 *mombe* should be slaughtered and arbitrary compensation paid. By the end of 1953, only 254 out of 4,723 *mombe* inoculated in Mkota had survived. Meanwhile, the repeated inoculations—twenty-five to thirty times at two-week intervals—had created a super-resistant *hutachiwana*. Test after test after such inoculations was positive, with relapses the order of the day.

To solve the problem, the veterinarians increased the dosage strength. The relapses continued.[32]

Desperate, the veterinarians switched to antrycide, but it was just as bad in terms of drug resistance. Attention was redirected to the trial of ethidium bromide; it too failed to cure *n'gana* and had the same relapse rate as dimidium bromide. Further trials showed that antrycide was effective in curing cases resistant to ethidium and dimidium bromide. In 1954, twelve thousand dimidium inoculations were carried out, but they only resulted in a high number of photosensitization cases (10 percent of the herd).[33] In 1955, thirty-six thousand inoculations were undertaken with dimidium, resulting in 2,326 deaths and two hundred cases of photosensitization.

By 1956, it had become customary for *varungu* to praise themselves when everything went well but blame *vatema* when things went wrong. The shortage of white staff meant that trained *vatema* were deployed to inoculate *mombe*; they became the scapegoat for the "toxic symptoms [of] overdosage … or too frequent dosing" in Mkota. Said one veterinary officer that year: "Admittedly a professional officer did supervise the block treatments, but the native inoculators had access to further supplies of dimidium, which no doubt they used (and sold) on the same animals many times after the block inoculation." The officer also found that "resistant strains … negatived [*sic*] any therapeutic effect of the drug and in fact probably speeded up its toxic effect by overdosage."[34] The "native inoculators" were relieved of their duties, but the mortalities still continued.

By 1958, some seventy thousand head of *mombe* were under prophylactic and curative drugs throughout Southern Rhodesia. The inoculation campaign was operated at *madhibhi* (cattle dip tanks) (see figure 14.1), with each stockowner issued a stock card to be presented to the dipping attendant every dipping week. The attendant was supposed to tally the numbers of *mombe* physically presented for dipping with those indicated on the cards. Then, the beasts were driven into the plunge area and, as they exited, were injected with the drug using a syringe. Alternatively, a proper dose was poured through the mouth using a Coca-Cola or Fanta bottle. The dipping attendant (or vet) then marked the card, and off the stockowner went. In the absence of dip tanks, operated by *vatema* on the Native Department's payroll, it was virtually impossible for the vets to dispense drugs.[35]

In some cases, the treatments took place at each homestead's *danga* (kraal) full of *mombe*. The animal to be inoculated had to be cast (brought down by tying and pulling its legs in different directions to unbalance it) amid "a permanent cloud of dust." The white vets' characterization of the

**Figure 14.1**
A typical dipping tank in Zimbabwe, as it was during the days of Rhodesia.
*Source:* Author 2016.

*vatema* as "usually inexperienced, frightened or frankly lazy" is inconsistent with the depth of *ruzivo rwemombe* (cattle knowledge and practice) we discussed in chapter 1.[36]

As the number of antrycide-fast strains of *T. congolense* increased, the Veterinary Department decided to switch to berenil as a sanative and ethidium as a routine curative. Ethidium turned out to be effective; for three months, there were no infections.[37] Without discounting the drugs' efficacy, it is possible that the improvement was also a result of insecticidal sprays, which drastically reduced *mhesvi* catches at Makaha fly chamber in Chikwizo from thirty-four in 1963–1964 to just seven in 1964–1965. Nonetheless, the dramatic reversal of explosive *n'gana* situations at Chikwizo Centre and Zano owed much to the use of berenil.[38] At the latter center, berenil curative treatment slashed the cases from 170 in 1967–1968 to a mere fifty-six in 1968–1969. By 1970, the *n'gana* situation had stabilized in Chikwizo and Mkota.

Just south of Mudzi, the Nyanga North chemotherapeutic campaign got under way in 1958, essentially in response to the spillover of *mhesvi* and

*n'gana* from Chikwizo. By chance, a Tsetse Department employee coming back from leave in his village captured one *mhesvirutondo* as it tried to bite him. Even after this early warning, the Tsetse Branch did not post regular patrols. Two years passed. Then in January 1960, in a community called Fombe, several outbreaks of *n'gana* took place barely a month after antrycide treatment.

