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Read, Ian

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## A triumphant decline? Tetanus among slaves and freeborn in Brazil

*Um declínio triunfante?  
Tétano entre escravos e  
livres no Brasil*

**Ian Read**

Assistant Professor of Latin American Studies/  
Soka University of America.  
One University Drive  
92656-4243 – Aliso Viejo – CA – EUA  
iread@soka.edu

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### Abstract

Tetanus and other widespread endemic diseases of Brazil's early national period speak to intimate details of common life and give clues to big, vexing questions, such as why Brazil's population expanded dramatically at the turn of the twentieth century. Tetanus was for a long time one of Brazil's deadliest afflictions, especially among infants, but historians know very little about it. Using archival sources from across the Empire and early Republic, this article argues tetanus disproportionately killed the enslaved population, but gradually diminished in virulence for nearly all groups across the country by the second half of the 1800s. This decline should be attributed only partially to medical knowledge. Rather, indirect demographic and technological changes were more important factors in Brazil.

Keywords: tetanus; demographics;  
slavery; endemic diseases; Brazil.

### Resumo

O tétano e outras doenças largamente endêmicas no período inicial do Brasil nação nos permitem entender pequenos detalhes da vida comum e grandes e inquietantes questões, tais como por que a população expandiu-se dramaticamente no início do século XX. O tétano foi uma das afecções mais mortais no Brasil, especialmente entre as crianças, mas os historiadores ainda conhecem pouco sobre ele. Utilizando fontes arquivísticas do Império e do início da República, este artigo argumenta que o tétano era desproporcionalmente severo entre a população escravizada, e que sua virulência reduziu em praticamente todos os grupos do país a partir da segunda metade do Oitocentos. Esse declínio deve-se apenas parcialmente aos conhecimentos médicos. Mudanças demográficas e tecnológicas indiretas foram fatores mais importantes no Brasil.

Palavras-chave: tétano; demografia;  
escravidão; doenças endêmicas; Brasil.

On a cold May day in the dusty interior of Piauí in 1856, a powerful landowner and the province's Vice-President, Coronel José de Araújo Costa, tied his slave Victorino to a post and whipped him until flesh broke. What brought Araújo Costa to violence is unknown; perhaps Victorino had run away or had broken a rule on the large cattle ranch where he labored.<sup>1</sup> In any case, Victorino did not recover from his torture. Some cuts began to heal, others turned tender and swollen. He developed a fever and cried in agony as pains pulsed through his body. The flesh near the bites of the whip turned a deep red and the tissue emitted pus and a putrid smell. When a doctor was called to Victorino's sickbed, he felt tight, contracted muscles in his patient's neck and mouth, telltale signs of tetanus. There was little medicine could do. As the slave neared death, Victorino's back muscles contracted sharply and his body bent backwards. During his final hours, it appeared as if two taut cords stretched from each foot to the edges of his face, pulling Victorino's mouth into the familiar yet dreaded *risus sardonius*. Even death brought no relaxation, since tetanic spasms set in *rigor mortis* (Warthin, 1921, p.49). Word of the slave's death spread widely, giving one of Vice-President's enemies an opportunity to accuse him of assassination in the *Jornal do Commercio* (22 ago. 1869). In response, Araújo Costa sued for calumny and won (Brasil, 1870a, p.107-108). The judge was sympathetic to Araújo Costa, for he saw Victorino's death as an unlucky and unintentional consequence of a man's constitutional right to 'moderately' punish his sons, slaves and pupils.<sup>2</sup>

As oddly terrible as Victorino's afflictions sound today, some of his enslaved friends or family witnessed this particular struggle with death. Like a majority of adults in Brazil during the early to mid-1800s, they had seen the characteristic tetanic contraction of skeletal muscle fibers and gradual asphyxiation strike their newly born children. In teens and adults, lockjaw was common enough to be recognized and feared. Three decades after Victorino's death, however, traumatic and neonatal tetanus had largely disappeared in some parts of Brazil and become infrequent in others.

Tetanus's decline was remarkable for several reasons. First, the disease diminished quicker among the freeborn population, implying that treatment of slaves may not have uniformly improved during the 1860s and 1870s as some contemporaries and historians have suggested.<sup>3</sup> Second, its decline brought little relief to the medical community. Public health officials commonly worried about public health in their country after 1849, when Brazil was battered by unfamiliar and terrifying sets of epidemics that included yellow fever, cholera and bubonic plague (Cooper, 1975). On top of these fears, Brazilian cities became increasingly crowded places, and doctors fretted over growing numbers of tuberculosis, smallpox and syphilis cases (Marcílio, 1993; Chalhoub, 1996; Meade, 1997; Benchimol, 2001). Few publicized the fact that tetanus, a disease that had contributed heavily to overall mortality during the early and mid-1800s, especially among slaves, was decreasing in their neighborhoods, cities or provinces.

What explains tetanus's decline? It was certainly not the realization that wounds and cuts, including that of the umbilical cord, had to be kept free of germs: incident rates of tetanus began falling more than a half century before germ theory was widely accepted. Rather, most Brazilians continued to perceive the disease as they had for centuries. Some doctors were certain that it was caused by changes in temperature, diet, or parasitic worms,

but many drew on information widely shared across the Atlantic World that simply saw it as a mysterious affliction of the nervous system with unknown cause. For many victims, its appearance was very bad luck and understood in Christian or syncretic Christian-African terms. Only by the late 1880s and 1890s – when it had become a fairly rare disease in Brazilian cities – did the nation's most prestigious and connected medical authorities believe tetanus was an infection caused by microbial contamination of wounds and cut umbilical cords. By then, this disease infected five times fewer victims than it had when bacteria became implanted in Victorino's back by the cut of his master's whip. We must search, therefore, for other factors that either reduced *Clostridium tetani* bacteria in the environment or made them less likely to enter wounds and umbilical stumps.

Historical medical studies of tetanus deserve our attention because this disease was an enormous killer among Brazilians and was a worse problem among the already disadvantaged enslaved population. The disease impacted daily life in a marked way; therefore, if we wish to learn about common hopes and fears of quotidian life in the nineteenth century, we need to know something about tetanus's role. Second, the fact that it mysteriously diminished before the 'germ revolution' suggests that changing environmental or social conditions (intentionally or unintentionally produced) were the real reason for its decline. This will be fully discussed in the conclusion. Finally, tetanus's decline contributed to Brazil's population growth. Between 1872 and 1910, Brazil's population grew exponentially, setting the stage for a much more populated, urbanized country in the twentieth and twenty-first centuries. European immigrants are often credited with this growth, but a 'natural' increase through rising birth and survival rates mattered much more (Merrick, Graham, 1981, p.316-317). That neonatal tetanus transformed from a common affliction into a rare one in Brazil's big cities is especially important since it reduced overall mortality rates, especially among newborns. Despite these good reasons to learn more about this disease, no history of medicine in Brazil contains more than a paragraph or two on the disease.<sup>4</sup> A similar lack of attention can be found in the study of other slave societies of the Americas (McMillen, 1991, p.294). Slaves in the US and the Caribbean, like their counterparts in Brazil, died more frequently of tetanus infections than the freeborn, yet beyond this fact very little is known about morbidity and mortality rates or about past views of pathology and etiology, cultures of treatment, social and economic consequences, and tetanus's connection to fatalism and perceptions of infancy.

Fortunately, the history of medicine in Brazil is attracting new attention, especially among younger scholars, broadening the context for the study of this particular disease. In the last fifteen years, historians have searched for the commonest and deadliest afflictions among slaves (Porto, 2006), although a great need still exists to compare the health and treatment of slaves to that of free people. Furthermore, an uneven amount of attention is paid to epidemic diseases since these make for a stirring narrative. Between the first outbreak of yellow fever in 1849 and the destructive influenza pandemic of 1918, we can find a number of grisly stories of mass hysteria, local anarchy and corpses left to rot on streets or in abandoned houses. Common endemic diseases such as tuberculosis and tetanus, however, killed far more slaves and free people in the long run. Thus, they speak better to the day-to-day concerns and experiences of Brazilians in the nineteenth century.

This article sets out to answer several questions. To what degree were different groups, such as the enslaved and freeborn, infected and affected by tetanus? How did the disease vary temporally or geographically? Brazil is, after all, a ‘continental’ country that, between 1830 and 1910, evolved from an unsteady young Empire to a more powerful and oligarchic Republic. Wage laborers replaced slaves in cities that grew fivefold. Did the ways that people understand and treat tetanus also change? What actions did people take to save their suffering newborns or loved ones, and what might have altered in the environment or society to give people much less to worry about when it came to this disease? Tetanus is an exceptional affliction, worthy of special note, because there are no other fatal diseases so closely connected to particular obstetric care and the handling of wounds. Lastly, what larger story can we tell about the history of Brazil or its endemic diseases when both the institutions that ensured slavery and a scourge that took a disproportionate number of enslaved lives concurrently weakened? Indeed, by 1885, both slaves and death by tetanus were rare enough in Brazil’s big towns and cities to be considered by many as a scourge of a bygone generation.

