Chapter Four

SEMMELWEIS AND CHILDBED FEVER: A CASE STUDY

As a simple illustration of some important aspects of scientific inquiry, let us consider Semmelweis’ work on childbed fever. Ignaz Semmelweis, a physician of Hungarian birth, did this work during the years from 1844 to 1848 at the Vienna General Hospital. As a member of the medical staff of the First Maternity Division in the hospital, Semmelweis was distressed to find that a large proportion of the women who were delivered of their babies in that division contracted a serious and often fatal illness known as puerperal fever or childbed fever. In 1844, as many as 260 out of 3,157 mothers in the First Division, or 8.2 per cent, died of the disease; for 1845, the death rate was 6.8 per cent, and for 1846, it was 11.4 per cent. These figures were all the more alarming because in the adjacent Second Maternity Division of the same hospital, which accommodated almost as many women as the First, the death toll from childbed fever was much lower: 2.3, 2.0, and 2.7 per cent for the same years.

Carl Hempel

CHILDBED FEVER

Just imagine what it must have been like to be young, poor, and pregnant in the early 1840s in Vienna, and find yourself assigned to the First Division of the “lying in” ward at the Vienna General Hospital. Your chances of dying from a terrible disease known as childbed fever, or puerperal fever (pere in Latin for “child” and parere for “to bring forth”) was between ten and twenty percent. The word on the street
was that this was true, as in the halls of government that instituted a commission to study the problem, and of course the doctors were all too aware of the severity of the disaster.

Childbed fever was recognized and formally identified by western medicine all the way back to ancient Greece. Although an obviously serious medical issue, it had only reached epidemic proportions in the last century or so. In order to appreciate the significance of Semmelweis’s discovery of the cause and treatment of childbed fever we need to imagine a time, surprisingly recent, in which the true nature of infection, the role of microorganisms, and the so-called “germ theory” of disease were completely unknown. Doctors had speculated about what was going on in these usually fatal cases, but in fact they really had no clue about the disease’s etiology. This fancy Latin term simply means “cause.” In fact there were two distinct sorts of causal ignorance about childbed fever. One had to do with the causal origins of the disease. The other was the causal progression of the disease within the victim’s body. This helps us to understand why the prevailing theories about the disease were so wildly misguided.

The two theories that focused on the second causal question both misunderstood, indeed misidentified, the massive amounts of pus in the poor infected woman’s body. One theory that went clear back to the Greeks, misdiagnosed the putrid fluid as a corrupted form of lachia, the naturally occurring fluid that accompanies normal delivery. This led to much speculation about the affects of the latter stages of pregnancy. The other, which was favored by Semmelweis’s teachers and supervisors, misidentified the pus as corrupted and misdirected milk. The reason for this physiological disaster was a complete mystery.
A very different causal account of childbed fever focused on its causal origins. Doctors were struck by the epidemic proportions of the disease, and other epidemics they were familiar with like cholera and smallpox. They attributed all this to a generic cause called miasma, or "atmospheric cosmic-telluric changes." But as to what all this really was they were again completely ignorant. Nevertheless, at least three theories about puerperal fever were on the table for doctors to investigate.

1. Lochial discharge theory
2. Lactescent fluid theory
3. Miasma

**Ignác Fülöp Semmelweis**

It is time to introduce the tragic hero to our story, Ignác Semmelweis. He was born of relatively humble origins in Hungary in the year 1818. At this time Hungary was a relatively insignificant part of the Austro-Hungarian Empire, and its capital, Budapest, took a distinct backseat to the empire's capital of Vienna. Even within his Hungarian background, Semmelweis faced another source of prejudice -- his family spoke a rather obscure regional dialect, and he carried a distinct accent for his entire life. All of this is relevant to understanding Semmelweis's academic and professional life because like many victims of ethnic prejudice, he seems to have always seen himself as an outsider within the privileged class, and suffered from something many of my students can identify with -- a fear and loathing of formal academic writing.

Nevertheless, Ignác was a gifted student. He began college as a law student at the University of Vienna, but was soon won over to the study of medicine. Splitting time
between the University of Vienna and the University of Pest, he completed his degree in 1844, and went looking for what we would now call an internship at the Vienna General Hospital. He was unsuccessful in his applications to study under two gifted younger researchers in the pathology department, and was forced to “settle” for an assistantship in the obstetrics department. At this time obstetrics was a new and rather undistinguished specialty.

