John would soon be knocking to say the cab was waiting. She sensed her irritation mounting, but she had no intention of rising to this bait. Eventually the Matron stopped kvetching.

After a few more moments, the Superintendent began speaking calmly to Mrs. Clarke. Everything is sorted. Lady Canning knows which patients will be ready for discharge after their two months in the institution and will present their names to the Committee when it meets on Fridays and Mondays. The Quarterly Report is in the Committee’s hands. They have been informed that you want to return to Yorkshire, so the search for a new Matron will be set in motion when summer holidays are over.

The three nurses have also been informed that they will receive daily instructions from the Matron for a fortnight or so. They should be able to manage the patients on their respective floors without assistance. The lift from the kitchen is working fine. Good that the boiler in the attic was repaired so that hot water is again available on
August 31, 1854

A warm afternoon. Won’t need the shawl for long. Two quick left turns and she was east-bound on Weymouth Street.

She didn’t look back.

* * *

Mary Jarrow untied the string around the hospital’s blue-gray folder containing papers about the young woman in the black silk dress sitting across from her. She gave her a quick glance: Seems very relaxed, self-absorbed even. No matter. This was no interview. Everything was already arranged. She opened the flap of folder and extracted the contents.

It was a thin file. Topmost lay a generic circular sent to all metropolitan hospitals at the end of 1853, requesting information about the organization of nursing services, spiritual instruction and lodging offered the nurses, and annual income of nurses at all levels of experience. Jarrow remembered it well. She unfolded a foolscap copy of the report prepared largely by herself, gave it a rapid scan, refolded it, and turned to the next item.

Jarrow carefully re-read a letter of reference in which hospital administrators were...
August 31, 1854

requested to send Miss Florence Nightingale the desired details and permit her to make personal inquiries thereafter. Hers was by no means an official or governmental investigation of private hospitals. The purpose is solely to collect data on the current state of nursing training and services which she will analyze in advance of preparing a report that may include recommendations for improved nursing care and education. Findings about individual hospitals will remain confidential; any policy recommendations will be made solely on the basis of aggregate data.

The letter underscored Miss Nightingale’s qualifications to undertake such a study. It had taken her less than six months after appointment as Superintendent of the Establishment for Gentlewomen during Illness in Upper Harley Street to revamp the institution into a small private infirmary with a matron and small nursing staff who provide efficient and sanitary medical care. She had supervised the remodeling of new premises according to a rational plan of her own devising: a nursing station on each of the three floors; bells from each patient room to this station to alert the nurse when assistance was required; and two time-and-energy saving conveniences – a mechanical lift to convey meals to each floor, and hot-water outlets on each floor, gravity-fed by a boiler in the attic. Moreover, she held the nurses under her supervision to high standards. Her assumption is that each is a Sister in training.

Miss Nightingale had developed her notions of effective nursing over many years. In 1847 she had undertaken a systematic study of Parliamentary Blue Books on public health, the two Reports of the Health of Towns Commission, as well as those by various sanitary commissions and annual reports from municipal hospitals throughout the United Kingdom and the Continent. Previous to her superintendence at Upper Harley Street, she had visited the Institution for Practical Training of Deaconesses at Kaiserswerth on the Rhine, written a pamphlet to introduce the Kaiserswerth model to English women interested in nursing, and spent several months there as a trainee herself. In addition, Miss Nightingale had made investigative visits to the major hospitals in Berlin and nearly every hospital in Paris, including the Salpêtrière and the one managed by the Sisters of Charity.

The letter concluded with a few biographical particulars of note: Born 12 May 1820 – that would make her 34 now; Father, William Edward Nightingale, gentleman, of Embley Park, Hampshire, and proprietor of the manor and lead mines at Lea Hurst, near Matlock, in Derbyshire; Mother, Fanny Nightingale, née Smith, daughter of William Smith, Member of Parliament for forty-six years; privately tutored in mathematics and the classics, mainly by her father.

Florence Nightingale sat patiently whilst the Matron re-familiarized herself with the person who had volunteered to substitute as a Sister to
superintend nurses on one of the hospital wards. This Matron was thorough and conscientious – characteristically so, as Nightingale recalled from her investigations at Middlesex Hospital in the spring. She had memorized the Matron’s personal particulars: born 1811 in St. Andrews, Scotland; service at Morningside Lunatic Asylum in Edinburgh before coming to London and the Middlesex.

Nightingale was there that afternoon only because she had decided to hedge her bets if an administrative job at King’s College Hospital did not materialize to her liking. In June she had been thrilled when Mr. Bowman recommended her for the position of Matron/Superintendent of Nursing in that hospital’s projected re-organization plans. Nightingale heard nothing until July, however, when she was asked to submit her conditions of employment. She minced no words: re-training of currently employed nurses, including spiritual instruction; a school to train novices; sleeping quarters for all nurses on their assigned wards; substantial meals for nurses on duty; and augmented salaries so her nurses would not have to keep body and soul together by taking in needlework or prostituting themselves. Then came dispiriting interviews with senior medical doctors and surgical misters; disgusting displays of jockeying for power interleaved with meaningless compliments about her Sanatorium for sick governesses; rifts and intrigue amongst these leading men of King’s, some, like Mr. Bowman, who wanted a nursing superintendent with teeth, others a business-as-usual matron with minimal ward responsibilities. But Bowman was only an assistant-surgeon. They simply didn’t see that sub-standard nursing care would undo all the wonders they wrought in the surgical theatres and on the medical wards. Did they want King’s to become like Barts – a horrific place to work if a nurse, a virtual death sentence if a patient, for all its storied past?

Nightingale had stiffened her spine. She was simply not going to compromise on a nurses training school. If King’s won’t promise at least that, another London hospital surely would at some point in the near future. Perhaps the Middlesex, which had scored the highest amongst English hospitals in her recent survey of nursing services. A few weeks as Head Ward Nurse would prove that pudding, one way or another. If Middlesex Hospital fell short of expectations, then Westminster Hospital or St. Thomas’ were worth another look.

Nightingale was in no hurry. The notice she had sent the Ladies’ Committee permitted her to remain at Upper Harley Street through January, if she so desired. Any longer was unthinkable. Truth be told, she had known her Sanatorium would fall short of expectations when the Ladies’ Committee had dismissed her suggestion to relocate to the wing of a major hospital or the new hotel opposite Paddington station, where the patients would lie on bona fide wards. The
house in Upper Harley Street they were now using had been a private residence, so patients were in single or double rooms. This situation was completely unsuitable for training Sisters since major hospitals were organized on a ward basis. Nightingale’s contractual year was over. High time to move on to something that approximated here vision.

She didn’t have to kowtow to anyone, ever again. An allowance of £500 per annum from her father made her an independent woman.
August 31, 1854

SUPPLEMENT TO THE WEEKLY RETURN.

CHOLERA AND THE LONDON WATER SUPPLY.

It was shown in the Registrar-General's Report on cholera, that elevation of soil was the most constant and striking in its results; and that the influence of water supply, density of population, and wealth could also be detected in the progress of the epidemic. But it is evident that to estimate exactly the value of any of these forces, it is necessary to obtain similar conditions as regards the rest; for example, to measure the effect of good or bad water supply, it is requisite to find two classes of inhabitants living on the same level, moving in equal space, enjoying an equal share of the means of subsistence, engaged in the same pursuits, but differing in this respect—that one drinks water from Battersea, the other from Kew. If such a district as that of Russell and Tavistock squares, or such a rookery as that of St. Giles was in 1849, had extended from the river to Hampstead, the effect of elevation would have been less marked, though still considerable; whom happiness or misery has equalled, place would less exalt above its equals. But of such experimenta crucis the circumstances of London do not admit; for it will be found generally that the poorest and lowest, if not densest districts, use the worst water.
August 31, 1854

Previously, at the GRO on Thursday 24 August 1854

So far, the experimentum crucis is yielding anticipated results!

William Farr looked perplexed. What was Snow talking about?

Snow noted the uncertainty. He extracted a duodecimo-size pamphlet from an inside pocket of his black, single-breasted frock coat, turned to one of the middle pages, and laid the pamphlet on the table in front of Farr: “Supplement to the Weekly Return. Cholera and the London Water Supply.”
A rather routine report, Farr recollected, put together by himself and his team of GRO clerks last November as the first wave of the present cholera epidemic was subsiding. Just a prose and tabular summation of information that private companies supplying the metropolis with potable water had provided in response to an inquiry sent out under the signature of his boss, the Registrar-General.

Farr looked up. He had not taken the point. Snow tapped his finger at two passages, lightly ticked in the margin, on the first page of the Supplement:

to measure the effect of good or bad water supply, it is requisite to find two classes of inhabitants living on the same level, moving in equal space, enjoying an equal share of the means of subsistence, engaged in the same pursuits, but differing in this respect, — that one drinks water from Battersea, the other from Kew. . . . But of such experimenta crucis the circumstances of London do not admit . . . .

He got it.