### General characteristics

In order to understand tetanus historically, it is useful to put it into the context of contemporary pathology and medical geography. The disease appears when *Clostridium tetani*, an anaerobic, ‘tennis racket’-shaped bacterium, is transferred from soil, a dirty object or feces into a wound that then becomes infected. The bacteria cannot survive when exposed to air and sunlight but can last for decades (and even millennia!) when self-encapsulated in an endosporic state. Endospores sit dormant, usually in soil, until they die or conditions allow replication. Since a *C. tetani* bacterium is about one micrometer (1/1000 of a millimeter), it can easily enter a wound on contaminants far too small to be seen. Furthermore, the microbe is distributed widely to an amazing degree, and has been found in fields, forests and plains throughout the world, but thrives in cultivated farmlands, especially when manure or night soil is used as fertilizer. Tetanic infection has long been more of a problem in equatorial regions, but whether this was due to a higher prevalence of bacteria or more direct skin-to-soil contact is unknown. Additionally, aseptic practices, hygienic postnatal care and immunization have taken root far more slowly in the intertropical zone than in the northern and southern temperate zones, creating sharp pathological inequities. Today, about 74,000 infants die annually from neonatal tetanus, mostly in impoverished nations in Africa and Southeast Asia. It remains a tragic, preventable disease (Guilfoile, 2008; Bytchenko, 1966; Mandate, 2010). Yet much has improved: if global rates had not fallen so dramatically since the nineteenth century across the world, today we would experience one to three million more neonatal deaths each year.

*Clostridium tetani* usually enter a wound when the skin is punctured by a dirty object or the umbilicus is cut with an unsterile instrument or covered with a harmful cord application. Once in the wound, levels of oxygen are usually too high for *C. tetani* to colonize. Only within deep abrasions (e.g., a nail in the foot) or when other bacterial infections diminish oxygen in the skin or muscle tissue can the bacteria replicate. As it does, *C. tetani* produces

tetanolysin and tetanospasmin, two chemicals with unknown functions. Tetanospasmin is the most powerful poison discovered; only one tenth of a milligram is needed to kill a person (in comparison, a grain of rice is about 25mg.). As a neurotoxin, it causes the clinical manifestations of tetanus by tightly binding to neural gangliosides and altering their molecules until efferent nerve action is restricted. In this state, muscles contract involuntarily but still tire, creating enormous pain for a victim. As was likely the case for the slave Victorino, an infected body will often pull into a sharp arch called opisthotonos. Facial muscles are also commonly affected, and the *risus sardonicus* 'smile' was identified by Hippocrates in his classic *Aphorisms* (400 BCE). Hippocrates also noted, as doctors do today, that contractions can become so severe that victims find it impossible to swallow and difficult to breathe. Thus, in a basic sense, the nervous system becomes poisoned, malfunctions and causes the body to asphyxiate itself. Death is common: even in places with advanced medical facilities and life support, about half of all patients with clinical tetanus infections die (Guilfoile, 2008; Miller et al., 1998, p.4110-4116; Stoelting, Hines, Marschall, 2008, p.482).

Was that which contemporaries called 'tetanus' the same as what doctors consider to be 'tetanus' today? There is also good reason to question nineteenth-century nosography, or the naming and categorization of diseases. This was a time when many afflictions were identified by their symptoms rather than their causes. Other noted causes of death, such as *dentição* ('teething') and *endema* ('dropsy'), confused chance correlation with direct causation. The symptoms related to tetanus were potentially bewildering since other diseases cause muscle spasms, contractions or seizures before death, especially among infants. While it is certainly true that the diagnosis of *tétano* (tetanus), *trismus neonatum*, *tétano umbilical*, *mal de umbigo*, and *mal de sete dias* (all neonatal tetanus) were sometimes mistaken by contemporary standards, in most instances this did not happen because tetanus commonly displays a unique set of symptoms. It has a particular timing, for example, usually occurring within six to eight days of a wound or cut umbilical cord. Additionally, (1) muscle contractions are more often sustained than spasmodic; (2) affect particular muscles of the back and neck; (3) shape the body in a certain way; and (4) cause *risus sardonicus*, a symptom rarely attributed to other diseases.<sup>5</sup> When these clues were put together, tetanus was one of the most easily diagnosable afflictions, recognizable without autopsies and to the medically untrained. *Convulsões* ('convulsions') was another common cause or description of death during the nineteenth century. One popular Brazilian medical textbook from 1865 remarked that one "can distinguish a *spasm*, which consists of one permanent contraction of the muscle fibers, from a *convulsion*, which alternates between contraction and relaxation of the same fibers."<sup>6</sup> It added, "tetanus is the most elevated degree of spasm, while epilepsy is of convulsion" (Langgaard, 1865, p.537). Following these instructions, ailments commonly causing seizures, such as rapidly increasing fevers, low blood sugar levels, brain damage, infections such as meningitis or encephalitis, parasitic worms, and tetany caused by malnutrition were more often classified as 'convulsions,' not as tetanus.

### **The incidence of tetanus in nineteenth-century Brazil**

Tetanus was generally a worse problem in Latin America than in Europe or North America, and some parts of Latin America, such as coastal Brazil and northern Argentina, appear to have had some of the highest rates of infection. Global comparisons are hard to make since data do not exist for parts of the world that had the highest rates in the twentieth century, such as Africa and Southeast Asia. But compared to Northern Europe, the US and India, Brazil stands out.<sup>7</sup> Of these places, Europe had the lowest rates, with tetanus infections causing less than one percent of total deaths in London, Dublin, Vienna and Porto during the 1840s and 1850s (Agnew, 1878, p.434; Virchow, 1855, p.174; Portugal, 1858, p.146). Tetanus in the US was also generally rare, although Americans in certain parts of the south, such as New Orleans, Charleston and Savannah, suffered much more, especially among the enslaved and free blacks. In fact, people of color in Charleston were three times as likely to die as whites. Still, all Brazilians faced higher risks from tetanus during the early to mid part of the nineteenth century than did the slaves of Charleston and New Orleans. Even Bombay, with its widespread poverty and tropical climate, may have been less plagued by this disease (Jones, 1876, p.255-263). Like Brazilians, Argentines were also afflicted by the disease in unusual numbers. Residents of Buenos Aires, especially those “in houses overcrowded with people who know not how, or are unable, to give their infants the care which is required by their tender years” (Rawson, 1877, p.1049) were killed in proportions higher than in Brazil’s capital. In 1875, Havana had levels of tetanus infection that were worse than Brazil’s southern cities but better than its northern cities.

Tetanus rates varied considerably in Brazil, with tetanus more of a problem the more tropical the climate and poorer the local economy. As shown in Table 1, Natal and Aracaju had the highest rates, while São Paulo and – by the 1860s – Porto Alegre had the lowest. Much of this has to do with climate and income levels. The lowest monthly temperatures in Natal and Aracaju are between 23 and 24 degrees Celsius, rarely cold enough to require much clothing. In the nineteenth century most men wore a pair of light trousers and short sleeve shirts and women wore light cotton dresses or blouses. Such weather rarely discomforted bare feet. By contrast, winter in São Paulo and Porto Alegre is considerably colder, with average lows between 14 and 16 degrees. In these parts of the south, certain months of the year required thicker pants, jackets and, presumably, fewer bare feet.<sup>8</sup> It seems logical that more clothing reduced direct skin-to-soil contact and the chances of skin punctures and abrasions. Rio de Janeiro and Santos make interesting comparisons because even though they are fairly far south, Atlantic currents give them warmer, wetter climates than the rest of the south. Santos is, in fact, the only major city below the Tropic of Capricorn with a tropical environment. Its winters are similar to Natal and Aracaju and, as we can see, its residents suffered disproportionately from tetanus. Rio de Janeiro and Salvador both experience mostly warm, wet winters, yet their rates of tetanus were in the mid-range between 1850 and 1870, suggesting that wealth and urbanization also had an effect on tetanus. During the nineteenth century, these two were Brazil’s largest and wealthiest cities, although Rio de Janeiro greatly surpassed Salvador in wealth and population by the second half of the century.



**Table 1: Tetanus deaths as a percentage of total deaths for various Brazilian cities, 1835-1906**

Year	City	As percentage of total deaths	
		Neonatal tetanus	Tetanus affecting all ages
1835	Porto Alegre	5.2	7.7
1841	Porto Alegre	4.0	5.8
1845	Rio de Janeiro	4.1	4.1
1854	Salvador	2.2	4.0
1856	Recife	6.7	7.1
1857	Porto Alegre	1.7	3.0
1857	Niterói	3.3	4.0
1865	Santos	7.1	8.8
1866	Porto Alegre	2.1	2.6
1866	Niterói	2.4	3.0
1869	Rio de Janeiro	3.2	4.1
1869	Salvador	4.9	5.8
1870	Recife	3.5	5.2
1871	Natal	10.8	nd
1872	São Paulo	0.0	0.0
1875	Rio de Janeiro	2.2	3.2
1875	Recife	nd	5.7
1875	Aracaju	8.4	nd
1875	Porto Alegre	2.4	2.5
1877	Salvador	nd	4.9
1881	Manaus	nd	6.0
1885	Rio de Janeiro	1.2	1.9
1888	Santos	3.4	4.3
1889	Porto Alegre	0.1	0.3
1890	Niterói	1.5	2.0
1892	São Paulo	0.9	1.3
1896	Rio de Janeiro	0.3	1.2
1906	Santos	0.9	1.3
1906	São Paulo	0.4	0.6

Sources: Compiled by the author based on: Porto Alegre (Livro de óbitos..., 1835, 1841, 1843; Santa Casa..., 1850-1896); Rio de Janeiro (Jornal..., 22 ago. 1869; Brasil, 1869, 1875, 1896; Mello Alvim, 1902, p.26-29); Salvador (Brasil, 1855, 1876; Gazeta..., 1869); Recife (Livro de óbitos..., 1857, 1870; Brasil, 1876); Niterói (Silva, 1893, p.159-161); Santos (Livro de enterramentos..., 1865-1888, 1906b; Livro de enterramentos..., 1906a); Natal (Brasil, 1872); São Paulo (A Província..., 1876; O Estado..., 1892); Aracaju (Brasil, 1876); Manaus (Brasil, 1882). 'nd' indicates no data.