In response, the vets dispensed sanative treatment with berenil and followed it up with antrycide pro-salt prophylaxis. However, twenty days later, twenty-four *mombe* tested positive to *n'gana* again. Based on the Mkota and Chikwizo experiences, *hutachiwana* were now known to be resistant to antrycide, so the switch was made to inoculation with samorin. It worried the authorities that the *mhesvirupani* density in Ruenya was so low, and yet the risk of *hutachiwana*—never mind one that was becoming drug-fast—was so high.[39]

The position deteriorated even further in 1961—not just at Fombe, but also at Mandeya, Chimusasa, Chifambe, and Matisi. In the first two centers, all *mombe* were subjected to a bimonthly prophylactic regimen of antrycide pro-salt. Chifambe and Matisi were placed under review due to outbreaks of civil disobedience among *vatema*, who rejected any sort of diagnostic or therapeutic measures, which they blamed for the miserable deaths of their *mombe*. Two hundred died untreated. In Mandeya, where twelve cases had been diagnosed, *vatema*'s civil disobedience was so serious that police had to be called in and arrests and prosecutions made. In Nyanga North and Holdenby, 13,032 *mombe* were treated, 1,353 of them with prophylactics and 11,679 with curative drugs. Out of the 10,527 inoculations, 5,036 were antrycide DMS, 954 antrycide pro-salt, and 4,537 berenil.[40]

*Vatema*'s refusal to cooperate in producing their *mombe* for treatment at *madhibhi* continued throughout Nyanga North, barring Fombe, Chimusasa, and Chapferuka. The political temperature in the urban and rural areas of Rhodesia was rising. The Federation of Rhodesia and Nyasaland had ended in 1963, with Northern Rhodesia (Zambia) and Nyasaland (Malawi) being re-Africanized as independent states. However, the *vadzvanyiriri* (downpressors, or white settlers) in Southern Rhodesia refused to hand over power to black people and moved instead toward a Unilateral Declaration of Independence (UDI) from Britain to form the Republic of Rhodesia. Already in 1964, "African nationalist" leaders had been rounded up and dumped in remote areas, where they were held in detention camps, but with freedom to hold rallies within a fifty-mile radius. They were accused—not without truth—of inciting the rural population to civil disobedience. In particular, they urged *vatema* in rural areas to boycott dipping their *mombe*, which

was the most important mechanism through which vets could inspect and inoculate *mombe*.[41] It was such threats that led the government to unleash the Law and Order Maintenance Act (LOMA) on the rural countryside in 1965, which coerced *vatema* to capitulate to chemotherapy.

Even on veterinary grounds, it was considered unbeneficial to carry out immediate treatment, because the drug resistance had become so widespread. Two months were needed to choose and field-test drug regimens to control the various strains of trypanosome not responding to treatment. Ultimately, the vets turned to samorin.[42] It seemed for a brief period that the drug had solved the problem.

Unbeknown to the vets, samorin was about to become a problem as well. In June 1966, the animal health inspector for Nyanga reported at least forty deaths in *mombe* at the Chifambe, Samakande, Manwere, Nyamasara, Chipatarongo, Ruvangwe, Fombe, Nani, and Matisi inoculation centers. His postmortem indicated extensive liver damage strongly suspected to stem from the drug samorin. The *mombe* had been treated in the week starting May 9, with berenil inoculation ten days later. After three weeks, the deaths started. By June, the death toll had risen to 152 *mombe*, with 154 more infected and "likely to die." At Chipatarongo, one owner lost five or six *mombe*, others none—a reflection of the tendency of the strain to be concentrated at certain cattle kraals.[43]