John Snow had observed something that had escaped Farr whilst putting the Supplement together: the makings of a natural experiment already existed that should establish, once and for all, the role of impure water in the propagation of cholera. Farr listened intently as Snow described the investigation he had begun a fortnight ago. The good Doctor sitting opposite was certain his inquiries would confirm his notion that there was a single exciting cause of cholera — the mediate-contagion hobby he’d been riding for, gracious me, almost five years to the day.

But if truth be told, Farr had been riding a theoretical hobby-horse of his own much longer.

**Zymotic diseases and elevation**

William Farr became involved with the General Register Office around July 1837, when it began managing the registration of births, deaths, and marriages in England and Wales. He saw in this new institution an opportunity to implement principles of hygiene and statistics he’d confronted during a short period of medical study in Paris before fulfilling requirements for admission to the Worshipful Society of Apothecaries in England. Then, after just two years of scrounging a clinical practice in London, the first Registrar-General made him an unrefusable offer: a position as Compiler of Abstracts in the Statistical Department. He quit the practice of medicine in a heartbeat.

Farr and his small team of clerks were responsible for tabulating quarterly reports from the vital data submitted by about two thousand district and sub-district registrars, appointed either by Boards of Guardians from the recently established Poor Law Unions or vestrymen in those parishes temporarily exempted from the new Poor Law. He supervised production of the first annual GRO report to Parliament in 1839. When
metropolitan registrars began submitting weekly reports in 1840, Farr’s small division added the Weekly Return of Births and Deaths in London to its regular output. New tasks were piled onto his capable shoulders: assisting with the first national census of 1841, then quarterly reports for England and Wales, plus special reports whenever Parliamentary Committees requested them. In 1842, a new R-G promoted him to superintend the entire Statistical Department.

Farr used the fourth annual Report of the Registrar-General (1842) to set forth a revised “statistical nosology” for categorizing causes of death in all subsequent GRO publications. His obsession was to make the central bureau in the national system of registering vital statistics a model of Benthamite reformism. Data collected on births and deaths would be statistically manipulated to produce a scientific understanding of human health and sickness. Quarterly and annual reports should be pitched at a level comparable to official Blue Books and provide the kind of data and analysis Parliament needed to make laws that yielded the greatest good for the greatest number. The Weekly Reports should guide the medical profession in a parallel quest.

To actualize the Benthamite greatest happiness principle, it was essential to understand causes of death, especially premature deaths. Early in 1842 Farr realized that his previous manner of listing communicable diseases — “epidemic, endemic, and contagious” — was outmoded.

How could he, in a time of rapid revolutionary reforms in medical thinking and practice, effectively analyze morbidity data organized under a rubric that called to mind centuries-old disputes about disease causation. The hallmark of the new medicine of the mid-nineteenth century was a redefinition of itself as a science, informed by new research in allied sciences such as chemistry, biology, physiology, and pathology. Farr wanted a classification of diseases that fit the spirit of his age, not Sydenham’s.

His Eureka moment came later in 1842 whilst reading two volumes on agricultural and animal chemistry by Justus von Liebig. In them the German professor discussed new research in inorganic and organic chemistry, much of it accomplished in the laboratory he had established at the University of Giessen. Liebig distinguished between miasms and fermentations as causes of human diseases. When it came time to publish the Fourth Annual Report, Farr added an appendix in which he popularized Liebig’s distinction:

Miasms are poisons evolved during the process of putrefaction which cause incommunicable diseases like ague (malarial, intermittent fever), rheumatism, and neuralgia — without these poisons reproducing in the host. Carbonic acid (H₂CO₃) and sulphuretted hydrogen (hydrogen sulfide, H₂S) are examples of morbific miasms, defined as poisonous gases given off by putrefying organic matter that may cause disease if inhaled.

Fermentations, however, are a class of diseases caused by singular organic molecules
of unknown chemical composition which, if they gain entry to a host and encounter corresponding molecules in the blood, are transformed into exciters — agents directly responsible for causing particular diseases. Unlike miasmatic poisons, fermentation-exciters reproduce themselves at the expense of the host’s organization. Once established in the host, these exciters can propagate to other hosts by contagion (such as inoculation and contact) or infection (inhalation).

Laboratory analysis had shown that the addition of yeast to wort containing gluten and sugar produces more yeast, alcohol, and carbonic acid. Although the precise chemical mechanism remained unclear, Liebig hypothesized that yeast is gluten in a state of transformation — a potential exciter. That would explain why yeast induces fermentation when, if temperature and moisture content are conducive, it encounters organic molecules similar to itself in the gluten.

Farr’s variation on this explanation by Liebig was to equate miasms with conventionally denominated endemic diseases, fermentations with well-known epidemic and contagious diseases, and to rename all of them zymotic diseases. Farr hypothesized that each of these diseases had a specific exciting cause or principle, a zyme, probably (in his mind) an unorganized, non-living organic poison that induced a morbific physiological process analogous to fermenting yeast. In the case of endemic diseases, miasmatic exciters were locally prevalent, albeit seasonably variable. Most contagious and epidemic exciters, however, were propagated person-to-person, either by contact or the inhalation of effluvia emanating from the bodies of the sick; place and time were inconsequential or, at most, incidental. Farr thought it likely that the blood is the primary seat of all zymotic diseases, pathological manifestations in other organs secondary. Similar causation mechanisms and physiological properties swayed Farr’s decision to place all these diseases in one statistical category that could be disaggregated as needed.

*Zymosis* in Greek means fermentation; Hippocrates employed it, as had Thomas Sydenham, the revered English clinician and medical theorist. Good enough pedigree for William Farr, whose right foot was firmly grounded in medical knowledge of yore. He expanded the meaning of *zymosis* to include “the morbid process and their exciters,” an obvious reference to Liebig for the modern, scientific-minded medical man. Farr then proposed scientific names for about a score of diseases in the zymotic class. He also coined names for the corresponding principles existing in the environment which became morbific exciters of disease when they encountered replicas of themselves in a host and initiated pathological transformation of tissue. Thus, the zymotic term for smallpox was *variola*, and *varioline* its morbific principle;

for cowpox, *vaccinia* and *vaccinine*;
for measles, *rubeola* and *rubeoline*;
for cholera, *cholera* and *cholerine*; etc.

Although Farr listed distinct exciters for each *zymotic* disease, he admitted that disease specificity was more a statistical convenience than accepted medical opinion.

It was possible that some exciters were inter-convertible, others structurally quite similar (which might explain why vaccination often prevented smallpox), and a few were even capable of producing diseases that had no exciters of their own. Both *enterine* and *cholerine*, for example, seemed at times to be sufficiently transformed within some human hosts to excite no more than ordinary diarrhea; this *zymotic* affliction *sans* exciter never eventuated in either dysentery or cholera. Yet diarrheal symptoms usually preceded both dysentery and cholera, leaving the clinician to wonder if the presentation was premonitory of either disease, and Farr, the medical statistician, unsure how he should categorize diarrhea when given as a cause of death in times of cholera.

The 1848-49 English cholera epidemic proofed Farr’s pudding. He and his team of clerks at the GRO spent more than two years analyzing mortality data submitted by registrars throughout the country and by meteorologists from the Royal Observatory in Greenwich. G.R.O staff looked for possible correlations between cholera mortality and natural variations in atmosphere and place, between mortality and social conditions such as age, gender, income, occupation, street widths, and population density. Farr wrote a hundred-page overview of their findings. His team produced 400 pages of supporting documentation for the full report on the 1848-49 epidemic, published as a separate volume in 1852.

Only geographical distributions correlated, statistically, with cholera mortality. The GRO identified nine particularly intense “cholera fields” in England, each centered on a port town or city. In general, coastal areas experienced three times the cholera mortality suffered at higher, inland locations – a pattern that replicated scantier data gathered during the 1832 epidemic.

Farr decided to concentrate on just one of the cholera fields, the thirty-six registration districts in metropolitan London. He found no significant correlation between mortality and population density at the district level. What seemed like a promising inverse relation between cholera mortality and taxable property values proved insufficiently strong to generate a law of disease. But the relation between a district’s elevation above the high water mark of the Thames and the number of cholera deaths in that district compared to other districts was sufficiently promising for Farr to generate an explanatory law.

The architects who delineated metropolitan registration districts in the mid-1830s followed contours of the winding Thames. South
of the river, the flood plain extended many miles before giving way to the low hills of Streatham, Dulwich, and Norwood. North of the river, however, the land rose gradually. The aggregated mortality rate for southern districts was considerably higher than any north of the Thames. What, wondered Farr or one of his clerks — a baker’s dozen of whom were crunching numbers every which way to Sunday — would a reconfiguration of districts into twenty-foot terraces reveal about cholera mortality? When completed, a tantalizing indirect relation appeared.