Tetanus fell as a risk for nearly every location where serial data exist. In Porto Alegre, 7.7% of all deaths were attributed to tetanus in 1835. By 1889, this had fallen to only 0.3%. Recife, Niterói, Santos and Rio de Janeiro also saw lower numbers after 1870. At the parish level within Rio de Janeiro, rates also fell but varied considerably. For example, tetanus averaged 2.3% of all deaths in the carioca parishes of Engenho Velho, Glória and Santa Rita in 1896. Much lower rates existed in Lagoa, São Cristóvão and Santo Antônio, with 0.8% of deaths attributed to tetanus (Mello Alvim, 1902, p.XXVI-XXIX). Engenho Velho, Glória and Santa Rita had a much higher population density, averaging 10.2 people per domicile. Lagoa, São Cristóvão and Santo Antônio averaged 7.5 people per domicile. When these two sets of parishes are compared in terms of literacy or levels of legitimate births, there is very little difference, suggesting that within each parish there was significant economic and social diversity (Brasil, 1907, p.31-107). Only Salvador and São Paulo did not demonstrate a steady decline but with only a few data points available for each, it is hard to tell if this reflects a general trend or the effect of outliers. Porto Alegre saw steeply lower rates as early as the 1850s; in Niterói, tetanus fell in the early 1860s.



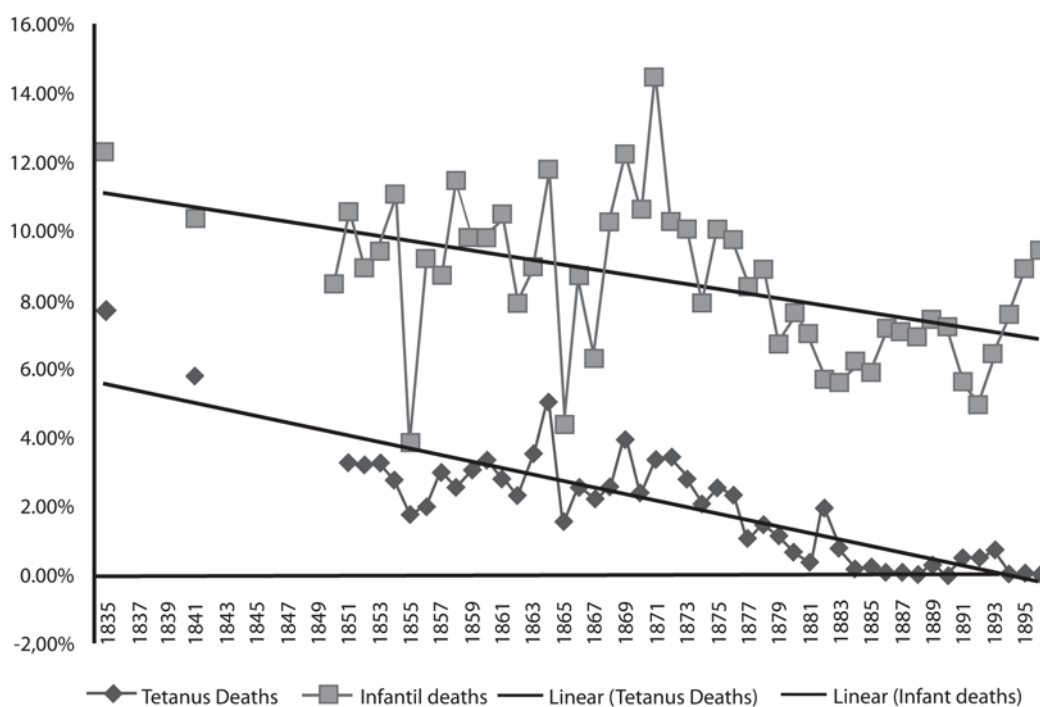
The general decline of tetanus in many urban parts of Brazil preceded the widespread knowledge that this was a disease caused by germs entering and infecting wounds or umbilical stumps. Joseph Lister's ideas on aseptic surgical practices did not become widely applied until the mid to late 1870s. Robert Koch's *Postulates* were published in 1890. It is true that for centuries there was a small minority who argued that diseases could be transferred by microscopic particles. For example, in 1546 Girolamo Fracastoro invented the idea that *seminaria* (seed-like entities) could disseminate epidemic diseases (Patrick, 2007, p.310). Most Brazilian doctors of the mid-nineteenth century scoffed at such 'outdated' ideas, believing instead that disease was spontaneously generated by the local environment, or that environmental conditions became ripe for disease when triggered by an imported *excitador*.<sup>9</sup> Much depended on the particular affliction, however. There was less debate that certain diseases, such as influenza, common colds, smallpox, scarlet fever and measles were contagious even among the most hardheaded miasmists. Germ theory, on the other hand, was embraced by Brazilian doctors only at a time when tetanus rates had already fallen to a fifth or less of their earlier rates. Why this is the case will become more apparent after we discuss the ways that tetanus victims were treated.

What about Victorino's untimely death and rates of tetanus among slaves generally? Various cities and provinces published serial data on tetanus deaths, but these rarely distinguished slaves from freeborn. Instead, one must turn to old parish obituary and cemetery registers. Seven hundred and seventy-five burials in Recife (in 1856 and 1870), 3,556 burials in Santos (1857-1858, 1865-1888, 1906) and 54,213 burials in Porto Alegre (1834, 1841, 1843, 1850-1890) allow us to compare risk of tetanus among slaves and freeborn across coastal and urban Brazil. These data reveal that tetanus affected slaves to a greater degree than the freeborn, and while infection rates declined rapidly for freeborn, they sometimes 'increased' for slaves. In Recife, the proportion of tetanus deaths among freeborn fell from 5.6% in 1856 to 3.4% in 1870. Among slaves for this same period, rates increased from 14% – an extraordinarily high proportion – to nearly 17%. In Porto Alegre, tetanus increased among slaves from an average of 3.8% for 1851-1855 to 5.2% for 1867-1871. Among freeborn in this city, the disease decreased slightly from 2.4% to 2.2% for the same periods. If we include data from the parish obituary records for 1834 and 1841, slave tetanus may have declined for both groups over the long run. As seen in Graph 1, two trend lines based on linear regressions suggest that tetanus rates for both enslaved and freeborn fell, but more steeply for the free population. Therefore, tetanus appears to have had an increasingly disparate effect when it came to freedom in Porto Alegre. Finally, in Santos, the risk of tetanus also declined for both slaves and freeborn at similar rates. Between 1865 and 1874 tetanus fell from 7.8% to 2.6% of total deaths among free people. The risk of tetanus for slaves fell from an average of 8.1% for 1865-1871 to 2.1% for 1872-1886 (Read, 2009, p.73). These numbers boil down to this: while tetanus became far less of a risk among the Brazilian free population after 1870, the same was not always true for Brazilian slaves.

Despite the fact that slaves suffered disproportionately more, tetanus was not among the diseases closely associated with slavery. A few noted greater incidences among slaves, such as José Martins da Cruz Jobim (1835), João Baptista A. Imbert (1843) and Theodoro

J.H. Langgaard (1865), but these doctors attributed the problem to the conditions within which slaves lived or to postnatal customs among their midwives. Nearly all who wrote about tetanus within and outside of Brazil claimed it was a far worse problem in the 'torrid zone,' but did not discriminate among those who lived in or entered this climate. A medical thesis on tetanus written by a Brazilian student in 1869 pointed out that it was more common among adult men and newborns of both sexes yet, again, did not mention any special propensity by legal condition (Serra, 1869, p.7). Such lack of attention to the higher incidence rates among slaves contrasts sharply with the discourse of US doctors, who frequently connected the disease to slaves, yet were also more likely to consider its origins mainly in slave 'habits' and 'customs' (McMillen, 1991, p.309).

**Graph 1: Deaths from tetanus in Porto Alegre (as percentage of total deaths, by legal condition)**



Source: Compiled by the author based on death records from Santa Casa de Misericórdia de Porto Alegre (Santa Casa..., 1850-1896)

Data on tetanus incidence in this study is derived mainly from Brazil's urban areas. This only gives us a piece of a bigger picture because most Brazilians during the nineteenth century lived in isolated rural communities or in or near small towns. Brazil was a deeply rural and agricultural country until the mid-1900s, when mass migration brought millions into Brazil's rapidly growing cities. One might expect rates to be higher in the countryside since agricultural practices brought more people into contact with soil and where midwives may have practiced more traditional birthing techniques. Additionally, the countryside offered fewer medical resources to properly treat wounds. Until more work has been done using parish and cemetery registers from Brazil's rural areas, we can only speculate on what

diseases and afflictions killed the majority of rural Brazilians, freeborn and enslaved, before the twentieth century.