So, at the age of twenty-eight, Ignác Semmelweis, began as the second in charge of the Maternity Division of the Vienna General Hospital. Medical education was very different in those days, and this young, newly minted doctor assumed major responsibilities in clinical medicine, research, and hospital administration. We will pick up the sad conclusion to Semmelweis’s biography directly, but we need to first turn our attention to the scientific problem he immediately encountered, and his systematic discovery of the solution.

THE VIENNA GENERAL HOSPITAL

For someone who is far from young, it pains me to admit how often the history of science reminds us that the truly significant scientific breakthroughs are made by younger researchers. There is nothing particularly surprising about this, of course, because younger thinkers are almost by definition less tied to the past, both in terms of prevailing knowledge, but also their own personal and professional standing.

The Vienna General Hospital was a classic mix of these generational divides. The Germanic system put great value on experience, loyalty, and political connection. The senior members of the faculty were described by one writer in 1876 in the following colorful language.
[a] generation that had been reared in an intellectual straight-jacket with dark spectacles before their eyes and cotton wool in their ears. The young people turned somersaults in the grass, and the old men, whose bodies had been hindered in their natural development by the lifelong burden of state supervision, felt their world about their ears, and believed that the end of things was at hand.¹

On the other hand, the hospital possessed some of the finest young medical researchers in the entire world. Three of them deserve brief introduction.

In 1844 Karl von Rokitansky, at the age of forty, became the director of pathological anatomy. He made huge contributions to medical knowledge, and formalized the practice of conducting autopsies by trained experts of every fatality in the hospital. Semmelweis was a true disciple of Rokitansky’s methodology, and although not a member of the pathology department, was trained by him in the proper technique of conducting autopsies of the fatalities in the maternity division.

Joseph Skoda, who among other things invented the stethoscope, was also an advocate of pathological anatomy. His sole professional interest seems to have been in the diagnosis of disease, not its treatment. He felt that medicine, at least in his time, should concern itself with the prevention of disease, through an understanding of its causes, and not worry about the treatment, since it always seemed so ineffective anyway. From Skoda, Semmelweis learned the importance of careful pathological observation and a fixation on understanding both the causal origins of disease, and its causal progression within the victim’s body.

Ferdinand von Herba was really a contemporary of Semmelweis. He, just like his young friend and colleague, was very much a disciple of the new pathological and
diagnostic techniques being taught by Rokitansky and Skoda. He took this background and used it to remarkable
advantage by almost single handedly beginning the specialty
of dermatology. Herba’s role in Semmelweis’s story is
twofold. He was friend and confidant to Ignác as he
suffered through the childbed fever epidemic, and he was
the first to finally share the discovery with the medical
community, both in a professional presentation he gave, and
a short medical journal publication.

**WHAT WAS THEN KNOWN**

I want to share with you a rather lengthy excerpt from a
marvelous book on Semmelweis, *The Doctors’ Plague*, by the
contemporary medical historian Sherwin B. Newland. You
will notice that Dr. Newland summarizes Semmelweis’s
background knowledge of magnitude and details of the
childbed fever epidemic by introducing each short paragraph
with the phrase “observation no..” It is almost as though
Newland is consciously schematizing the preliminary
evidence in terms of our ITTBE recipe, and we can mentally
substitute our convention of replacing the observation #
with the appropriate e and subscript.

Observation no. 1: The same number of deliveries
took place in the hospital’s two obstetrical divisions,
usually between 3,000 and 3,500. The only difference
between them was that deliveries in the First Division
were carried out by doctors and medical students and
those in the Second Division by midwives and students
of midwifery. In the First Division, an average of 600 to
800 mothers died each year from puerperal fever; in the
Second Division, the figure was usually about 60 deaths,
one-tenth as many.

Observation no. 2: Although childbed fever raged
violently in the First Division, there was no such
epidemic outside the hospital walls, in the city of Vienna.
The mortality of home delivery, whether by midwives or
private doctors, was low. Even when they gave birth in
Semmelweis and Childbed Fever

alleyways and streets, the so-called Gassengeburten, mothers who self-delivered rarely died.