Farr then used this equation to calculate predictive mortality rates per 10,000 residents at each of the eight elevations and then compared them with the rates in the reported column:

<table>
<thead>
<tr>
<th>Feet above Thames HWM</th>
<th>Reported cholera mortality/10,000</th>
<th>Calculated cholera mortality/10,000</th>
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<tbody>
<tr>
<td>0</td>
<td>177</td>
<td>174</td>
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<tr>
<td>10</td>
<td>102</td>
<td>99</td>
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<td>30</td>
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<td>100</td>
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<td>20</td>
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<tr>
<td>300</td>
<td>7</td>
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</tbody>
</table>

Three of eight correlations were spot on, two within a 3% variance, none exceeded 18%. Although probability was the best anyone could hope to uncover when studying complex biological and social systems, Farr had a hunch he could account for the differences between reported and calculated mortality rates at each terrace. He was jubilant. The GRO team had figured out that in London, where people happened to live largely determined their likelihood of dying from cholera. This statistical law of cholera dispersal meant that future sanitary
policies in the metropolis could be formulated on a rational, scientific basis. Moreover, analysis of returns from the entire kingdom showed that the law was generally applicable: “The epidemic began and was most fatal in the ports on the coast; and in ascending in the rivers step by step, we saw it grow less and less fatal. . . The more exact information which we possess respecting the London districts establishes this connexion beyond doubt. . . [I]t will always be the general rule that the mortality of cholera is inversely as to the elevation of the people assailed above sea level.” Jeremy Bentham, mummified and seated in a cabinet at University College, two kilometers to the northeast of Somerset House, would have been impressed by this incarnation of his belief in the power of statistics to reveal patterns in a mass of collected facts.

In the 1852 report, Farr incorporated the GRO team’s findings on the influence of elevation for the propagation of cholera into an explanatory zymotic dispersal-mechanism for metropolitan London. He proposed that the Thames teemed with miasmata, nonliving particulate products of organic decomposition. They came from the alluvial soil lining its banks, by-products from London’s tanneries and slaughterhouses, and sewage draining from various parts of the metropolis. Whether Thames miasmata remained in the river or became airborne largely depended on complex contingencies inherent to the epidemic constitution: interactions among differences in land, air, and river temperatures at different times of the day; barometric pressure; dew point; humidity; and wind direction and speed as measured at ground levels.

The River Thames also teemed with zymotic material, especially non-living organic molecules of cholerine, endemic in English waters since 1832. Farr believed that cholerine in water has no deleterious effects on human health as long as it remained dormant. But statistical analysis of mortality data suggested strongly that cholerine becomes potentially morbific at water temperatures above 60° Fahrenheit if — and it was a critical if — the water contains sufficient amounts of decomposing organic matter. According to the zymotic hypothesis, harmless cholerine molecules would only metamorphose into exciters if they encounter organic detritus undergoing pathological transformation. Human excrement fit that bill of laden. A process of continuous molecular change gets underway as the cholerine proliférates exponentially, reproducing at the expense of corresponding molecules in the excrement. If the dew point over the River Thames drops sufficiently to produce one of the infamous London fogs, volatile activated cholerine and miasmata lift from the surface of the river and spread as prevailing winds dictate.

Everyone in the path of an effluvial London fog inhales activated cholerine, but not everyone comes down with cholera. In fact, most do not. Farr believed his hypothesis had the legs to
explain this observed fact. First and foremost, the elevation law reflected a natural process. Simply put, river-induced fogs nearly always settle onto low-lying districts in the flood-plain and dissipate in direct proportion to increasing altitude above the high water mark. If the fogs contain invisible choleraic matter, one would expect more deaths in low districts than those situated higher and farther from the Thames. Elevation with its varying degrees of exposure to effluvial fogs seemed to be the principal contingent factor in this zymotic disease. But there were others.

Old-timers spoke of choleric constitutions as a predisposing cause for various diseases. Although humoral medicine was giving way to cell theory, there was still too much mystery to be seen when squinting through a microscope for Farr to jettison Sydenham’s seventeenth-century physiological terminology. In the 1842 nosology, reprinted in the report on the 1848-49 cholera epidemic, he thought it justifiable to state that “Sydenham referred, in the following passage, to zymotic diseases . . . :

If the humours are retained in the body beyond the due time, either (1.) because nature cannot digest and afterwards expel them, or (2.) from their having contracted a morbific taint from a particular constitution of the air, or (3.) lastly, from being infected with some poison . . . these symptoms . . . are in reality disorders that proceed from the essence of the species newly created to this pitch [zymine]; so that every specific disease arises from some specific exhalation, or peculiar quality of some humour [zymin] contained in a living body.”

Farr understood that many of his medical colleagues remained Hippocratic-minded, even if they no longer employed humoral terminology. So it seemed prudent to make Sydenham conform with new research in organic chemistry: if a person’s blood contained molecular bodies compatible with cholerine, cholera would likely develop. If absent, a person disposed of any inhaled cholerine through natural elimination pathways. When it came to cholera, constitutional predisposition either set the stage or gave you a free pass.

Environmental factors could mitigate constitutional effects of a weak inheritance. Anyone conceived under an unlucky star could still bolster natural healing defenses by following the sanitarian regimen for a robust animal economy: fresh air, a wholesome diet, pure water, good hygiene, regular exercise, plentiful rest, strengthening habits, cleanly surroundings, and proper sanitation. When choice dictates otherwise, however, the outcome could be dire. Intemperate consumption of liquor and fresh fruits and vegetables, weaken the animal economy as much as living in filth. Debilitating habits of any kind exacerbate weak constitutional predispositions and march even the strongest to the brink of disease.

The poorest among us, especially the urban poor, are already predisposed to illness by circumstances beyond their control. It infuri-
ated Farr that so little was being done to improve their lot. They often live in overcrowded and inadequately ventilated housing. Lack of money makes it difficult to purchase wholesome food or pay the rent required for housing with decent kitchen facilities. Consequently, their bodies were deficient in proper nourishment, resulting in the very pathological transformation of tissue that cholerine requires. Personal intemperance magnifies environmental predisposition.

The agent reproduces rapidly when internal environments are made fertile by constitutional and environmental predisposition. The body responds by marshalling its natural defenses. But if vomiting and diarrhea fail to eliminate the cholerine, the patient’s blood thickens and becomes congested with *virus*, an infectious form of cholerine that escapes into the surrounding air by exhalation and emanations from the skin.

When this happens, every Londoner with full-blown cholera becomes, metaphorically, a tiny effluent of the River Thames, exuding invisible mists of morbific cholera matter into the surrounding atmosphere. If the sick room or the hospital ward is well ventilated, choleraic effluvia dissipate and cause no harm. But when patients lie in overcrowded, filthy rooms, when the windows, if any, open onto a dingy muse, a dark alley, or a narrow street — then the situation is opportune for person-to-person transmission. Family members, neighbors, and other visitors inhale the infectious *virus* emanating from cholera patients. Thereafter, constitutional and environmental predispositions will determine, once again, if the newly infected also come down with cholera or escape its agonies.

So thought William Farr.

John Snow was unconvinced by William Farr’s *zymotic* theory as applied to cholera. In his view, new cases can only appear when people inadvertently swallow the peculiar morbid agent evacuated by previous sufferers. Call the agent *cholerine* if you wish; the term has the advantage of disease specificity, which medical evidence from Liebig’s laboratory bears out. But cholera requires a specific agent to cause the same disease in others.

Snow dismissed multicausality and predisposition as relics of humoral medicine, an erudite way of saying we have no idea why some individuals become sick while others remain well whenever cholera is epidemic. The *zymotic* hypothesis is promising, but why frame it as a modernization of Sydenham’s humoral thinking? Why accessorise the dress of this long dead medical emperor with Latin neologisms when it’s sufficient to trumpet Liebig’s new organic chemistry?

But Snow remained a gentle and sympathetic critic of Farr’s *zymotic* hypothesis. His former interpretation of cholera — that it propagates as an infectious contagion — had much in common with it.
Snow’s early thinking about cholera
In English medical terminology of the 1840s, the common feature of contagious diseases is transmission from person to person. Contagionists believed that cholera only appears in a new place after the arrival of people from other places where it already prevails. In ordinary contagion, the invisible disease matter that defines cholera is presumed to reproduce in the bodies of its victims and communicated to others via touch, fomites (porous substances that harbor morbid matter), or, most likely, infection.

Contingent contagionists borrowed infection as a mode of transmission to account for cholera outbreaks untraceable to atmospheric, non-contagionist causes. They hypothesized that in rare circumstances (contingencies), victims produce a virus during the course of the disease which suffuses the surrounding atmosphere via exhalation and evaporation from sweaty skin. Contingent factors then determine whether or not a healthy bystander comes down with cholera. A warm and humid sick room augments effluvial skin evaporation, thereby increasing the dosage of virus inhaled by those in close proximity to the victim. Overcrowding and poor ventilation multiply the risk of infecting anyone; but susceptibility is greatest when a person’s animal economy is already weakened by an unwholesome diet or debilitating habits. For then our natural healing forces are insufficiently powerful to withstand the cholera virus as it passes from the lungs into the blood, rapidly reproduces, and spreads throughout the body of a new victim.