By the turn of the century, state governments became more active in monitoring public health. For example, between 1895 and 1933, São Paulo's Office of Sanitary Service (Diretoria do Serviço Sanitário) published detailed demographic statistics, including causes of death for nearly all of its municipalities in the state. Nearly a third of the 146 municipalities listed in 1906 had death rates of tetanus over 3% of total deaths. This was considerably higher than the cities of São Paulo and Santos (see Table 1). The townships of Agudos, Conceição de Monte Alegre, Pilar, Ribeirão Branco and Rio das Pedras had rates over 10%. Conceição de Monte Alegre and Ribeirão Branco also had unusually high rates of tetanus six years before, suggesting that rural 'foci' of tetanus had geographic persistence (Anuario..., 1900, p.50-97; 1906, p.84-179).

These high rates were probably caused by traditional and unhygienic midwifery practices and a high degree of contact with soil that harbored the *C. tetani* bacteria. Many of the towns with tetanus 'epidemics' had higher than average levels of animal breeding (*zootécnica*). Farmers bred and raised cattle, horses, mules, sheep, swine and oxen, all of which can transfer *C. tetani* through their digestive tracts. The degree of crop farming for each municipality does not correlate with tetanus rates (Piza, 1903, p.568-571).<sup>10</sup> Despite its scattered foci of infection, rural São Paulo mirrored national trends since tetanus generally appeared to be declining as a threat. In 1900, about forty people died from tetanus for every one thousand people born among 69 largely rural municipalities. In 1906, the rate had fallen to about thirty. By comparison, in 1960 there were six deaths for every one thousand births in São Paulo state (Bytchenko, 1966, p.80).

While tetanus was far less of a threat in the twentieth century, Brazil appears to have presented a higher risk of tetanus relative to other places of the world, even as this disease became much less common. In 1960, the disease caused 9.8 deaths per 100,000 people in São Paulo state. This surpassed the rate the World Health Organization estimated for Mexico and Central and South America (8.5 deaths per 100,000). Considering that São Paulo was Brazil's wealthiest state in 1960, rates were probably higher in other parts of the country. Tetanus was much worse in Brazil than the US (0.2 per 100,000) but better than parts of the Caribbean (the Dominican Republic had rates of 68 per 100,000). Again, we can see the interplay of wealth and climate on tetanus rates. For example, Florida, with tropical and subtropical climates, had rates of 1.5 per 100,000 in 1957, the highest among all US states (Bytchenko, 1966, p.75-81). There were other US states poorer than Florida, but they lacked such warm temperatures and had lower incidence rates. Tetanus improved in the US and Brazil, yet maintained a lingering persistence. Much had changed in a century and a half: in 1872, one of the only years in which fairly reliable census figures exist for the nineteenth century, Salvador and Rio de Janeiro had tetanus mortality rates of 131 and 135 per 100,000 respectively. In 1991, only one of every 100,000 people died of tetanus in Brazil (Moraes, 2000, p.271).

## The evolution of tetanus's etiology

It is tempting to look back at the nineteenth century and view theories of tetanus pathology as remote, foolishly misguided or appallingly incomplete. After all, its bacteria were not identified and the disease was not believed to have been transferable until the mid-1880s. Yet what becomes clear from the medical literature is that although many doctors modestly acknowledged how little they knew, most had already arrived at an outline of the nature of this disease that is in basic alignment with today's views. Additionally, medical research on the causes, symptoms and treatment of tetanus was internationalized, at least among the countries of the Atlantic World. As will be discussed, the most popularly used textbook on diagnosis and treatment in Brazil mostly mirrored knowledge shared by doctors in Europe and North America.

First, there is the modesty. In 1862, Samuel David Gross, immortalized by the painter Thomas Eakin (Figure 1), confessed the limits of general knowledge of tetanus:

It might reasonably be supposed that a disease which is characterized by so much violence during life would leave some traces of its existence after death; but to show how erroneous such a conclusion is, it is only necessary to refer to the fact that all the dissections that have hitherto been made of persons dead of this affection have utterly failed to throw any satisfactory light upon its pathology and morbid anatomy ... We are no wiser now in regard to the real lesions of this disease than our forefathers were centuries ago. The whole subject is, in truth, still a mystery (Gross, 1862, p.670-671).

Similarly, a British doctor observed in *The Lancet* in 1853 that “we need hardly say how obscure is the pathology of tetanus ... but this very obscurity materially endows all the cases of this fearful malady with a considerable share of interest” (Gull, 1853, p.165). Uncertainty and interest were shared by Brazilian doctors, where this “fearful malady” was far more common and, thus, more easily observable. The Emperor's personal doctor, Joseph François Xavier Sigaud (2009, p.246), referred to the pathology of tetanus with a series of unanswered questions. Claudio Lisboa Serra (1869, p.9) wrote that “on the nature of this illness we cannot speak with any certainty, because we still know nothing about it decisively”.

A few took the liberty to speculate wildly on the mechanisms of the disease. James Marion Sims, the ‘father of American gynecology,’ emphatically believed that neonatal tetanus resulted from excessive pressure on the brain from misshapen occipital bones.<sup>11</sup> Others noticed that strychnine poisoning produced symptoms that closely resembled tetanus and drew a connection between the disease, blood poisoning and miasms (Rosenau, Anderson, 1908, p.12). Yet these ideas, especially those of Sims, were seen by most doctors as farfetched. In fact, one can detect an emerging trans-Atlantic consensus on tetanus pathology in the mid-nineteenth century that in hindsight makes Gross's admission of collective ignorance sound exaggerated. This consensus built on the views of ancient Greeks such as Hippocrates and Aretaeus, who argued that tetanus was a consequence of wounds, minor and major, and that *trismus neonatum* was connected to an ‘aggravation’ of the umbilical cord. It also borrowed from five centuries of pathological anatomy in which countless corpses were cut open in a search for clues for how and why diseases killed.

When the lifeless bodies of tetanus victims were dissected, anatomists detected minute amounts of inflammation within the spinal cord and, occasionally, among nerves near wounds. Signs such as these were not easy to find; autopsies today reveal very little in the corpses of tetanus victims except for “local inflammation at the site of infection and the swelling of motor neurons in the brainstem and spinal cord” (Byard, 2004, p.192). Yet

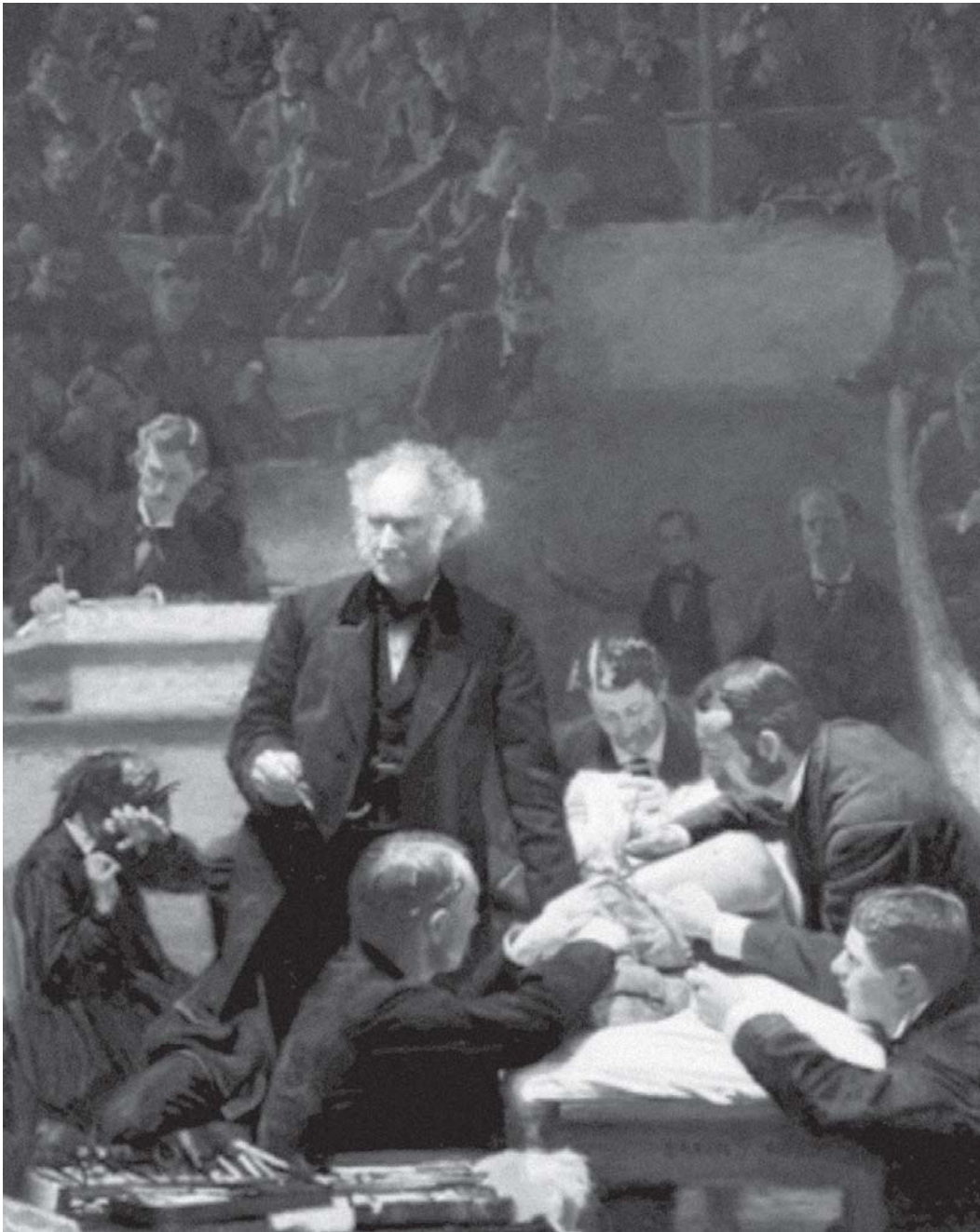


Figure 1: *The Gross Clinic*, Thomas Eakin, 1875 (<http://en.wikipedia.org/wiki/File:EakinsTheGrossClinic.jpg>)



with far fewer tools at their disposal, doctors took these small clues and came to the view, common by the mid-1800s, that tetanus resulted from some 'excited' state of the spinal cord and central nervous system reinforced by internal 'irritation' of the afferent (sensory) nerves (Martins, 1868, p.68-74; Watson, Condie, 1850, p.351; Langgaard, 1865, p.525). These early medical investigators were not far from today's mark: tetanospasmin, the tetanus toxin, links with interneurons of afferent and efferent fibers and prevents these nerves from performing their normal function (i.e., releasing acetylcholine) (Chamberlain, 2008, p.96). Although it is true that no one considered germs to cause tetanus, death from tetanus is not caused by the infection of *C. tetani* alone but by the neurotoxin they (incidentally) produce.

