Author: Henry James Hammond

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Professor: Rudolf Mrázek

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The Rise of Beriberi and the fall of Colonialism

Southeast Asian communities have sustained themselves on rice for millennia. Rice paddies dominate much of the landscape, and the cultivation of this rice in turn dominates the lives of many Southeast Asians. The centrality of rice to Southeast Asia is nicely represented in Carpenter's observation that in Malay, "the word *nasi* is used for food in general and for cooked rice in particular. In Thailand, the general term meaning 'to eat' translated literally means 'to eat rice'"(Carpenter, 15). The processing of rice changed dramatically in the 1870s with the advent of power-driven steam mills that made it possible to produce refined white rice cheaply and efficiently. Since white rice stores longer than brown rice, colonial governments used white rice preferentially in their institutions, and a large-scale dietary shift to white rice consumption ensued in colonial population centers. However, removing the rice hull to process white rice also removes many essential nutrients from the rice, including Vitamin B1, also known as thiamin. Vitamin B1 deficiency causes beriberi, which is a debilitating and if untreated, fatal, ailment. Large-scale production and consumption of white rice, made possible by power driven mills, resulted in an explosion in beriberi in Southeast Asia in the late 19th century. In 1896, Christiaan Eijkman serendipitously identified the cause of the disease as a nutrient deficiency associated with consumption of white rice. Even then, Western scientists continued to search for a cure for beriberi, failing to recognize or pursue the connection of the epidemic to colonial rice milling. The specific deficiency involved was not identified until the 1930s, and it was not until 1947 that Western scientists developed techniques for enrichment of white rice with thiamin that effectively ended the beriberi

epidemic. The history of this epidemic, including its ironic role in both threatening colonial ideology as well as prolonging it, offers a window on that ideology and its practice in Southeast Asia.

Beriberi threatened colonial rule both not only because it crippled colonial military forces, thereby hindering the enforcement of colonial dominance, but also because the epidemic, which was recognized even then as affecting primarily those integrated into the system, called into question the belief that colonial rule improved the lives of the colonized. Ironically, this conceit of Western superiority blinded colonial scientists to beriberi's real cause, gave those scientists a false impression of their control over the disease, and prolonged the epidemic until the colonial era had already come to an end.

Beriberi is a devastating and painful neurodegenerative disease. Robert Williams, who helped end the epidemic when he developed a technique to enrich white rice, describes the symptoms characteristic of the onset of beriberi, which begin with a growing numbness of the legs that:

> becomes an exquisite tenderness of the muscles so that severe pain is elicited by pressure on the calves…. [T]he muscles shrink and waste away and the gait is extremely lame….[T]enderness may extend to the arms so that feeding and dressing oneself becomes almost impossible. The victim ultimately becomes a helpless and shrunken skeleton (Williams, 62).

Despite Williams' euphemism 'tenderness', the creeping paralysis he describes must have been nightmarish, particularly for young colonial officers who found themselves incapable of moving their legs and, with time, unable to move their arms. J. Ridley, a British Army Surgeon serving in Ceylon in 1813, oversaw an area in which a serious outbreak of beriberi was killing five to eight men every day. One can only imagine his

anxiety when he awoke "'…with a sensation of tightness, as if a bar were placed across my breast…. I found my legs and feet perfectly numb and swollen, the space around my mouth, reaching nearly my eyes, felt numb'"(Carpenter, 26). Ridley recovered in full upon his return to England, but the soldiers he left behind died. Such continuous loss drained colonial armies of their manpower.