After arriving in London in 1836, Snow gave little thought to Asiatic cholera until the autumn of 1848, when the Weekly Returns, daily newspapers, and medical journals ramped up reporting of another pandemic’s ineluctable progress toward the British isles. So, when medical society meetings resumed after the summer recess, the cholera was often on the agenda. Contagionists and anti-contagionists resumed debates with an intensity not heard since the first English epidemic of 1831-32.

At one such meeting in October 1848, Snow’s mind turned to 1832, when, as a senior medical apprentice in Newcastle-upon-Tyne, his principal had sent him to the nearby colliery village of Killingworth to treat victims of Asiatic cholera. The coal pits were foul; the cottages were small and overcrowded. Entire families often occupied one undivided room, with kitchenettes just a few steps from cots on which the occupants slept, the sick and the healthy lying cheek by jowl. It was surreal — explosive diarrhea had soiled linen, soaked cots, and pooled on the dirt floors, yet there was often no fecal smell in the room. Caregivers tended the patients, presoaked soiled linens for washing, and prepared meals. Those in the middle stage of this horrific affliction clamored for cold water, consuming glassfuls in the hope of easing muscle cramps in their lower
back and extremities. Relief was often short-lived, however, as most victims vomited and voided more liquid than they had just taken in. If patients didn’t rally at this stage, dehydration produced total collapse, signalled by a weak pulse, congestion, and cold, dusky blue skin on face and extremities.

**Snow’s 1848/49 cholera hypothesis**

Hold on! Cold skin. Snow remembered treating no cholera victims who were initially feverish.

Nature’s response to poisoning of the blood is fever, the signature of every epidemic disease transmitted by effluvial vapors. Nature’s signature response to poisoning of the alimentary canal, however, is vomiting and diarrhea. It suddenly occurred to Snow that perhaps the morbid matter of cholera has to be ingested, not inhaled, to do its mischief. If so, then cholera isn’t a contingent contagion, it’s an ordinary, simple contagion communicated when someone unwittingly swallows something contaminated by a cholera victim’s evacuations. For the morbid matter seems to reproduce in the gut and would suffuse the diarrheal discharges. Food can be a fomes if it absorbs or holds such matter; since evacuations in the rice-water stage are odorless and without color, even the most conscientious meal-preparer without readily accessible hand-washing facilities would be unaware that the contaminant was being transferred from patient to food to be eaten by those who lived with or even visited the patient. That mechanism would explain a propagated cholera outbreak.

Snow now understood why the cholera had never felled him in 1832. His vegetarian regimen was the saving grace. He was used to boiling his own potatoes — his main staple — in Newcastle because the cook at William Hardcastle’s house had refused to encourage such teenage rebellion in an apprentice. Consequently, at Killingworth he had prepared his own meals and neither partook of food when making his rounds nor ate anything prepared by someone else at the cottage where he slept. Moreover, Snow washed his hands frequently, a routine developed under his mother’s tutelage when he lived in York.

What and where he ate was immaterial if cholera were a disease of the blood, but determining if its primary seat is the alimentary canal. He may have inhaled effluvia whilst treating cholera patients at Killingworth, but that effluvia wasn’t infectious. Then it occurred to him that he also never drank Killingworth water without distilling it first from the apparatus he contrived and set it up in his room. Why couldn’t water be a fomes? If cholera matter could spread from person to person through food, it might do so as well via water. That would explain epidemic outbreaks that aren’t propagated, person to person. Water supplies contaminated by cholera discharges would spread the disease to people who had never touched the sick or their effects.
He broached these views in the winter of 1848-49 with two medical colleagues. They were underwhelmed and advised Snow to play this hand close to his vest until he had more convincing evidence to offer than the observations of a nineteen year-old surgeon-apothecary apprentice.

The waiting period turned out to be a short one. Cholera began showing up in London early in the summer of 1849, and that August he learned of two neighborhood outbreaks south of the Thames that suggested water-borne transmission. While investigating them he saw indications of substantially more cholera mortality in south London than north of the river where he lived and conducted most of his anesthesia practice. The *Weekly Returns* confirmed this impression: in the two waves of the current epidemic the southern districts racked up more deaths than the rest of the metropolis combined, even though they only accounted for a quarter of the total population. All the waterworks serving south London were located on a stretch of the Thames heavily polluted by major sewer outlets.

Bingo!

He put the finishing touches on a manuscript, hand-carried it to the publishing house of John Churchill, near his own Soho apartment, and paid in advance for a small number of impressions. *On the Mode of Communication of Cholera*, a thirty-one page pamphlet appeared in September 1849.

Reaction was ambivalent at best, derisive at worst: Perhaps, wrote one reviewer, but Dr. Snow had arrived too late in the day to trace the contaminated water in Horsleydown and Albion Terrace to previous cholera cases. He presents no evidence that could not be explained by the dissemination of effluvial gases, commented another. And what a peculiar notion that piped Thames water could spread cholera throughout south London. Yes, it’s polluted, horribly so. But come now! The sheer volume of water would render the *materies morbi* of cholera innocuous if it even exists in the river.

William Farr at the GRO, however, pricked up his ears. This chap’s doings are worth watching.

**Natural experiments in Exeter and Hull**

A few months later Snow brought him striking information about what had occurred when the towns of Exeter and Hull altered their respective water supplies between the 1832 and 1849 epidemics.

In Exeter, the number of deaths dropped from 345 during the 1832 epidemic to less than 20 in 1849. Dr. Shapter from that town suggested this improvement was due to the construction of water works on the river Exe since 1832, two miles above the tidal reach. In short, the water supply of Exeter was no longer vitiated by communal sewage contamination.

The population of Hull had not fared as well from sanitary improvements undertaken be-
between the two epidemics. New sewer drains carried half the town’s waste into the river Hull, well below the point where the machines of a new water works extracted water for its system of pipes supplying the town. The architects of this scheme did not worry about a confluence with the river Humber less than three miles downstream. It was only twice a day that the tide carrying salt water from the north Atlantic into the Humber reversed the flow of the Hull for many miles, past the sewage outlet and past the inlet line to the water works. The solution seemed simple enough; shut down the water works on a waxing tide, resume operation when it’s waning. Nonetheless, the town of Hull experienced ten times more deaths from cholera and diarrhea in 1849 than during the 1832 epidemic.

Farr considered Snow’s information on Exeter and Hull significant because deleterious effects of impure water seemed to explain some, although not all, cholera mortality in the 1848-49 epidemic, which the GRO had just begun to analyze. Physicians since Hippocrates had proclaimed impure water injurious to health. Polluted water could predispose someone to a variety of diseases, whether, as outlined in Farr’s 1842 nosology, a sporadic disease such as dropsy or any of the zymotic diseases. Amongst the latter, the specific nature of contamination was determining since, hypothetically, each zyme is only excited by molecular structures similar to itself. Excremental waste would qualify as a cholerine exciter since it contains partially digested food; any inadvertent increase in the sewage content of a town’s water supply would, statistically speaking, augment cholera morbidity and mortality, as well as that of other epidemic diseases. Inversely, enhancing the purity of the water supplies should decrease the amount of all disease since it eliminates one predisposing factor, among many.

Snow would have replied that excrement-laced water is certainly offensive to taste and sensibility, but drinking it won’t necessarily cause or predispose one to diseases like cholera. The natural experiments in Exeter and Hull show that sewage-contaminated drinking water increases cholera mortality only if the contaminant is very specific: waste from previous cholera victims.

Not necessarily, according to Farr. All we know is that purer water in Exeter was associated with much-reduced cholera mortality in 1849, whereas the introduction of river-polluted water in Hull was associated with horrific mortality.

When the Statistical Office clerks confirmed Snow’s other findings (but not his mono-causal theory), Farr penned an introduction to the GRO report on the 1848-49 epidemic that gave credibility to Snow’s fecal-oral hypothesis as a possible, but not sole, mechanism for the transmission of cholera:

That cholerine is an organic matter, cannot, I think, be doubted by those who have studied the whole of its phenomena and the general laws of zymotic disease. The great questions
remain — Is cholerine produced in the human organization alone and propagated by excreted matter? Is it produced and propagated in dead animal or vegetable matter or mixed infusions of excreta and other matter out of the body? Is it propagated through water? through air? through contact? or through all these channels?

Snow’s steed had entered the lists. He was ready to tilt, confident about his prospects, in part, because Farr had promised to be with him, albeit out of the limelight, facilitating and collaborating.

**Snow and Farr as collaborators**
Even though Farr considered Snow deucedly hard-headed when he insisted that cholera had a single cause, their agreement that the ingestion of impure water is potentially deleterious to the bodily economy made the two keen to cooperate. Both were sanitarians who saw a mutual advantage in working closely together.

With Exeter and Hull in mind during the early 1850s, they stood shoulder-to-shoulder in opposition to expansions to a sewage system that would spew even more waste collected from northern districts of the metropolis into the Thames near London Bridge. Just a short distance upriver lay inlet pipes of several private water companies supplying the western and southern districts. That is, the same water that had flushed water closets in one part of the metropolis was being piped into the courts and kitchens in other parts — a feedback loop that prevented every right-minded sanitarian from sleeping soundly through the night.