The first known experiment to show that tetanus was a transmissible disease occurred in 1846, when an American doctor, W.A. McDowell (1846, p.578), surgically implanted "minute tack points in to the muscular spinal nerve" of two dogs named Gumbo and Watch. Both dogs developed tetanus and died, but neither McDowell nor anyone else continued on this path of research. McDowell's work was sometimes cited, but with attention to his demonstration of a "local irritation of the nerves" rather than its cause from a foreign body (Jones, 1876, p.219-220). Attention gradually shifted from endogenous to exogenous factors as microbes gained attention in the 1860s and 1870s. Some doctors, such as Adolf von Strumpell, argued in favor of an infectious origin for tetanus because it resembled sepsis and rabies, two diseases widely believed to be infectious (Rosenau, Anderson, 1908, p.13; Strumpell, 1887, p.751). In 1884, Arthur Nicolaier looked again at the effects of foreign particles in animals, apparently without knowledge of McDowell's previous work. Importantly, Nicolaier designed his experiments with the idea that tetanus's origins could lie in an external, microscopic cause. When mice, rabbits and guinea pigs developed tetanus after injected with small particles of dirt, he pressed forward. The following year, Nicolaier separated slender rod shaped bacilli from the pus of the animals' wounds but was unable to obtain a pure culture. This was done in 1886 by Shibasabur M Kitasato, a Japanese researcher working under Robert Koch, who confirmed that Nicolaier's bacilli caused this millennia-old scourge (Rosenau, Anderson, 1908, p.13; Joy, 1993, p.1044-1045; Guilfoile, 2008, p.22-24). The discovery that tetanus was infectious, like most important medical discoveries, coincided with parallel scientific developments (i.e., the foundations of microbiology) and a major paradigm shift (i.e., microbial origins of disease) and followed a long trail of similar but inconclusive experiments (i.e., by McDowell or Antonio Carle and Giorgio Rattone).

The discovery that tetanus was caused by bacteria did not do much to change how patients were treated, but it did strengthen efforts to prevent the disease and began a search for a vaccine. In therapies for tetanus, doctors in the nineteenth century relied mostly on anodyne and relaxant treatments (Chernoviz, 1864, p.746; Curling, 1837; Copland, 1844, p.1024-1033; Gross, 1862, p.671-675; O'Reilly, 1850, p.297). Far less is known about African or indigenous treatments in Brazil. At least among Western trained allopaths, the most common medicines in the West for tetanus were opium, alcohol and camphor. Cannabis, aconite (aconitine) and chloroform were also occasionally given. Doctors debated the effectiveness of amputation among traumatic tetanus cases involving wounds

on the limbs. A few doctors found that “dividing a nerve” or cutting peripheral nerves near a wound brought relief (Jones, 1876, p.342).<sup>12</sup> But for every success story of preventative surgery, there was another in which this action hastened or worsened the disease. Warm, cold and vapor baths and muscle massages (‘frictions’) were popular, each with their defenders. Before 1850, purgatives and bleeding were commonly administered, but these fell out of favor as a treatment for many afflictions, including tetanus, by the 1860s. Many other substances were employed during the nineteenth century in Brazil and the west, including belladonna and tartar emetics. As seen in Figure 2, musk – an odiferous and costly substance from the caudal glands of a musk deer – was also considered by many to be therapeutic for neonatal tetanus. One drug sometimes recommended in the Americas during the 1840s and 1850s was ‘balsam of Peru,’ a resin taken from the trunk of several varieties of trees that grew in South and Central America.<sup>13</sup>

The most commonly used medical guidebook in Brazil during the 1800s, Pedro Luiz Napoleao Chernoviz’s *Diccionario de medicina popular*, dedicated several pages to the symptoms of tetanus and recommendations for its cure. Chernoviz described tetanus and prescribed treatments in ways that were very similar to Western medical doctors across the Atlantic. For example, he wrote in the second edition, published in 1851, that tetanus is a “disease characterized by the rigidity and the convulsive and near permanent contraction of part or all of the muscles” (Chernoviz, 1851, p.515). He also described the ‘smile’ of trismus and distinguished tetanus from other diseases that might have caused more variable contractions. For treatment, Chernoviz recommended sulfuric ether (diethyl ether) mixed with water and gum syrup (gum arabic). Alternatively, a sufferer might receive a cup (equivalent to five shots!) of *cachaça* every two hours until it had produced “a complete drunkenness.” Outside of Brazil, sulfuric ether and alcohol, along with chloroform and tincture of opium, were widely used as anodyne treatments for tetanus. Only in Brazil, however, was *cachaça* the alcohol of choice.

In 1890, four years after Chernoviz’s death, the sixth edition of the *Diccionario* was published. Nearly everything printed in the second edition is reprinted in the sixth, although the amount of text dedicated to tetanus doubled, with new information on how to diagnose tetanus through observations of arterial palpation (pulse), respiration and digestion. The medical manual still recommended sulfuric acid and *cachaça* but added Sydenham’s laudanum (opium mixed with spices) and chloral hydrate as two other possible treatments. Opium and chloral hydrate continued the centuries-old traditions of medicines that alleviated pain but did little to slow the disease. At least among Brazil’s most influential allopaths, Brazilian medicine was not unique in its modes of diagnosis and treatment of tetanus.

Of all therapies of the 1800s – some of which may have been given in vain to the slave Victorino in 1856 – only amputation and surgically dismantling affected nerves may have prevented the tetanospasmin neurotoxin from reaching the central nervous system, but these operations carried enormous risks and a low rate of success. Medicines, if not administered in doses that caused other harm, treated the pain and symptoms of the disease but were not cures. Even today, no medicine exists that can reduce the effects of tetanospasmin once released in damaging quantities by *C. tetani*.



**TRATAMENTO DO TRISMO NOS RECEM-NASCIDOS PELO SR. PITSCHAPT.**

2628 No caso de trismo com convulsões, em menino de mama, sendo normaes as evacuações alvinas, o Sr. Pitschapt emprega a mistura seguinte: — Hydro-lato de flores de laranja 60 gram. — Sabão de Vênezia 1 gram. — Carbonato de magnesia 1 gram. — Al-miscar escolhido — 5 centig. — Xarôpe d'amendoas — 8 gram. — M. S. A. — Dá-se uma colher de sopa ou de café de hora em hora, segundo a idade.

Figure 2: Medicinal prescription for neonatal tetanus (Tratamento..., 1844, p.289). For a translation, see note 14.

Remedies were most frequently administered in the 1800s by visiting doctors who checked pulses and felt for fevers at a patient's bedside. In Brazil, like many parts of the western world, hospitals were largely reserved for adult men whose occupations or destitution made them transient. Additionally, evidence is mounting that Brazilian hospitals were seen as places that could treat 'certain' afflictions. For terminal or near terminal diseases, such as tetanus and late-stage tuberculosis, the hospital was avoided (Read, 2009, p.72). This is not to say that victims of tetanus, if they had no home or hotel, were always turned away. Among 3,816 patients treated in Santos and Porto Alegre during various years between 1845 and 1883, seven suffered from tetanus. All died except an African-born slave in Santos named Bernardo.<sup>15</sup> Mortality rates may have been better in the large public charity hospital in Rio de Janeiro but, again, it appears that tetanus victims were rarely treated within the main hospital of the country capital. Of 10,390 patients admitted to the Santa Casa de Misericórdia Hospital in Rio de Janeiro in 1869, 33 were treated for tetanus while an unknown number died (Brasil, 1870b). The percentage of patients treated for tetanus is far lower than the percentage of people who died from this disease, especially if neonatal tetanus is included.

Following the discovery of *C. tetani*, doctors and public health officials recommended actions to prevent bacteria from entering wounds and the umbilicus stump. Preventative measures had long been held as important against this disease, sadly because therapy was so terribly ineffective. When it came to the treatment of small or minor wounds before 1870, doctors commonly recommended ointments and liniments, usually composed of heavy metals, minerals or resins such as lead, mercury, sulfur or turpentine. Large wounds were immediately sutured or cauterized to stop bleeding, foreign bodies were removed and the wound plastered or 'dressed' (Webster, Reese, Parkes, 1856, p.1216). Lister's antiseptics were not used until the 1870s, but wounds had been rinsed with cold or warm water long before then. They were sometimes doused with wine or brandy (São Romão, 1858, p.183). Alcohol had disinfectant properties, of course, as did some of the chemicals in the popular

ointments. Some of the applicants certainly aggravated wounds and, in the case of the oils and ointments, may have restricted oxygen so as to create conditions for a *C. tetani* infection.