Along with a "capricious appetite," diarrhea was perhaps the most immediately recognizable sign of infection, with the beast suffering a running stomach for four to seven days before dying. In its final hour, the bovine "appeared tucked up, dehydrated, weak and thin," its temperatures subnormal. Its digestive system would also be in "ruminal stasis," the feces "hard dry and mucus-covered."[44] Many of the *mombe* had a "crusting around the eyes," evidence of excessive lacrimation. A few of them showed increased nasal discharge and excessive salivation. Some that were sick had recovered; others had not. The postmortem confirmed the lesions to be "a massive acute fatty degeneration of the liver" and the kidney.[45]

The laboratory tests took months to run. By the time prophylaxis resumed in April 1967, the reinfections had drastically fallen due to two factors. One was the prolonged protective effect of a series of samorin treatments; the other was the drastic reduction of *mhesvi* due to the 1966 spraying operation. Although the department could not distinguish the exact contribution of each factor, the cumulative effect was that no significant *n'gana* outbreak occurred until January 1967. Thereafter, bimonthly sanative berenil treatments were religiously applied until April, when a reduced 0.25 mg/kg dosage of samorin treatment commenced, which did not cause photosensitization.[46]

The weakened regimen covered Nyanga, Matisi, and St. Swithun's. First the Fombe, Chimsasa, and Chapferuka centers and then, from December 1967, Chifambe, Samakande, Manwere and Mangezi were treated with this drug. From March on, berenil was incrementally deployed as a sanative. The *n'gana* situation was brought under control temporarily, only for the infection to resurface in September.[47] Berenil was also effective in eliminating the mild outbreak at the Matisi and Nyamasara centers. In Sawunyama, the use of berenil was so effective that only thirty-four cases were recorded in 1967–1968, compared to a staggering 230 in 1966–1967. Meanwhile, at Matisi, Mangezi, Chifambe, and Samakande, just seven cases were recorded.[48]

*Mombe* in Nyanga District had maintained their condition well. "Cooperation from the local stock owners was of a very high standard," BTTC Assistant Director Gerald Cockbill commended. Prophylaxis with samorin was maintained at Chimsasa and Fombe until July, when the last treatment was given. Drug cover was then withheld from *mombe* at these centers so that incidence of *n'gana* could be used as an indication of the effectiveness of the 1969 spraying operation. Elsewhere in the central area, satisfactory control had been maintained by use of berenil on the individual *zvipfuyo* or on infected herds in areas such as Samakande.[49]

When *n'gana* broke out in Musikavanhu in Chipinge in 1954 and antrycide and dimidium bromide were deployed as the prophylactic and therapeutic, respectively, to contain it, everything seemed to be going smoothly for the Veterinary Department.[50]

To avoid the "human error" that it blamed for the buildup of drug resistance in *hutachiwana*, the department stripped all "native assistants" of the role of dispensing the medicines and closely supervised their activities. However, the deaths continued, with the blame now being shifted to the drug itself and the *mombe* that had grazed on toxic lantana types.[51] The director of Veterinary Services found that "in the course of time one or both jugular veins became practically occluded due to phlebitis as a result of injections being badly placed, or leakage of the irritant drug from the vein after withdrawal of the needle."[52] Some "experts" warned that the real nature of dimidium's toxicity would probably never be known. It was not clear if fresh grass prevented or worsened the drug's toxic effects, or whether in fact it was the lack of grass in November that caused heavy mortality in Chipinge in 1955. Rift Valley fever was known to exist in those *mombe*, which may have worsened the liver damage—but then, similar liver damage in other areas had nothing to do with Rift Valley fever.

The postmortem findings at Salisbury Central Laboratory revealed extensive peeling of teats and udders, enlargement of the liver and kidneys, and increased blood flow and accumulation of fluid in the lungs.[53] At the end of the day, dimidium bromide was considered to have so many unpredictable characteristics when specifically applied to Rhodesia that it had to be discontinued except in areas of small groups of *mombe*, such as *mapurazi*; ethidium bromide took its place. By 1958, dimidium bromide generally had fallen into disuse in Africa "due to a type of photo-sensitisation leading to liver and other tissue damage."[54]

After antrycide pro-salt was authorized for use in Chipinge, 13,517 head of *mombe* were inoculated. The 177 deaths that followed were blamed on the excessive dosage, which had caused emaciation, extreme dehydration, and internal bleeding. The lymph nodes were enlarged, edematous, and hemorrhagic. One animal had bled extensively into the duodenum and small intestine, where large clots were found during postmortem. To solve the problem, the dosage was reduced.[55]