For the European armed forces, beriberi represented a grave threat to colonial dominance in Southeast Asia. Carpenter remarks, "For the administrators of the European colonies in Southeast Asia towards the end of the nineteenth century, one of the worrying things was that the disease [beriberi] was a particularly serious and increasing problem among men in their employ, whether as soldiers or civilians"(Carpenter, xi). That is to say Beriberi imperiled the colonial state apparatus. Charles Smart, a Surgeon General in the US Army, reported in 1903 that "'The 39th and 44th companies of Philippine Scouts were the sufferers [of beriberi] at Iloilo […] In the third week of the quarantine the first case of beri-beri occurred, and soon afterwards one-third of the company was affected'"(Brandon, 103). The Dutch campaign against the Sultan of Aceh highlighted beriberi's ability to incapacitate a colonial fighting force. In 1886, an offensive against the Sultanate of Aceh was suspended in response to the "alarming increase in cases of beriberi among soldiers and sailors"(Carpenter, 32). When the campaign resumed, beriberi continued to decimate the troops. A table from *Koloniaal verslag* published in 1894 shows that in 1893 an average of 500 soldiers died every month from beriberi, and an additional 500 soldiers were evacuated, so that casualties from beriberi exceeded 1,000 soldiers a month, for a total of 13,096 casualties in 1893 alone (Hellwig & Tagliacozzo, 2009).

In addition to threatening the apparatus of colonial rule, the beriberi epidemic of the colonial era also flew in the face of the ideological principle upon which the colonists relied to justify their occupation and exploitation of foreign lands, that is to say, that they were improving the lives of the indigenous people. Beriberi was most prevalent in colonial-run institutions such as jails, asylums, hospitals, armies, and navies where the colonial institutions supplied white rice. Thus it was apparent that colonial rule was in fact detrimental to the health of native populations. In his 1913 book detailing beriberi, Captain Edward Vedder of the US Army Medical Corps remarked that, "Beriberi is, moreover, principally a disease of large towns, usually the low-lying ports of the city being principally attacked. Occasionally, certain centers and certain buildings are specifically attacked particularly jails, barracks, and hospitals"(Vedder, 136). Williams, writing in 1961, also noted the connection between colonial centers and beriberi:

> All who have read the early Dutch studies of beriberi have been struck by the fact that, at its worst, its appearance was limited to jails, armies, navies, and institutions where large numbers of persons were fed from a common kitchen. While beriberi had occasionally appeared spontaneously among the Javanese people in their homes, several of the Dutch writers of last century [19th century] emphasize that it never was a disease prevalent in the native villages where the life of the people had been little influenced by Western industry and culture (Williams, 230).

These descriptions of beriberi's distribution make clear that the disease was recognized to be most common in parts of the country under direct colonial administration where mass-produced white rice was principally consumed. Moreover, the incidence of beriberi increased in the areas of greater colonial control, such as large port towns and government run institutions. These facts raised the following question: why, if colonialism improved the lives of indigenous peoples, were those living

traditional lifestyles left unaffected by the epidemic while those who followed the lifestyle patterns of their colonial overlords the ones who suffered most?

Ironically, the observation could have led to elucidation of the etiology of the disease, and dietary solutions—such as were long practiced for scurvy, which was recognized as a dietary deficiency, and "solved" by consumption of foods supplying Vitamin C long before its specific cause was identified. In fact, in 1890, C.O. Gelpke, a Dutch medical officer, wrote that beriberi should be called

> 'the government disease' and … suggested that natives in their villages remained free from beriberi because they stored their rice crop in the husk, and hand-pounded only enough for their immediate needs, whereas in government establishments milled rice was kept for long periods before use (Carpenter, 42).

Gelkpe here implicated western technology, the West's gift to its colonies, as the cause of beriberi, and unknowingly suggested a way to end the epidemic: return to the native Southeast Asians' traditional methods for storing and processing rice. Gelpke's analysis, however, was ignored by a society entrenched in its ideology that colonization surely benefited local peoples.

Colonial scientists appear to have been so loyal to the ideological principle of colonialism as a beneficent force that they could not accept the possibility that colonial technology had caused the epidemic. Moreover, their belief in the efficacy of Western science, germ theory and Western methods of quantifying disease gave them a false sense of control over beriberi. This mindset ultimately prolonged the epidemic.