Since most tetanus deaths followed infections of the umbilicus stump, it is worthwhile to look closely at practices related to the handling of the cord and general postnatal care. Again, much less is known about African traditions in Brazil relating to child delivery. According to João Baptista Imbert, neonatal tetanus in Brazil was especially common among slaves because of their “pernicious custom” of putting pepper, castor oil or “some other irritant” on the umbilical cord (Freyre, 1986, p.381). Today, health workers still encounter groups across the world where it is customary to apply poultices made of cow dung, charcoal, clay or other substances. Physicians in the US south complained that slaves applied charcoal powder, manure and “umbilical dressings that included soot, ashes and dirt particles” (McMillen, 1991, p.302; Kiple, 1993, p.1047). The use of umbilical applicants should not, however, have been regarded simply as an exotic or ‘tribal’ custom by westerners. For example, in order to remove the vernix caseosa, the white ‘cheese-like’ substance that covers part of newborns’ skin, midwives and obstetricians in Europe and the Americas commonly covered a baby in lard, vegetable oil or butter before bathing (Plant, 1881, p.570). Francisco Basilio Duque (1864, p.19), in a dissertation on “the hygiene of children, their delivery and cutting the umbilical cord,” recommended to Brazilian readers to remove the vernix with “egg yolk diluted with a small amount of water”. Whether or not these products caused tetanus infections is uncertain.<sup>16</sup> Despite the widespread use of applicants applied to newborns in Brazil and much of the west during the 1800s, doctors had long speculated that neonatal tetanus was caused by “bad management of the cord” (Watson, 1859, p.28). This was an “old opinion” that was not unanimously accepted, according to one American doctor writing in 1856 (Churchill, 1850, p.97-98; Kenny, 2007, p.234). Nonetheless, it appears to have come back in vogue in the US and Brazil. In 1869, 15 years before Arthur Nicolaier identified the tetanus bacterium, José Pereira Rego, President of the Junta de Higiene Pública (similar to a Public Health Board) and Brazil’s most prominent public health official of his day, declared: “It seems beyond doubt in the face of clinical observations that the most powerful cause of [neonatal tetanus] is the lack of cleanliness and neatness in the care of the umbilical cord, and the use of a substance more or less irritating that some less respectful midwives apply to the umbilical cord” (Brasil, 1870b).

## Final considerations

To summarize, rates of tetanus began falling in many Brazilian cities and some rural areas in the middle and latter part of the nineteenth century. Rates fell faster for freeborn than slaves, but both groups seem to have benefited. Part of the explanation for why tetanus fell lies in intended actions taken against the disease. Yet unintended actions may have mattered more.

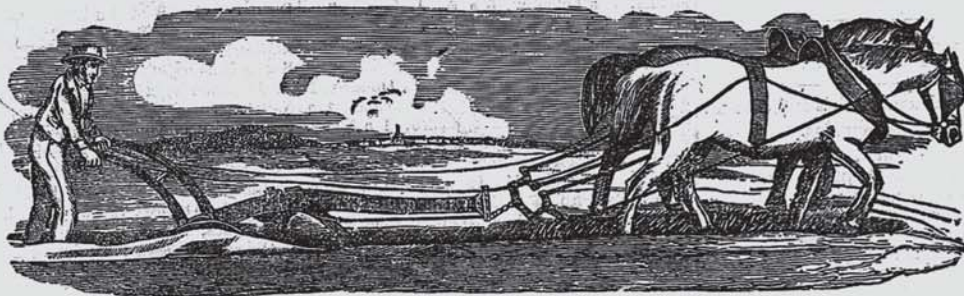
Let’s begin with the steps taken purposefully against this disease. For millennia doctors had drawn a connection between wounds and traumatic tetanus, on the one hand, and

'cord management' and neonatal tetanus, on the other. There is little to suggest that much changed drastically when it came to treating wounds until aseptic and septic practices became widely used in the 1870s, but traumatic tetanus made up only a small proportion of overall tetanus deaths. It does seem likely that violence, accidents and general 'disasters' causing major wounds declined in society during the 1800s, despite the risks of incipient industrialization (Read, 2009). Fewer wounds meant fewer cases of tetanus. Neonatal tetanus was a much bigger killer, but one that became far less so as the century wore on. Some doctors argued for novel theories of its origin, such as pressure from the occipital bones or blood poisoning. Yet the majority of doctors remained persuaded by the 'old opinion' related to umbilical cord procedures. Although obstetrics and pediatrics remained mostly in the hands of midwives during the nineteenth century, western medicine crept into the profession, especially when it came to risky cases and injuries. For example, the first ovariectomy was performed in 1809, use of diethyl ether as an anesthesia for childbirth began in 1842, and surgical correction of a vesicovaginal fistula was accomplished in 1845 (Duffin, 1999, p.242). While little evidence exists, we might deduce that both the recommendations made by public health officials and a general change in perspectives by mothers and midwives reduced the use of umbilical applications and improved general cleanliness during delivery and postnatal care.

Several unintentional actions and indirect changes in society may have had a more important effect in reducing the risk of this disease. First, Brazil remained a much more rural than urban country throughout the 1800s, but the general demographic shift from countryside to towns or cities began about the time that tetanus began to decline. As people moved from farms and hamlets into places increasingly populated, they simply had less contact with soil and manure. This is not to say that cities were clean places: authorities in Rio de Janeiro complained of 'mountains' of trash and excrement that collected in alleyways (Brasil, 1852). But in towns and cities, people farmed and gardened less and handled fewer domesticated animals. Second, mechanization in farming may also have lowered tetanus rates. Working with plows rather than hoes, for instance, may have meant less time or contact with dirt. Even though the majority of farmers in Brazil tilled with basic farming tools such as hoes well into the twentieth century, new agricultural instruments did find a market among the wealthiest plantation owners, as seen in an advertisement printed in the *Diário do Rio de Janeiro* in 1860 (Figure 3). Third, we know that the industrialization of clothing manufacturing made it much cheaper to wear clothing, thus better protecting skin from abrasions. By the 1860s, mirroring European styles, jackets and long overcoats became popular among the freeborn and slaves alike (Weimer, 1991, p.58). Shoes also seem to have become more common. The proportion of slaves, generally unshod, diminished and some of the remaining slaves took to wearing shoes (Read, 2012). This could have made a big difference because 37% of traumatic tetanus cases in the US between 1998 and 2000 were caused by a 'puncture' wound. Twelve percent resulted from a person stepping on a nail (Guilfoile, 2008, p.12-13). Finally, we should not rule out an environmental or biological change that lowered the levels of *C. tetani* or altered their virulence in humans. There is a surprising paucity of scientific research into the distribution of *C. tetani*. For example, I am aware of no studies on whether *C. tetani* levels change in soil over time.



# AOS SENHORES FAZENDEIROS



PRIMEIRO INSTRUMENTO DE LAVOURA.

A estampa acima apresenta um arado em exercicio; a vantagem de sua applicação é tão conhecida que seria ocioso descrevê-la. Quem tem conhecimento do amanho das terras por processos mecanicos, e procura utilizar-se delles, colhe um lucro incalculavel, já em relação á maior produção, já á qualidade desses productos.

Hoje já vai sendo entre nós conhecido este tão poderoso auxiliar da lavoura, e vamos vendo o desejado resultado em sua aquisição.

No entanto ainda se torna necessaria toda a coadjuvação dos Srs. fazendeiros, instruindo-nos com suas luzes, naquelles melhoramentos que julgarem apropriados ao terreno deste paiz.

É, pois, sempre com dupla satisfação que o annunciante, que tanto ha dedicado-se a este importante ramo, acolhe sempre essas judiciosas observações, filhas da pratica, para continuar a fazer executa-las perante essas importantes manufacturas de mecanica agricola, para melhorarmos quanto seja necessario para seu completo emprego.

As machinas de lavoura, importadas ultimamente, attestam quanto á vantagem de assim proceder. Essas machinas, hoje mais aperfeiçoadas e simplificadas, offerecem um emprego seguro a uma lavoura que ora começa a olhar para esses auxiliares, ainda de maior riqueza onde a falta de braços cada dia se torna mais sensivel.

O annunciante, empenhando-se quanto possa em concorrer para esse verdadeiro progresso, conta bem merecer, e nesse intuito a franqueza e lealdade de suas vendas, que garante, lhe asiançarão a maior preferencia.

Expõe uma parte do catalogo de machinas e instrumentos de agricultura e horticultura, e mais utensilios concernentes á lavoura.

**Arados** de revolver a terra, simples ou com roda e facão.

Ditos de dito, do de cama de virar, servindo para terreno montanhoso ou varzea, simples ou com roda e facão.

Ditos para preparar o terreno para plantação de canna.

Ditos para capinar, mesmo entre o cultivado.

**Arrancadores** de raizes de 2, 3 e 4 dentes, de grande força.

**Grades** triangulares e quadradas.

**Cultivadores** para alisar o terreno.

**Semeadores** para milho e outros grãos.

**Ventiladores** para café com separação, e peneiras tambem para limpar milho, feijão, arroz, &c.

**Moedor** de sabugo, raizes, &c., para alimentação de animais.

**Moinho** para moer milho.

Dito para fazer fubá.