But on the other side of the dispute, sewer commissioners and water-company directors slept very well indeed. They were confident that two ebb tides per day are more than sufficient to carry the city’s noxious waste into the North Sea. For them, the problem was simple. Surface filth is public enemy number one and the fundamental cause of impure water and escalating urban morbidity. The quality of London’s streams and groundwater will only be improved when effluent from offensive trades and leaking cesspools is diverted into a proper, fully integrated drainage system that empties into the Thames. The river will clean itself.

What about tidal flow, countered the sanitarians? Sewage clings to mudflats as the river recedes, and is then picked up several hours later and carried far up the Thames and its tributaries, especially the River Lea. That’s why so many Londoners will walk long distances to a street pump rather than dip their pails into a foetid cask of company water closer to hand.

Directors of the private water companies serving the metropolis claimed they had the problem of sewage-contaminated mudflats under control. Holding ponds and sand filtration capture everything untoward left behind by the tides. Pure water comes out the tap. Besides, sewage contamination of the Thames is moot since 1852, thanks to Parliament’s gross over-reaction to data
on cholera mortality presented by the GRO. The Acts of 15 & 16 Victoria (1852), which mandated relocation of remaining water works beyond the tidal reach of the Thames and its tributaries, demanded a massive investment of capital and less profit for our investors. But we follow the law, no matter how wrong-headed and in restraint of fair trade. We are making good progress and those of us who were ordered to construct new waterworks will have done so within a few years. That should be time a’plenty before a third cholera visitation reaches London; after all, the gap between the first two epidemics was sixteen years.

**London cholera epidemic of 1853**

No one guessed right. Epidemic cholera returned to London late in the summer of 1853. In the districts south of the Thames, which had suffered disproportionately during the 1849 epidemic, the Lambeth Water Company had opened new waterworks at Thames Ditton, above the Teddington Lock, in 1852 and no longer drew Thames water from works near the Hungerford Suspension Bridge. The company primarily served customers residing in the large registration district of Lambeth, from which it drew its name. Its chief
competitor, the Southwark & Vauxhall (S&V) Water Company, had a larger market share south of the Thames, serving 50% more houses than the Lambeth overall, mainly in Wandsworth. S&V directors had decided in 1852 to construct new works at Hampton and pipe raw river water to upgraded filtration reservoirs at Battersea; they estimated completion by September 1854. Until then, our customers will continue to receive potable water drawn from the Thames at the old Battersea works. That’s nothing to worry about, however. Elevation is the major determinant of cholera mortality. Mr. Farr of the GRO tells us so. Purer water will not help those unlucky enough to live in the south London floodplain.

How unlucky, precisely, wondered Farr as the 1853 epidemic entered its eighth week. Had purer Lambeth water resulted in lower predisposition to cholera in the districts it served? Had other companies made significant alterations since the 1849 epidemic that might affect cholera mortality in the metropolis? It was time to lay the statistical groundwork for an Exeter-like comparison, starting with updated information on the state of the London water supply. The GRO requested updated information from all ten private water companies in the metropolis: had anything changed in sources, filtration, and areas served since 1849?

Replies dribbled in over a period of six weeks, after which Farr’s clerks began their analysis. Only the Lambeth Company had made significant improvements in supply since 1849, providing purer water from the new water works at Thames Ditton. The impact on cholera mortality appeared to be substantial: in the aggregate of districts supplied by Lambeth and S&V, there were fewer deaths per 100,000 inhabitants than in districts supplied solely by S&V, whereas it had been the reverse during the 1849 epidemic. The GRO published supplements and tables in successive Weekly Returns in November and December of 1853 which showed how the two companies were faring in this unanticipated and unwelcome competition.

Snow found the makings of an Exeter-like natural experiment in the table at the end of the supplement on water supply attached to the 19 November 1853 return. Mortality in districts joint-

<table>
<thead>
<tr>
<th>Water Companies</th>
<th>Sources of Supply</th>
<th>Aggregate of Districts supplied chiefly by the respective Water Companies</th>
<th>Deaths from Cholera in 12 Weeks ending Nov. 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lambeth and Southwark</td>
<td>The Thames at Thames Ditton and at Battersea</td>
<td>Elevation in feet above Trinity High-water Mark</td>
<td>Population</td>
</tr>
<tr>
<td>Southwark</td>
<td>The Thames at Battersea</td>
<td>8</td>
<td>118267</td>
</tr>
</tbody>
</table>

ly supplied by Lambeth and S&V was two-thirds of those where S&V was the sole provider. He was certain the reduction was due to the fact that the Lambeth Company no longer pulled water from a heavily polluted part of the Thames, whereas S&V
still did so. Such before-and-after comparisons were crude and easily challenged because factors other than water purity were impossible to rule out. But it was something to follow up when the epidemic was over.

The return for the week ending 26 November was a game-changer. Snow’s eyes spied an entry at the bottom of the summary table on the first page of the return: “In three cases (marked with an asterisk) the same districts are supplied by two companies.” He focused on the second of the three:

<table>
<thead>
<tr>
<th>#</th>
<th>(1) Lambeth and Southwark</th>
<th>(2) Southwark</th>
<th>The Thames, at Thames Ditton and at Battersea</th>
<th>1</th>
<th>346363</th>
<th>2:11</th>
<th>61</th>
</tr>
</thead>
</table>

Snow remembered reading Joseph Quick’s testimony before the commissioners inquiring into the State of Large Towns and Populous Districts in the mid-1840s that parts of South London, unlike registration districts north of the river, were served by competing water companies. He had paid little attention to this situation since such competition was insignificant from a public health perspective as long as both companies provided water drawn from similar sources. They no longer were.

Lambeth had shifted its supply between two cholera epidemics, S&V had yet to do so. If the cholera mortality comparison could be narrowed to the intermixed districts — a relatively contained area supplied by both companies, one providing pure water, the other impure water to the same socio-economic ranges of residents — then most extraneous factors would be eliminated and the GRO’s findings on cholera mortality no longer subject to serious scientific challenges. Farr was wrong. A chance event had changed everything: Snow believed an *experimentum crucis*, a defining investigation, was possible in South London if he could establish which of the two companies supplied each household where someone had died of cholera.

But he couldn’t pull that off this late in the season. The winter solstice was less than a month away and there was already insufficient daylight, given his current schedule, to
track down the requisite information for over two hundred cholera deaths since the epidemic began fourteen weeks ago. December was a busy month for operations at London hospitals, all but one situated north of the Thames. Snow would often be administering anesthesia until noon, at least. He would not have time to walk to distant parts of south London, conduct interviews, and return before the sun had set between three and four in the afternoon. The *experimentum crucis* was only doable if the cholera epidemic resumed before Southwark & Vauxhall could shift its supply source to new works near Hampton by late summer, 1854. Not exactly something to hope for. But if it happened, he was determined to be ready.

Snow did what could be done over the winter. The Statistical Office published a list of all cholera deaths in London from the epidemic’s beginning in August 1853 until its conclusion in January 1854. No names, of course, but there was a street address where each death occurred. Snow aggregated this data by sub-districts. Thereafter Farr provided documentation of sub-district capital investment in pipes and property by S&V and Lambeth. Snow also consulted a watershed map from the 2nd Health of Towns Report (1845) that he had used in an 1851 paper read before the London Epidemiological Society, then organized sub-district cholera mortality data according to whether the water supply was provided by S&V alone, Lambeth alone, or both.

In the next meeting with Farr, it quickly became obvious that the results were bitter-sweet, at best.

Snow was quick to point out that his hypothesis was supported by the fact that registrars had recorded no deaths from cholera in three sub-districts served only by the Lambeth Company. The combined effects of purer water and no person-to-person propagation.

Maybe, maybe not, countered Farr. Look at the average elevation of these three sub-districts: from 66 to 128 feet above Trinity High Water mark. The elevation law explains why they were free of cholera. Activated cholerine carried by mist off the Thames had condensed before it reached the higher elevations.

Snow took his point, but responded that the elevation law doesn’t explain why the average cholera mortality per 100,000 residents in the twelve sub-districts served exclusively by S&V (at eight feet above THW) was nearly 110, whereas it was just 59 in sixteen intermixed sub-districts at one foot THW. The absence of London sewage in water provided by the Lambeth Company would explain what elevation cannot.

Perhaps. But so would predisposition. Impure water renders some individuals susceptible to aerial infection who would otherwise be unaffected. That’s why there will always be residual mortality due to impure water after correcting for elevation.

Predisposition is not an explanation, it’s
August 31, 1854

an admission of medical ignorance. Why fall back on that when the simple answer is there for all to see? Everyone who draws a bucket of impure water will not receive a morbid prize. The mater-\textit{ies morbi} from cholera patients draining into the Thames is diluted, but not infinitely so. Its basic cellular structure remains intact and capable of spreading the disease to the few unfortunates who inadvertently drink it.