Subsequently, several recommendations were made: First, a rest period of two hours should always be observed before and after inoculation. This involved watering *mombe* before the commencement of the inoculation and shade afterward. Second, *mombe* must not be beaten or chased into the inoculation arena, but walked in gently. Should some beast run away, it was not to be chased and brought in for inoculation kicking and mooing, but must be returned under calm conditions, after the meek ones had been treated. Third, all inoculators were supposed to be familiar with the live weight estimation of the beasts they were working with.[56]

## Conclusion

By 1973, only ethidium, berenil, and samorin were listed as treatments. Hurungwe had become ethidium and berenil country; Lupane, Hwange, Kadoma, Hurungwe, Nemakonde, Rushinga, Centenary West, Mutoko, Nyanga, and Chipinge had become exclusively berenil country after the resistance to antrycide and photosensitization problems with samorin; and Binga, Gokwe, and Guruve were berenil-samorin combo country.[57] All told, Rhodesia's *mhesvi*-prone districts were almost equally split between berenil and samorin, with ethidium used only in token quantities.[58]

Meanwhile, investigations were under way to determine whether a 1:1 mixture of berenil and pyrroli-dinomethyl-tetracycline (reverin) was efficacious against *n'gana*. A 1973 experiment found that this mixture could cure *hutachiwana* infections in *mombe* if administered at a dosage rate of

2.5 mg/kg of the berenil fraction. The experiment was simultaneously undertaken at Central Laboratory in Salisbury and at the three BTTC field stations—namely, Lusulu (Binga), Rekomichi (Hurungwe), and Gwebi (outside Salisbury).[59]

At this point, the thinking in the Branch of Tsetse and Trypanosomiasis was that chemotherapy was only an interim measure to "maintain the health of cattle until such time as the vector is eliminated."[60] The primary enemy of both the glossinologist and the veterinarian was *hutachiwana*, especially what *vachena* called *T. congolense*, the chief *nyongororo* of cattle. This microbe had thus far, after seven decades of combat, exhibited "a remarkable facility for antigenic change that makes it resistant to the host's antibody and its ability to develop drug resistance."[61] As an enemy, it was a nifty, ever-shifting target, "capable of altering its sensitivity to hazardous substances or detrimental material introduced into its milieu to such an extent that it becomes insusceptible." Consequently, the *nyongororo* shifted the matter of success or failure of *vanhu*'s assault upon it away from the strengths of *hurumende yevadzvanyiriri* (the government of the downpressor) and blamed it on two factors: "the lack of a vaccine and the frequent appearance of drug fast strains of trypanosomes."[62] In both, the Rhodesian state was vulnerable.

No other country in *mhesvi*-occupied Africa had executed chemotherapy and chemoprophylaxis to the extent and with the efficiency that Rhodesia had. The stoic persistence in maintaining *mombe* in areas with the greatest *mhesvi* challenge had astonished its critics who cautioned about drugs that started with much promise, only for *hutachiwana* to get used to them and resist their effects. The Rhodesians beat their chests, crowing that they had succeeded in using *mishonga* against *hutachiwana* since 1950. Yet that view did not make anything of the suffering of those that lost entire herds in the name of experiments the outcomes of which, they dreaded, would only bring social, spiritual, and economic ruin as their *mombe* breathed their last. *Hurumende* did not care, because *mombe* and the lives it was experimenting with were of *vanhu vatema*, not *vachena*. In its approach, *vatema* and their *mombe* were not only *midziyo* (instruments) for clearing lands of *mhesvi* and keeping *hutachiwana* from getting in. They were also experiments in a vast laboratory that was *ruzevha* (the Native Reserve). Indeed, in experimenting with the one thing that was so central to *vatema*'s livelihoods, wealth, and social being, *hurumende yevachena* was experimenting with *vatema* themselves.

# The Mobile Workshop

## The Tsetse Fly and African Knowledge Production

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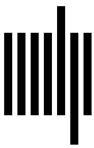

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