**Debulhador** do milho simples.

Dito de dito com debulhador e separador.

**Debulhadores** de ferro, novo systema, verticaes, de cavalete e de Virginia.

**Trades** de abrir em poucos minutos buracos na terra, de grande profundidade.

**Machinas** de thermometros para fazer manteiga.

Ditas de prensar queijos.

Ditas de picar e applicar carne para salchichas.

Ditas para extrahir o suco da canna para assucar.

Ditas para cortar canna.

Ditas para cortar capim para dar aos animais.

**Picaretas, Machados e Pás** para excavação.

**Carros** de mão para aterro e jardins.

Ditos de transportar pesos.

**Ancinhos, Fouces, Podadeiras e garfos.**

**Cangas** para bois, argolas e mais pertences.

**Serretes, Alviões e Pás** de madeira e de ferro.

Outros muitos machinismos tendentes á agricultura e horticultura se acham expostos á apreciação dos Srs. fazendeiros, a quem pedimos a bondade de vir examinar este especial deposito de — **Machinas de Lavoura.**

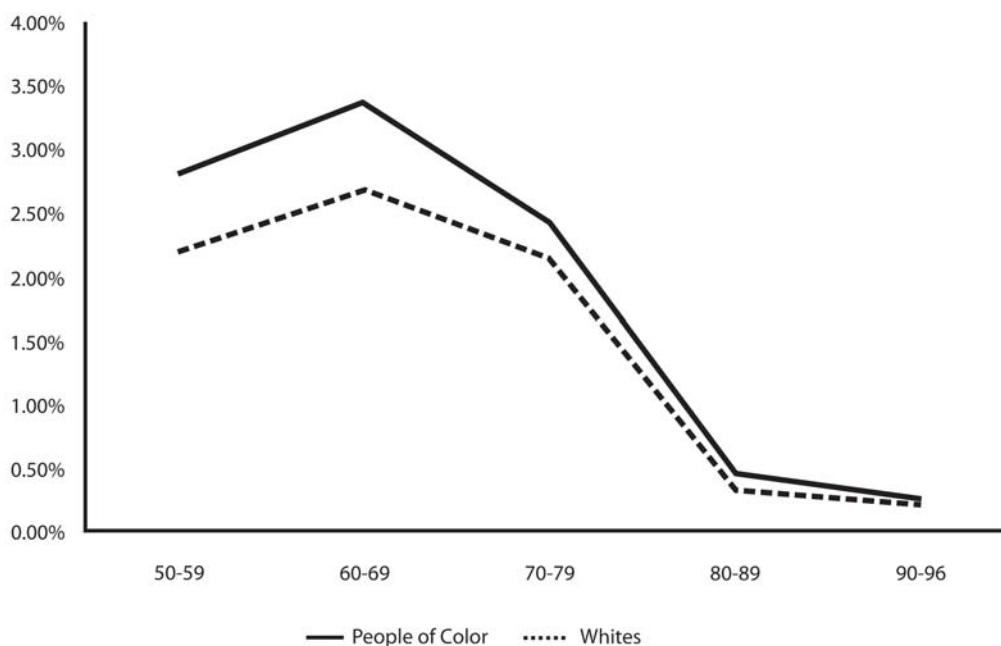
**MANOEL OLEGARIO ABRANCHES**  
**10 RUA DA ALFANDEGA 10**  
**DEFRONTA DO BANCO DO BRAZIL**

Figure 3: New agricultural instruments (Diário do Rio de Janeiro, 1 maio 1860, p.4)

The fact that rates of tetanus fell less for slaves than the freeborn begs us to ask if general conditions of slave life and their treatment did not improve after 1850. With the end of the international slave trade to Brazil, slave prices certainly climbed, giving an economic reason for masters to better 'look after' their chattel. Were slaves given greater medical attention and their newborns better postnatal care, for example? Evidence from Santos suggests that treatment did improve, but in a way that may have widened the gulf between slaves owned by wealthy and high status families and those owned by poor and low status families (Read, 2012).<sup>17</sup>

The relatively small number of remaining slaves in Brazil were freed in 1888. What happened to their descendents, at least in regard to tetanus? Or in other words, what were the relative risks of tetanus to the children of the children of the slave Victorino, if such a lineage existed? Despite the patriotic celebrations of abolition and the new Republic's (1889) many promises to correct the inequities of monarchical society, racism and economic discrimination remained firmly entrenched in Brazilian society. Thus, the descendents of slaves struggled on in many of the same ways their enslaved mothers and fathers had (Butler, 1998). Yet, at least in Porto Alegre and Santos, it appears that the gap of incidence of tetanus that existed between whites and people of color during the nineteenth century, aided by the large difference between slaves and freeborn, diminished or vanished by the twentieth century. Graph 2 demonstrates this among whites (*brancos*) and people of color (including *pretos*, *pardos*, *morenos*, *caboclos* and others) in Porto Alegre. Among 380 deaths that occurred in Santos in 1906, five people died from tetanus, all listed as white. By this point the proportion of white European immigrants had greatly expanded in Santos, but

**Graph 2: Tetanus deaths as a percentage of total deaths, 1850s to 1890s, Porto Alegre**



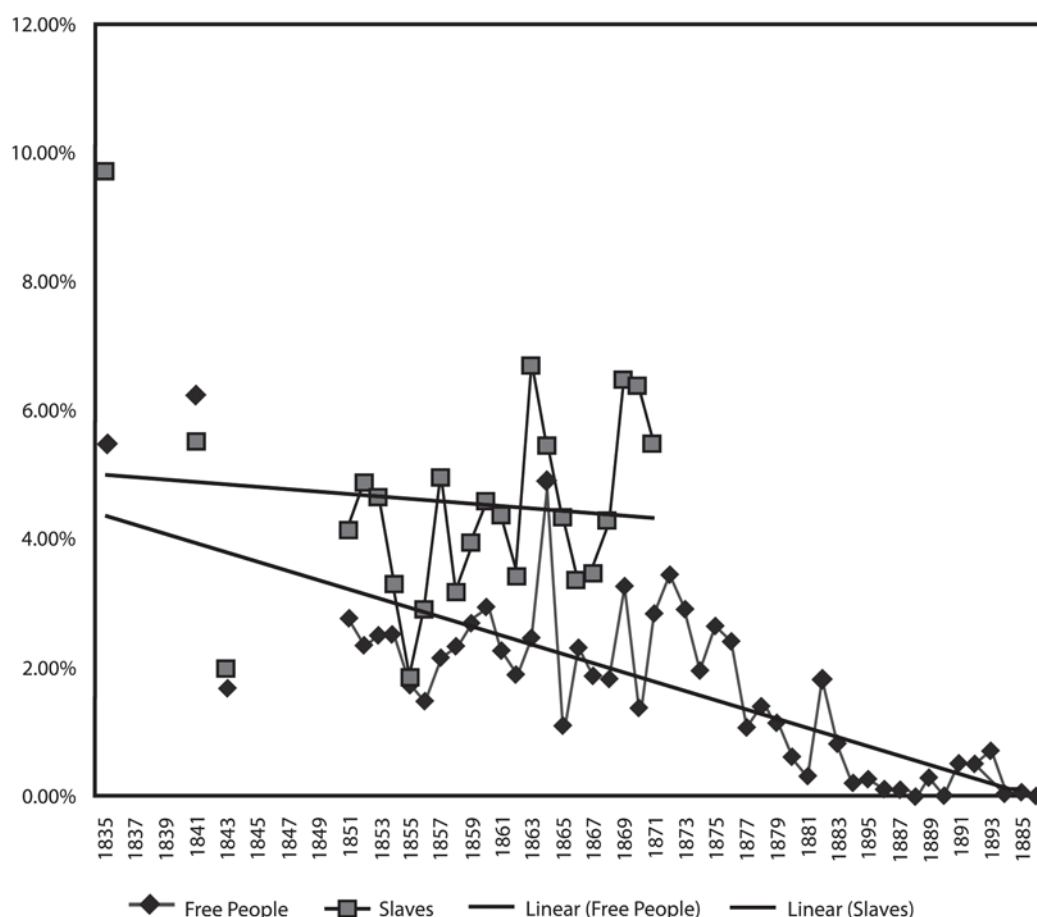
Source: Compiled by the author based on death records from Santa Casa de Misericórdia de Porto Alegre (Santa Casa..., 1850-1896)

it remains a surprise that no people of color were killed by tetanus within this (admittedly small) sample.

Perhaps the largest consequence that tetanus had on Brazilian society was demographic. Brazil grew during the turn of the century, aided by immigration. Approximately 2,740,000 immigrants entered Brazil between 1887 and 1914, at an average rate of 100,000 per year (Fausto, 1999, p.166). Between 1872 and 1890, the Brazilian population increased at an average of 230,000 people per year. From 1892 to 1910, the rate of population growth again jumped, with about 405,000 more people in Brazil each year because of childbirth or immigration. Based on these numbers, immigration contributed about a third of the total increase, a less important role than the 'natural' increase within Brazil. This fact, it must be added, is rarely acknowledged in the literature, nor is the role that health may have played.