Cells? The cholera is a living animalcule? Not necessarily. But it’s a morbid poison, so the active agent would be similar to the fluid in smallpox postules which appear to be cellular. In the absence of clear identification, it may be called a special animal poison or, in \textit{zymotic} vocabulary, an organized ferment.

Farr was immovable. Snow lacked the trump card he thought would have won him this hand: a proper house-to-house investigation of water usage in all houses in the intermixed sub-districts where someone had died from cholera. The sub-district numbers suggested a two-fer in the making. If mortality in houses supplied with water by the Lambeth Company hovered in the range of simple contagious propagation found at higher elevations in the metropolis, then the natural experiment would put to rest Farr’s elevation law for London. It would also offer incontrovertible proof of his own hypothesis that drinking sewage-adulterated water in times of cholera explains the epidemic expression of that disease. That was the sweet part. The bitter part was a realization that this chance opportunity was unlikely to re-occur in 1854; the two previous cholera epidemics in London were concentrated in a single year.

**Epidemic cholera returns to London in July 1854**

Then, once again, chance favored a prepared mind. Cholera was raging in St. Petersburg and Kronstadt, where the British Baltic Fleet was deployed. The British Admiralty forbade shore leave and any contact with Russian shipping. But there was no policy against dropping a pail on a rope into the Gulf of Finland and pulling up drinking water considerably fresher than what was served from casks stored in the ship’s hold. Cholera began appearing on the large naval ships shelling Kronstadt early in the summer of 1854; steam vessels carried the sick and the injured out of the Baltic Sea, through the Kattegat and Skager-rak, across the North Sea, and up the Thames to Greenwich. Five cases of cholera ended in death during the second week of July; all were on board ships or at riverside residencies near Greenwich.

The following week’s tally of cholera deaths confirmed that the disease had unexpectedly revisited metropolitan London for a second year in succession. Twenty-six died in the week ending 22 July — the first official week of the 1854 epidemic, followed by 133 the fourth week of July.
Wednesday 9 August.

Snow carefully studied the most recent Weekly Return. There was no tabular breakdown, just a long list of individual cases, 399 in all. He concentrated on the 290 in south London. Almost seventy-three percent of all deaths in the London metropolis had occurred south of the Thames. As in 1853, south London districts were suffering disproportionately from the get-go. This epidemic was shaping up to be an 1853 reprise. Time to accelerate preparations.

This Return for the week ending 5 August organized cholera deaths by sub-districts. It was a simple matter for Snow to tick each of the sixteen intermixed sub-districts he had identified over the winter. He did the same for each of the three previous Returns, which he had been saving, and systematically recopied the deaths in every inter-mixed subdistrict during the first four weeks of the epidemic. By his reckoning, if the inordinate mortality in the southern districts was due to cholera discharges in Battersea water, the disparity between houses served by S&V and Lambeth would be the greatest early in the epidemic. Once normal propagation set in, some residents receiving Lambeth water would contract the disease by other means, thereby partially masking the actual impact of drinking pure and choleraic-polluted water. The 134 addresses in these sixteen sub-districts would constitute the basis of his investigation if the epidemic continued.

Snow checked his diary: very few chloroform administrations scheduled after the middle of the month. Try to keep it that way. Next week could be the tipping point.

Thursday 17 August 1854.

Snow scanned the Times at breakfast. “Health of London,” a synopsis of the most recent Weekly Return for 12 August 1854, was on page 9. Deaths were climbing, 644 in the last week, 446 — a horrific 69% — in the south districts. But in the fourth week of the 1849 epidemic, 823 had died with an even higher percentage south of the Thames. Could the difference be due to purer Lambeth water drawn the river at Thames Ditton, above the Teddington Lock? Most likely.
Time to find out if a natural experiment was underway in the inter-mixed areas served by the two competing companies. If so, there should be significantly less cholera mortality amongst Lambeth customers than S&V customers receiving Battersea water drawn within the tidal reach of the Thames.

Snow looked at the *Times*’ breakdown of cholera mortality by sub-districts in every district. He ran his finger along the figures for the eleven southern districts. The district of Lambeth stood out with 71 deaths. Hmm! Quick double-check of the 5 August *Return*; there were 33 that week, so mortality had more than doubled. Thirty of the 71 deaths were in the two Kennington sub-districts. Although the watershed map from the Health of Towns Report showed that both Kennington sub-districts were only partially within the intermixed area, the capital outlay figures for the competing companies were roughly equal and the extent of their services might be as well. He’d begin the investigation there.

Snow had three chloroform administrations scheduled that morning. After breakfast, he walked west on Piccadilly to St. George’s Hospital at the intersection of Hyde Park Corner and Grosvenor Place. No complications during the removal of a breast tumor or a below-knee amputation. He left the hospital, walked through Mayfair, crossed Oxford Street near Cavendish Square, and continued to a private residence near Portland Place, just south of Regent’s Park. The patient there, however, reacted violently to chloroform and required restraint while Snow held the mask to his face, suddenly became worrisomely rigid, and then just as suddenly lapsed into insensibility. The surgeon applied a lime-potassa paste to the patient’s venereal warts as Snow turned a valve on the face-mask. The patient inhaled fresh air and gradually awoke. Snow waited at the bedside long enough to note, with relief, that the man exhibited neither sickness nor depression.

Snow walked quickly the mile and a half
August 31, 1854

to his house, deposited his anesthesia kit, and continued on to the GRO offices in Somerset House. Farr gave him access to the full list of 644 deaths in the 12 August Return. For the time being, Snow copied just the thirty addresses for the two Kennington sub-districts, put them behind the fourteen that had died in those sub-districts as listed seriatim in earlier Weekly Returns, left Somerset House, and crossed the Waterloo Bridge into South London. The shoe-leather epidemiologist was afoot.

Snow was amazed by the intermingling of the two water companies in Kennington. There weren’t just parallel pipes in most streets, as Joseph Quick, the engineer at S&V, had testified before the Commissioners who wrote the Health of Towns Reports a decade ago. The pipes of both companies ran into most alleys, courts, and mews. Now and then a house is supplied just by one company, depending on which an owner selected when rate wars were in place. They are the exception. Even so, it’s typical that a house has a different supply from its neighbors on either side. Both companies are used by rich and poor, both supply magnificent family homes and near-derelict row houses.

In just these two sub-districts alone, at least thirty thousand people are unwitting subjects in a huge natural experiment. Over the course of the present epidemic, it will become clear if the inhabitants of households supplied with impure S&V water from within the tidal reach of the Thames at Battersea are more likely to come down with cholera than their neighbors who happen to receive Lambeth water piped in from the Thames at Thames Ditton, which is largely protected from north Atlantic tides by the Teddington lock.
Thursday 24 August (resumed from pp. 7-8).

William Farr agreed with Snow that the distribution of cholera fatalities during the first six weeks of the current epidemic in metropolitan London suggested a repeat of what had happened in 1853. There was dramatically higher mortality in every district supplied, all or in part, by the Southwark & Vauxhall Company. On the other hand, there were very few deaths from cholera in the four sub-districts where the Lambeth Company is the sole provider of piped water; but that’s not surprising since all four are outliers in South London, with much higher elevations than sub-districts in the Thames floodplain.

Snow handed Farr a list of addresses where someone had died of cholera in two Kennington sub-districts during the first five weeks of the epidemic. He had visited every house in which the registrars for these two sub-districts had recorded a death from cholera during the first five weeks of the 1854 cholera epidemic. He had also made additional inquiries when a fatal attack and death had occurred at different addresses, plus ascertained whether water supply was from a pump or one of two competing companies.

Snow was excited. His preliminary results showed a disproportion of 10 to 1 amongst houses in Kennington. But if S&V pipes water to ten times more houses in Kennington than Lambeth does, then ten times the deaths in S&V supplied houses isn’t significant in terms of disease causation. Data on each company’s capital outlay in pipes throughout Kennington suggest each company serves roughly the same number of houses. The actual exposure ratios cannot be calculated until these mortality figures are divided by the precise number of houses each company supplies in the two sub-districts.

Farr looked puzzled. What do you mean by exposure ratios in this situation?

<table>
<thead>
<tr>
<th>Kennington, First Part</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Supply</td>
<td>No. of Houses</td>
</tr>
<tr>
<td>Southwark and Vauxhall</td>
<td>27</td>
</tr>
<tr>
<td>Lambeth</td>
<td>2</td>
</tr>
<tr>
<td>Pump-wells on premises</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>31</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Kennington, Second Part</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Supply</td>
<td>No. of Houses</td>
</tr>
<tr>
<td>Southwark and Vauxhall</td>
<td>11</td>
</tr>
<tr>
<td>Lambeth</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
</tr>
</tbody>
</table>

Shorthand for deaths per total units of exposure. Enumerators in the 1851 census recorded over 300,000 inhabitants in the sixteen sub-districts where these two companies have shared watersheds. During the 1849 cholera epidemic there were over 3900 deaths in these intermixed watershed sub-districts. Since the Lambeth Company now draws its water from a purer portion of the Thames,
my hypothesis is that there should be a significant decrease in overall mortality from cholera in the intermixed area. If so, we may add South London to the list of metropolitan natural experiments, such as in Exeter, where the introduction of purer water reduced cholera fatalities. But to take full advantage of the current situation, I intend to make inquiries at every house in the sixteen intermixed sub-districts where someone contracted a fatal case of cholera from the beginning of the epidemic in July until it peters out in the autumn, most likely. Eventually, we’ll know how many cholera victims in each intermixed sub-district drank S&V water, how many drank Lambeth water, and how many drank water from a pump, a ditch, or a pail dipped into the Thames. But total cholera fatalities potentially attributable to the two water companies in each intermixed sub-district must be adjusted to reflect the proportion of houses each company supplies.