In 1878, William Anderson, a British doctor, began his report on beriberi to the Asiatic Society of Japan with the statement that beriberi " is peculiar to the East and is unlikely to excite much interest in Europe and America"(Anderson, 155). The false perception that beriberi was an exotic disease of the Far East resulted from the conceit

that the tropics were an unsanitary region requiring the aid of Western science. Beriberi's supposed exotic nature is apparent in its very name. There is no single dominant theory about the origin or meaning of the name 'beriberi,' but all explanations connect beriberi to an ancient past in tropical Asia. Jacobus Bontius, a Dutch doctor, proposed that 'beriberi' was derived from a local Javanese word for sheep because the victim's weak gait resembled that of a sheep. Carpenter states that some of the alternative explanations have been, "the Hindi word *bharbari* (swelling); or the combination of the Arabic words *buhr* (shortness of breath) and *bahri* (marine)" (Carpenter, 25). Dr. Sarat Ghose, a physician in Calcutta, reinforced the connection between beriberi and Asia in his book published in 1910: "The name Beri-beri has been given to this malady by Nalanban—singalese for weakness and the repetition means extreme weakness. … [I]t is a malady known to the people of the greatest antiquity and was prevalent in China from an extremely distant period"(Ghose, 1-2). Thus, the only consensus among these etymological theories is that this mysterious word originated in the East, reinforcing Western beliefs that beriberi was a fundamentally Asian disease with an etiology unique to the tropics.

Captain Edward Vedder, though well aware in 1913 that beriberi was a nutrient deficiency disease and not restricted solely to Asia, not only accepted a historiography of the disease tying beriberi to ancient China but also ignored the relationship of the epidemic to the introduction of the rice mill. He postulated "Chinese customs and conditions of life have changed little with the passing of centuries, and were probably much the same then [2697 B.C.] as they are now. We should expect that beriberi, a food disease, would be prevalent then exactly as it is now"(Vedder, 1). Writing in 1913, after

Eijkman had established that the disease was related to white rice consumption, Vedder nevertheless could not see that, while rice had been the staple crop of Asia for millennia, its nutritional quality had been significantly reduced under colonial rule. In this way, he avoided acknowledging that the epidemic was fostered by the colonizers and could instead conceive of beriberi as an endemic plague that the white colonists valiantly set about to conquer.

Early theories on beriberi's cause reveal not only a similar insistence that beriberi was a peculiarity of tropical Southeast Asia but also an excessive enthusiasm for Western germ theory. In his book, Braddon presented three theories of beriberi's etiology that were prominent in 1907. "Wright's Theory of Dirt Infection," according to Braddon, was that victims pick up and carry a germ living in the dirt (Braddon, 26). Durham, according to Braddon, presented a similar theory that a microbe living in the ground only in the tropics caused beriberi. Finally, Braddon proposed the "Pure Miasma Theory" that "[t]he extrinsic cause […] a mold, a microbe—produces a toxin—dust, liquid, a gas—the continual absorption of which, disseminated or spread through air into the system produces beriberi"(Braddon, 32). While these theories differ in their particulars, they all espouse the idea that there is some contagion particular to Southeast Asia causing beriberi.

Braddon rejected those theories, asserting instead that stale rice produces a toxin. However, this theory also specifically linked beriberi to the rice-eating people of Southeast Asia and it did not consider the role of Western food technology in causing the epidemic. Indeed, the idea that beriberi was an ancient tropical disease and that the epidemic had nothing to do with colonial development was so entrenched that even

Eijkman, the first to classify beriberi as a nutritional deficiency resulting from the consumption of milled rice, thought for a period of time that beriberi resulted from "infections present in Indonesia which were acquired, in combination with poor diet" (Carpenter, 53). Eijkman was also predisposed to believe that there was some contagion in Southeast Asia that caused beriberi, suggesting that Western scientists' investigations were shaped by colonial prejudices that perverted their insights.