Consider tetanus, one of the most important causes of infant deaths during the early to middle part of the 1800s. On average, tetanus comprised between 10% and 60% of all deaths of babies under one year of age, depending on the location and social group. Because of the high ratio of deaths to births, including those caused by neonatal tetanus, the Brazilian population grew more slowly and unevenly. After 1870, infant mortality improved in many parts of Brazil, but not all. In Porto Alegre, between 1835 and 1870, 9.4% of total deaths were infants. This fell to 7.8% between 1871 and 1896. As shown in Graph 3, one of the most rapid periods of decline in infant mortality occurred between 1875 and 1885, at the same time that there was a big fall in neonatal tetanus. In Niterói, the percentage of deaths of children 15 years old and younger dropped four points between the 1857-1870 and 1871-1890 periods. Bahia and Recife in the Northeast do not demonstrate notable improvements between the 1850s and 1870s and it may not be a coincidence that these cities were also becoming relatively poorer compared to several cities in the south.<sup>18</sup> Yet money may have mattered less if we consider São Paulo. There, child mortality (defined as newborn to five years old) climbed from 32.7% of total deaths in 1872 to 60.7% in 1906, despite wealth generated by the coffee boom. Common sense suggests that mortality rates generally improved in Brazil when several endemic diseases lessened as threats. Additionally, new epidemiological knowledge and urban sanitation campaigns reduced the toll of serious infectious diseases, such as smallpox and yellow fever. Lower death rates probably combined with higher fertility to spur the natural increase. In a few of Brazil's largest cities, however, we may find more evidence that conditions worsened under the heavy weight of uncontrolled urbanization and laissez-faire attitudes.

The far more populated, urban nation that Brazil became in the twentieth century may be attributed in some part to the decline of a disease that is so much less feared today that one prominent Brazilian medical journal recently published an editorial titled "Trismus, opisthotonus and risus sardonicus: Who remembers this disease?" (Tapajós, 2011). That tetanus could have had such devastating effects not long ago but is largely 'forgotten' today suggests two things for the history of health and medicine in Brazil. First, mostly unknown but widely lethal endemic diseases (or broad categories of diseases) of the nineteenth century such as tuberculosis, gastroenteritis and parasitic worms need our attention. Second, more can come from research that looks at Brazil broadly and contextualizes it internationally.

**Graph 3: Tetanus and infant deaths as a percentage of total deaths, 1850-1898, Porto Alegre**

Source: Compiled by the author based on death records from Santa Casa de Misericórdia de Porto Alegre (Santa Casa..., 1850-1896)

## ACKNOWLEDGEMENTS

I am indebted to Glynnis Kirchmeier and Kaori Kodoma for fine research assistance and editorial support. Final drafts were closely read and corrected by Michael Weiner and Kay and Edward Read.

## NOTES

<sup>1</sup> George Gardner, a Scottish botanist, visited the area in Piauí where Victorino worked in 1839. While it is uncertain whether Gardner met José de Araújo Costa, he did rest for the night at his estate 'Canabrava.' Even though his description of the farm is short, the botanist gave many details of the area, including of the slaves who worked there (Gardner, 1849, p.181-190).

<sup>2</sup> The main facts of this story are confirmed by the historical record. We know that Coronel José de Araújo Costa personally whipped Victorino in April or May 1856 and the slave died from tetanus and gangrene not long after. As a consequence, the wealthy landowner defended himself in court against a charge of assassination made by Antonio Coelho Rodrigues. Rodrigues would later be called an abolitionist. Araújo Costa won his case with support of the civil law code that stated that men had the right to apply "moderate punishment" to slaves and other dependents (Brasil, 1860, p.84). From the witnesses of the calumny trial we know that a man (presumably a doctor) who was called to treat Victorino diagnosed tetanus. A few details of the story, however, are based on assumptions that require disclosure. We do not



know what the weather was like on the day Victorino was whipped, but May is often a relatively cooler month in Piauí. The bondsperson may or may not have shown the most common symptoms of tetanus (i.e., *opisthotonos*, *risus sardonius*) and gangrene.

<sup>3</sup> Cristiano Benedito Ottoni, senator of Minas Gerais, declared in 1884: "I, Mr. President, have completed my seventy-third year; for more than half a century I have had the full use of my faculties. I see, I hear, I observe, and I can bear witness that the treatment of slaves in Brazil has gone on steadily improving" (translated and quoted in Andrews, 1887, p.315). Agostinho Marques Perdigão Malheiro (1866) saw evidence of an improvement in general treatment of slaves in the disappearance of iron face masks and foot shackles. He also took note of many more slaves well-dressed and shod (see p.114-115). Emília Viotti da Costa (1997, p.324) agreed with these contemporary assessments.

<sup>4</sup> See, for example, Anderson (1986, p.296), Costa (1997, p.313), Karasch (1987, p.153-154), Moura (2004, p.136) and Peard (1999, p.99).

<sup>5</sup> In large clinical series of neonatal tetanus that occurred in the 1980s, "cessation of suckling is reported in 69% to 100% of clinical cases, rigidity or spasms in 97% to 100%, and *trismus/risus sardonius* in 71% to 100% of cases. Fever or umbilical sepsis is reported in 20% to 50%" (Galazka, Stroh, 1986).

<sup>6</sup> In this and other citations of texts from non-English languages, a free translation has been provided.

<sup>7</sup> It is important to ask whether or not cases of tetanus were accurately counted by church and government officials. We might assume that among slaves and the newborn, there was less incentive to report a death than a freeborn adult. I do not believe this to be true, mostly because in this deeply Catholic country, people were concerned about the fate of souls. An unbaptized baby, and thus one unregistered by parish officials, was excluded from heaven according to many (The Catholic..., 1913, p.258-259). Parish records appear to show parents and family members going out of their way to have deaths registered, including those from tetanus. A fuller discussion of this issue can be found at: <http://www.empireofbrazil.org/storage/A%20Note%20on%20Possible%20Bias%20in%20Obit%20Data%20on%20Tetanus.pdf>. (Access on: 19 July 2011).

<sup>8</sup> Shoes were a marker of freedom during slavery, yet this was not a hard and fast 'rule.' Some free people went without shoes and some slaves, such as domestic servants of wealthy owners, wore shoes.

<sup>9</sup> See, for example, the first page of "Miasmas, sua origem, seus efeitos..." within the provincial report from Alagoas (Brasil, 1855).

<sup>10</sup> Average levels of animal breeding (*zootécnica*) were measured by the per capita value of this industry for each municipality. To determine this, we divided the total income of agricultural input by the township's population in order to arrive at an indicator of farming's 'importance' regardless of population levels.

<sup>11</sup> In a series of ghastly procedures, Sims bored holes into the back of the skulls of living newborn babies in an attempt to relieve the pressure. Furthermore, as Stephen Kenny has argued, he only performed such operations on the heads of enslaved babies, viewing the procedure as too experimental for white infants (McMillen, 1991, p.304-307; Kenny, 2007, p.13).

<sup>12</sup> For example, Luiz, a 26-year-old runaway slave, was missing one of his index fingers. It had been amputated "because of the tetanus that he once had" (Diário..., 26 set. 1844).

<sup>13</sup> 'Balsam of Peru' is most likely copaiba, a substance that may have anti-inflammatory antiseptic properties (Veiga, 2001). Some historians have suggested that copaiba played an important role in ending an epidemic of neonatal tetanus on the Westman Islands during the mid-nineteenth century (Jacobsen, Hem, Sigurdsson, 2011, p.704-705).

<sup>14</sup> "Treatment of *Trismus neonatum* by Mr. [Johann Adolph?] Pitschaft. In the case of trismus with convulsions in an infant with normal bowel movements, Mr. Pitschaft uses a mixture of the following: Hydrolat of orange flowers 60 grams – Magnesium carbonate 1 gram – Select musk – 5 centigrams – Almond syrup – 8 grams – M.S.A. [*misce secundum artem*, or 'mix accordingly'] – Give with a teaspoon of soup or coffee every hour, according to age."

<sup>15</sup> Data from Santos include enslaved patients and free patients (Santa Casa..., 1861-1883). Data from Porto Alegre include enslaved and free patients (Santa Casa..., 1845, 1856, 1866, 1875 and 1883).

<sup>16</sup> One study on neonatal tetanus in rural Pakistan claimed that the traditional practice of applying ghee – a type of clarified butter – to the umbilicus is an important risk factor. Several of the original authors of this study, however, later argued that ghee by itself was not dangerous, but becomes a risk factor when heated with dry cow dung, a common fuel in rural Pakistan (Traverso, 1989; Bennett, 1999).

<sup>17</sup> Scholars have done very little on this question, despite the fact that it speaks directly to the common experiences of slaves. Since the 1970s historians have ignored 'treatment' often because it implies slaves had no or little agency. Today, there are no serious scholars who argue slaves were generally indolent and kowtowing 'Sambos' without much of a will or need to resist. Time has come to renew our attention to the actions and agency of slave owners. Doing so does not privilege the history of slaves' oppressors, but allows us to see the continually negotiated relationship of slaves and their owners as a complete equation.

<sup>18</sup> Maria Luiza Marcílio (1993) found that mortality rates exceeded fertility rates in Rio de Janeiro from 1830 to 1900. The city's rapid growth was driven by arriving African slaves, European immigrants and Brazilian migrants. She also found that infant mortality fluctuated considerably between 1859 and 1877. According to figures reported by the Imperial Minister, the proportion of child deaths (defined 0 to 4 years) fell compared to total deaths between 1870 and 1885, but at a very uneven pace.

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