Farr was silent a moment before giving Snow some unexpected news: The GRO doesn’t have such information.

Pardon?

The most recent returns made to Parliament by the two companies detail only the total number of houses supplied in South London during the calendar year, 1853. Let’s see: 40,046 by S&V, 26,107 by Lambeth. The specificity you seek wasn’t requested by the Registrar-General last October; we know that S&V’s watershed extends into twenty-eight sub-districts, Lambeth’s into twenty. But we do not know how many houses each supplies in individual districts, let alone at the sub-district level. At this juncture, it would take a special Parliamentary request to get something more detailed for 1853. Assuming Parliament agrees — and that’s no certainty in the current political climate, where water company directors and investors remain suspicious of a government that considered eliminating them altogether in the wake of the 1849 cholera epidemic — the GRO would not receive such information until well into next year.

Snow was stunned. The method he had devised to test the effect of metropolitan water supply on the communication of cholera within the intermixed watersheds of South London assumed the availability of data on the number of houses the competing companies served in each sub-district.

Farr wondered if Snow’s project could be salvaged by using population data instead of houses for the denominator? The 1851 census provides figures on total population in each registration sub-district, from which the current population of the intermixed area can be estimated.

Not really. Half of the intermixed sub-districts are still dominated by one company, some to the degree that the competitor only supplies a smidgen of houses. Population-based ratios using, for example, 10,000 inhabitants as the denomina-
tor, would yield unreliable and, perhaps, unfair results in some of the intermixed sub-districts. However, we could expand the scope of the experiment to include cholera fatalities in all sub-districts that either company serves, divided by proportionally adjusted figures on total numbers of houses. What were they again?


If the denominator becomes total number of houses served per 10,000, then the proportion is 4.0 S&V to 2.6 Lambeth. Totals for cholera fatalities in all houses supplied by the two companies must be adjusted in order to account for the difference in the extent of their respective watersheds. Let’s check our arithmetic on my preliminary results in the two Kennington sub-districts:

S&V, 27 + 11 = 38 ÷ 4.0 = 9.5
Lambeth, 2 + 2 = 4 ÷ 2.6 = 1.5.

Simplifying the comparison: if 9.5:1.5::x:1, then x = 9.5 ÷ 1.5, or 6.3.

So, if the proportion of total houses each company supplies were to exist in Kennington, then South London inhabitants receiving piped water from S&V were between six and seven times more likely to die from cholera than those living in neighboring houses connected to Lambeth Company pipes.

The preliminary figures for Kennington are for numbers of houses in which a fatal attack occurred. What if there were more than one fatality in a house, as we know there are in other sub-districts?

Pure happenstance. Total
fatalities will be the numerators, but I’ll keep track of numbers of affected houses, regardless of how many deaths occur in them, per company as well, in case that seems significant later. Although I’ll aggregate fatalities in the respective company’s entire watershed, I’ll also keep totals by sub-districts in the event Parliament requests additional information from the water companies.

Wait a tick. Farr looked at the latest Weekly Return. Nearly 1200 people have been registered as dying from cholera in South London during the first six weeks of the current epidemic, and the peak is still not in sight. One person cannot visit that many houses. I can instruct all registrars throughout metropolitan London to inquire about water supply at every house where someone dies of cholera after the 26th. We’ll tally the results and give you all specifics for South London.

Offer accepted, with gratitude.

However, the GRO cannot assist you in making inquiries on fatal attacks. My hunch is that seven-week totals for South London will exceed 1500. Cholera fatalities at addresses in Greenwich and Lewisham served by the Kent Water Company don’t affect your study; but the reduction is likely to be marginal. You’ll need to hire a co-investigator.

Snow was silent for several moments, then reluctantly agreed that Farr’s assessment was spot-on. However, Snow insisted that he should personally investigate all fatal attacks in the sixteen intermixed sub-districts so he could complete the analysis of the natural experiment as originally envisioned when the water companies provided sub-district level data on the numbers of houses they served.

If! Not when!

Point taken. Snow then suggested that he also make inquiries in the four sub-districts where Lambeth is the sole provider of piped water, since then one person would have investigated every fatal attack potentially connected to the company distributing purer water early in the epidemic, when propagation by other methods was less likely than it would become as the epidemic advanced. He would put out feelers for a medical man to undertake all inquiries in the twelve sub-districts where S&V had no competition.

Farr agreed that this plan was far better than abandoning the natural experiment in South London altogether. Analysis of the two companies’ complete watersheds would at least be a natural experiment on the Exeter/Hull models — a before/after comparison of cholera deaths subsequent to a change in the quality of water. And it has the potential to be very persuasive.

The two Waterloo Road sub-districts suddenly
moved to the top of Snow’s to-do list. Both lay fully within the intermixed area. Both were easily reached from his house via the Hungerford Suspension Bridge.

He’d return with his findings next week.

Thursday afternoon
31 August 1854

Snow put something else on the desk. Farr picked it up. Two pieces of proof paper containing a printer’s impression for an insert in the coming Saturday’s issue of the Medical Times and Gazette. Farr scanned it quickly. Yet another letter to the editor by John Snow of 18 Sackville Street.

Farr’s attention settled on a tabular section containing sub-district data. The Kennington figures he had seen previously would have to be updated; cholera mortality there had again doubled in a week. The figures for the two Waterloo Road sub-districts through 19 August were new to him. A 6.5 to 1 mortality difference between S&V and Lambeth; not as striking as the initial investigation of the Kennington sub-districts Snow had shown him last week, but normal propagation of cholera may have begun. The Waterloo Road numbers included a calculation for number of deaths per company, reflecting the altered dimensions of the natural experiment, although Snow had chosen not to mention this in his letter to the editor.

Snow moved on. Mr. John Joseph Whiting, recently qualified as Licentiate in the Apothecaries Company, has agreed to conduct inquiries in Bermondsey and the rest of the S&V exclusive sub-districts. He’ll definitely investigate the 200 deaths that occurred in there through 5 August. Thereafter he may be leaving London to take over his father’s practice in King’s Lynn. That would leave 644 un-investigated cholera deaths in those sub-districts between 5 and 26 August, after which the registrars take over inquiries. May have
to estimate death rates for those three weeks, based on what happens in other sub-districts.

What’s the current status of the investigation in the intermixed sub-districts?

Snow noted that Farr was looking at the printer’s proof of the table. Mind you, the letter to be published by *MTG* on Saturday isn’t up to date. Making good progress. Early difficulties are now sorted. Should be able to finish inquiries on the water source of deaths registered through 26 August at all addresses in the Kennington sub-districts in the coming long weekend, perhaps make some inroads in Brixton and Clapham as well. May overnight at Mr. Richardson’s in Mortlake to save travel time.

He didn’t recognize the sister accompanying the Matron. Another *locum tenens*, most likely. There’d be more of them; the silly-season lasted until the end of September, when all summer vacations would be over.

Matron made the introductions. Miss Nightingale, substituting as head ward nurse, met Alexander Stewart, M.D., Assistant-Physician to the hospital. He normally took late afternoon and evening rounds on the medical wards. Miss Nightingale would meet Dr. Hawkins, one of the
hospital Physicians, in the morning. She would do as either bid.

Most of the dozen or so beds were occupied. Standing at the sister’s station at the end of the ward, Stewart began a preliminary overview: whooping cough and scarlatina in several infants and young girls.

Nightingale nodded. She had observed childhood diseases among cottagers at her father’s summer estate in Derbyshire and at the orphan asylum run by the Kaiserswerth Institute in Rhenish Prussia.

Advanced typhus, both the Irish fever and typhoid varieties.

She had seen this as well.

Two cases of choleraic diarrhea, three with well-marked cholera.

Nightingale puzzled the distinction.

Stewart took notice and came to the rescue. Based on collective experience with Asiatic cholera since the first London epidemic in 1832, this hospital’s medical staff believes that ordinary diarrhea rarely progresses to full-blown, malignant cholera, even during the height of a major epidemic such as this one. Diarrhea is one of the body’s natural responses to temporary disequilibrium of the constitution. The offending cause could be morbid cholera matter, of course. But it’s more often due to other causes, such as eating meat that has gone off; intemperance; fear; or a host of constitutional indispositions that settle in the alimentary canal. It’s best not to over-react or over-treat. In most instances the body eliminates whatever is untoward and the natural equilibrium of the bodily economy is restored without outside intervention.