The resistance to the idea that beriberi was simply a nutritional disease like scurvy, rather than a tropical disease reflecting something sinister and unsanitary about Southeast Asia, reflects these colonial prejudices. John Malcolmson, the Assistant Surgeon General in Madras, stated in an 1835 report that "[t]he easy circumstances of many of the native soldiers who suffered [from beriberi] are fatal to any supposition of the disease depending on deficient and unhealthy diet"(Malcolmson, 42). Eijkman only determined the nutritional cause of the disease when he noticed that laboratory chickens fed a diet of milled white rice developed symptoms of beriberi, and recovered when they received unshelled rice. Other scientists, however, resisted Eijkman's theory. In 1910, Dr. Ghose wrote, "we will be fairly convinced that these conditions [eating milled rice] prevail over vast areas of India, but epidemic dropsy, or beriberi is, by no means, a widespread malady"(Ghose, 25). Long-lasting dissent to beriberi as a nutritional disease reflects incredulity that under enlightened European rule there could possibly be a dietary deficiency. Notions of Western cultural superiority thus prevented western scientists from seeing what now seems obvious: colonialism caused the beriberi epidemic.

Confidence in the West's scientific superiority also convinced colonialists that applying Western methods allowed them to control and understand beriberi. While evaluating Japanese medical descriptions of beriberi, Anderson remarked:

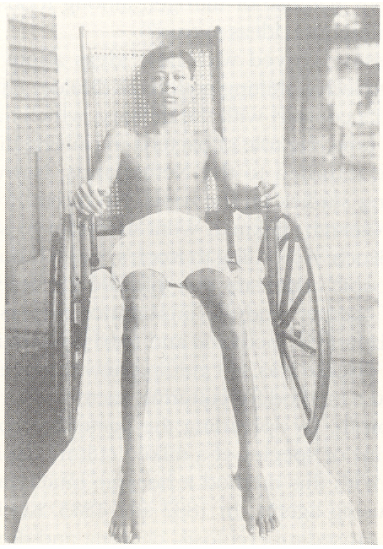
> The description by Tachibana Nanke, which is still considered the best in this country, is very curious and interesting, but its usefulness is greatly limited by the groping after tortuous explanations of natural and morbid phenomena in the misty influences of the *in* and *yo*, and the complex interrelationships of five pseudo- elements of Chinese philosophy to the neglect of close observation and scientific research (Anderson, 156).

Anderson's distaste for non-Western medical theory is evident in this quotation; his concession that Nanke's description is the best in Japan only reinforces his patronizing dismissal of what he considered pseudo-scientific medical theories. Moreover, Anderson's emphasis on the importance of "close observation and scientific research" implies that Westerners can understand and gain control over beriberi through the simple application of the scientific method. There is also more than a hint here that Westerners, armed with scientific knowledge, are more fit to rule than Asians mired in superstition. However, the irony in Anderson's remarks is that, as described above, Westerners propounded many pseudo-scientific theories about beriberi while they failed to control the epidemic and only gradually groped their way toward an understanding of the disease.

Meanwhile, colonial scientists relied on the trappings of Western science, quantitative measurement and documentation, to give the impression that they both understood and controlled the disease. Almost every primary source analyzed for this paper contains extensive tables and graphs documenting the incidence of beriberi in different populations. The table *Casualties from Beriberi in the Aceh War, 1893* from the *Koloniaal verslag* counts victims by race—European, African, Ambonese, and indigenous people—month, and whether the patient died or was evacuated. What the

table also shows is that the Dutch—rather in the manner of whistling in the dark to convince them that they were really, as befitted the colonial masters, in control—conflated measuring the disease with controlling it. Braddon's book similarly contains an intricate foldout graph that tracks average rainfall, case-incidence of beriberi, mortality, and Chinese rate of sickness, at thirty-one stations over twenty years (Braddon, 336). The graph reveals how Braddon carefully, keeping with the best traditions of Western science, examined the incorrect variables when trying to understand the epidemic. Perhaps intentionally, it also gives an impression of Britain's extensive power in the Malay and reinforces the sense that colonial rule was a well-established, almost pre-ordained, feature of the Malay Peninsula.