Of course, in epidemic times people assume, for good reason, at the earliest signs of diarrhea that they have come down with cholera. For diarrhea is frequently the first symptom of Asiatic cholera. They should seek competent medical advice. Hundreds of people have visited the hospital dispensary with complaints of diarrhea since the current cholera epidemic began the second week of July. Many receive emetics or cathartics designed to encourage the body’s natural eliminative processes, and successfully so in most instances. Many more are exposed to the morbid matter of cholera than predisposed to suffer its ravages in full measure.

It must be acknowledged, however, that sometimes the drugs dispensed do not operate as hoped, or the administration of them at home was improper, or patients delayed too long in seeking medical advice, or the initial disequilibrium was too extreme to be managed on an out-patient basis. In such cases where the duration and severity of the diarrhea increases rather than abates, it is quite possible that the bodily economy is being pushed into cholera. Choleraic diarrhea is the hospital’s term for an indeterminate condition that is worse than simple diarrhea but short of rice-water discharges, a definitive expression of Asiatic cholera. As a precaution-
ary measure, patients presenting symptoms of choleraic diarrhea are admitted to hospital, as is anyone with indisputable cholera, rather than treated on an out-patient basis in the dispensary. During an epidemic, everyone is treated, regardless of station or means.

In normal times, what kinds of patients are admitted to this hospital?

Respectable artisans in Marylebone, and their families, who cannot be nursed at home and come to the dispensary with a letter of recommendation from a hospital subscriber or governor may be placed on a medical ward. In addition, this is the nearest hospital for many residents of St. James, Westminster south of Oxford Street; medical wards are available to them on the same terms. Admissions to the surgical wards depend entirely on circumstance and need during the immediate post-operative period; no letters are required unless patients wish to be transferred to a medical ward at a later point. Currently, the surgical wards are nearly empty since elective surgeries are postponed until early October when the fall medical school session commences and ward-walking students are available. A few students from the summer session are still about, and they are recruited to assist in the operating theatres for emergency surgeries. They are also kept in reserve for service on the medical wards if there is a spike in cholera admissions.

Isn’t that highly unlikely? The weekly reports from the Registrar-General’s Office indicate that there is little cholera north of the Thames compared with low-lying districts to the south.

True enough. The five northern districts were little visited during the first phase of the epidemic last year and the same so far this summer. A similar pattern occurred in the two previous epidemics of 1832 and 1849. Fortuitously, the northern districts are situated at an elevation well above the high-tide mark of the Thames, so miasmatic emanations rarely reach the parishes served by Middlesex Hospital. But there are many other sources of putrefaction in this part of the metropolis than the River Thames. Stagnant water and marshes harbor miasmata, rotting flesh and excrement emit effluvial poisons, all of which disperse aerially with local variations in concentration related to shifting weather patterns, especially the wind. If inhaled, these morbid compounds disrupt blood physiology, circulate through the body, and cause fever. The precise manifestation depends on local contingent factors such as dosage, individual susceptibility, diet, habits, cleanliness, as well as each disease’s epidemic constitution.

Epidemic constitution?

Particular seasons and atmospheric conditions are conducive to specific fevers, some of which take on an epidemic form. English cholera is often active every summer, occasionally lasting into early autumn; the meteorological excitor
are still unknown. It finds synergistic partners in miasmata which, if inhaled, may result in a fever peculiar to that disease that eventually produces derangement of the stomach and bowels. Projectile vomiting or explosive purging is the earliest symptom. Signature characteristics of epidemic cholera are well established: pervasive, but outbreaks are locally limited by prevailing winds; when outbreaks do occur, they are seemingly instantaneous with the greatest number of victims in the first few days; and such outbreaks are not traceable to human contact. There is currently an epidemic of summer cholera in Marylebone and St. James, Westminster. It began the middle of July and has accelerated this month. Two to three hundred cases of diarrhea treated per week in the hospital dispensary. Usually not worrisome, but since there is concurrently Asiatic cholera in the metropolis, one must take seriously the possibility that an annual and medically unremarkable epidemic of summer cholera may take a malignant turn.

Is Asiatic cholera contagious?
Not as an ordinary contagion such as smallpox, which is spread by touch and fomites. But it may develop infectious properties within the bodies of cholera victims who inhabit confined and filthy rooms in sub-standard housing. There are many such fever nests north of the Thames, where mostly impoverished laboring families live in unsanitary, overcrowded, and poorly ventilated rooms. Few in such circumstances can afford to call for a medical man when they develop diarrhoea. Instead, they swallow an inexpensive binding medication such as arrowroot, which has the inadvertent consequence of impeding rather than assisting natural eliminative processes. Or they dose themselves with a proprietary cholera preventive, often with disastrous results. Add unwholesome diets to the mix, and it’s inevitable that, under a propitious epidemic constitution when Asiatic cholera is about, some cases of diarrhea due to harmless summer cholera will be pushed into its severer form. Earlier today, an unemployed Irish farrier died in hospital of malignant cholera. Mr. Sibley, the hospital registrar, just visited the address given on admission, a dank mews in Ogle Street a few blocks from the hospital. The farrier lived with his family in unmitigated poverty in an over-crowded house next to a cow shed. He was in the collapse stage of cholera on admission, which means that if the cholera had become infectious within his body, the unventilated room in which he lay would be suffused with virus. Family members and visitors would now be at risk from cholera that had become contagious.

But he was admitted to a ward, where infection is also possible.

Theoretically, yes; practically, no. Middlesex Hospital wards have high ceilings and many windows; the ventilation is excellent. Beds are kept far apart. Wards are kept scrupulously clean. Patients are bathed regularly, receive
a wholesome diet and fresh, pure water. The density of virus particles required for infectious communication should not occur in these wards, and experience to date bears this out. Nineteen patients with cholera, roughly equal numbers of men and women, have been admitted to Middlesex Hospital since the third week of July. Most were already in a state of collapse when first seen by a medical man in the dispensary. Yet, nine of them recovered after constant attention and treatment. Sisters and nurses are well-fed and follow hygienic principles of cleanliness. So far, no hospital staff have been infected by vapors emanating from cholera patients who may have become contagious, despite close contact. Nor have any patients with other maladies housed in the same wards.

Some dispensaries in the metropolis, however, aren’t as fortunate. There the slightly indisposed sit cheek by jowl with the very sick, often in dank waiting rooms little better than fever-nest housing. Infection is highly likely under such circumstances. A few days ago, Mr. Tilly, a surgeon at the Westminster General Dispensary, died of Asiatic cholera contracted in that institution or whilst visiting patients in St. Anne’s rookeries.

The tenements south of Soho Square?
Yes. An outbreak of summer cholera began there ten days ago. Hundreds with diarrhea besieged the charity dispensary in Gerrard Street. Although most of them present simple, treatable diarrhea, the St. James Vestry has yet to initiate house-to-house visitation by medical men to insure that outbreak does not progress to malignant cholera. Mr. Tilly undertook home visitations, on his own volition and without remuneration from the vestry, and died from Asiatic cholera. So the infectious form is most assuredly present north of the Thames. Only time will tell if the mischief peters out for lack of fresh victims or finds a new source from which to spread to others.
A visit with the senior curate merits a spot of tea. Enough remaining in the tin for a small pot. Fresh Broad Street water, fetched by Thomas after supper, boiling in the kettle on the fireplace grate. The children’s bedrolls from the floor tidily stored in a corner. Floor washed this morning. Sprigs of mugwort, beaten free of insects, re-hung above the mattress and near the tiny window opening onto the back courtyard. A second chair, borrowed from Mrs. Wall, properly positioned for easy conversation. Bedsheet-curtains drawn to hide the portion of the room taken up by rent-paying tenants, after reading Miss O’Bryan and Mr. Plumb the riot act about the consequences of an intemperate remark whilst the reverend is present.

Three children greeted him at the door, barely containing their anticipation.

Everything’s as it should be, he assured them. Preparations for the outing are well in hand. The dry, warm weather seems likely to continue through the weekend, and that’s all to the good. Unlike the cloudy afternoon today, there should be plenty of sun all Saturday. Won’t let on where we’re heading, but it’s into the countryside. A meadow for games, a wood for a picnic lunch in the shade. The wagons will assemble outside the church at the church’s Maidenhead entrance. Departure set for 9:00. Twilight comes early now, so the wagons will return to the church by suppertime. Fun for all, eh?

The three smiling young’uns — Mary Ann, Charles, and James — will be there for sure, said their mother. James should be, as well, unless some unexpected errands crop up. Elizabeth cannot be released from her duties, unfortunately. Now let the reverend have his tea, children. Pop into the Walls’; they’re expecting you. I’ll fetch you in a spell.

* * *

Henry Whitehead said good-bye to Mrs. Plynn. He popped into the Walls for a quick hello, to remind the pensioners that their grand-daughter was wel-
come to join the Sunday School excursion, and to say good-bye to the three Plynn children.

He stood for a few seconds on the bottom landing as he gathered his thoughts, opened the front door, and stepped into Hopkins Street. One more parishioner to visit. Home late, again.