This idea of control through scientific technology also pervades photographs taken of beriberi patients during the epidemic, which seek to emphasize Westerners' supposed control of beriberi through science as well as their colonial subjects. In theory, photographs objectively portray their subjects, and present an ideal medium for the scientific study of disease. Through the selection of images and posing of subjects, however, photographs like those shown below, contribute little to understanding the beriberi epidemic, yet simultaneously convey the message that the colonialists had the tools to wage war against tropical diseases in Southeast Asia. Photography demonstrated that colonial doctors had access to the most advanced technology: if anyone were to unravel this mysterious, deadly disease, certainly it would be



those with the most advanced modern technology. These photographs also demonstrate the colonialists' power over their subjects, as they required them to strip and display their withered or swollen legs in the name of scientific progress and presumably their own good. And, by placing the subjects in laboratory-like settings, the photos also imply a connection to science and reinforce the impression of scientific mastery.

Finally, once it was understood that the disease was caused by consumption of milled white rice, the colonial powers chose to develop a "cure" rather than reverting to brown rice, ultimately prolonging the beriberi epidemic for another thirty years. In 1912, after Eijkman's theory that beriberi was a disease of nutrient deficiency had been widely accepted, the Far Eastern Association of Tropical Medicine met in Hong Kong to address beriberi. The Director of Health in the Philippines, Victor Heiser stated, "'[t]he time has come to bring this knowledge to the attention of all Governments concerned…. [P]eople are dying of beriberi by the thousands and the knowledge by which this can be prevented is at our command'"(Carpenter, 95). Heiser called upon colonial powers to use that knowledge to save thousands of Asian lives. Remarkably, however, rather than simply returning to reliance on un-milled rice to end the epidemic, the colonial delegates of the Far Eastern Association of Tropical Medicine resolved to isolate the nutritional factor missing in milled rice, and then replace it. The Association in effect rejected agricultural and dietary practices prevalent in Asia for centuries in favor of maintaining the conditions that caused the epidemic so that they would have a chance to correct the deficiency in milled rice.

It took Williams another two decades, until 1934, to determine thiamin's chemical composition, and two more years for him to synthesize it. It took yet another eleven

years finally to implement Williams' "cure" for the first time in 1947. It is telling that in his book on beriberi, Williams acknowledged the scientists whose accomplishments made his discoveries possible, thanking Vedder, Casimir Funk, Atherton Seidell, Jansen, Donath, R.A. Peters, Takaki, Eijkman, Grijns, Fraser, and Stanton. Of the eleven people acknowledged, only Takaki is Asian; of the Westerners, only Funk was not from a colonial power. Williams thus makes it clear that the conquest of beriberi was predominantly a colonial effort, propelled, but also impaired, by the colonial belief in the "white man's burden."

The ultimate demonstration of European colonial control over beriberi came too late to legitimize colonial rule. Beginning in 1947, one year after the United States recognized Philippine independence, Williams, in collaboration with Dr. Juan Salcedo, a Filipino Doctor, conducted the Bataan Experiment, which proved the efficacy of a thiamin coating to milled rice in preventing beriberi:

> The results of the experiment *in toto* seemed very significant of the benefits conveyed by rice enrichment. Considering the difficulties of the task and especially of controlling the desire of people of the control area to share in the benefits of the program among their neighbors, one could scarcely ask for better evidence (Williams, 198).

Perhaps it is the final irony of the epidemic that, while it was too late to maintain the colonial system that had fallen victim to the Great Depression and World War II, Williams nonetheless transformed beriberi from a colonially-caused disease into a colonially-cured one.

Beriberi is sometimes seen as a poster child for the triumph of Western medicine over disease. Eijkman received a Nobel Prize in 1929 for his work on beriberi, and William's technique to enrich rice removed beriberi from the world's consciousness.

However, while the conquest of beriberi is in some ways the story of the power of Western medicine, it is also the story of the unintended consequences of technology—and of how colonial ideology and, perhaps, arrogance, hindered Western science and extended an epidemic caused by technology.

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