Observation no. 3: Despite a general impression to the contrary, the decades of carefully kept statistics at the Allgemeine Krankenhaus showed that neither the incidence nor the mortality of puerperal fever was related to the weather, as epidemics often were.

Observation no. 4: Greater trauma during delivery appeared to increase the likelihood that a mother would develop puerperal fever. This was true of no other epidemic disease.

Observation no. 5: Closing the ward for a period of time would always stop the mortality. When mothers were delivered elsewhere during that time, they did not get sick.

Observation no. 6: The infant delivered of a woman who subsequently died of puerperal fever would not infrequently die of a fever similar to its mother’s. In such cases, the findings on autopsy were similar to those identified in the mother.2

DIFFERENCES IN THE DIVISIONS

Consider the confusion in the two kinds of disease etiology we have discussed. Semmelweis’s superiors endorsed simultaneously the milk and miasma accounts of childbed fever. Miasma, or “epidemic influences,” has a tough time explaining observations two, three, and four (e2, e3, and e4). It seemed obvious that the causal origin of the epidemic must lie in someway with differences between the two maternity divisions.

Most philosophers of my generation know of Semmelweis’s story because of a very influential little text book, Philosophy of Natural Science, by Carl Hempel, one of the most sophisticated proponents of the “pretty picture of science.” He interprets Semmelweis’s careful procedure as one of conducting a series of mini-experiments to rule out possible causal differences between the two divisions. As long as we remember that experiments are simply a
systematic way of gathering relevant new data, and then reassessing the explanatory virtues of the competing hypotheses, this is a very useful way of interpreting Semmelweis’s method.

One of the minor differences between the divisions was the position of the woman at birth.

A new idea was suggested to Semmelweis by the observation that in the First Division the women were delivered lying on their backs; in the Second Division, on their sides. Though he thought it unlikely, he decided "like a drowning man clutching at a straw", to test whether this difference in procedure was significant. He introduced the use of the lateral position in the First Division, but again, the mortality remained unaffected.

We are offered a new rival explanation:

\[ t_4 \text{. Delivery in the supine position causes childbed fever.} \]

The new data from Semmelweis’s mini-experiment, however, drops this hypothesis way down on the rank ordering.

\[ e_7 \text{. Changing to the lateral position for delivery in the First Division made no difference in the mortality rates.} \]

Another interesting difference had to do with the administering of the Catholic last rights, of all things.

Various psychological explanations were attempted. One of them noted that the First Division was so arranged that a priest bearing the last sacrament to a dying woman had to pass through five wards before reaching the sickroom beyond: the appearance of the priest, preceded by an attendant ringing a bell, was held to have a terrifying and debilitating effect upon the
patients in the wards and thus to make them more likely victims of childbed fever. In the Second Division, this adverse factor was absent, since the priest bad direct access to the sickroom. Semmelweis decided to test this conjecture. He persuaded the priest to come by a roundabout route and without ringing of the bell, in order to reach the sick chamber silently and unobserved. But the mortality in the First Division did not decrease.4

Again, we have a new rival explanation:

t5. The “terrifying and debilitating” effect of the deathbed priest’s appearance was causing childbed fever.

But the experimental new data makes that a very poor explanation.

e8. Changing the approach so that the priest could enter the sick room unobserved made no difference in the mortality rate.

“FORTUITOUS” NEW DATA

Not all scientific data is the product of experimental procedure; sometime its simply good luck. Semmelweis was out of the country at the time of a terrible tragedy at the Vienna General Hospital. Another of Rokitansky and Skoda’s disciples, Jakob Kolletschka, had been accidentally cut by a medical student’s scalpel as they were conducting an autopsy. He developed a massive infection and died a few days later. When Semmelweis returned soon after Kolletschka’s death, he studied the pathology report, and formed an exciting new hypothesis.

Totally shattered, I brooded over the case with intense emotion until suddenly a thought crossed my mind; at once it became clear to me that childbed fever, the fatal sickness of the newborn and the disease of Professor
Kolletschka were one and the same, because they all consist pathologically of the same anatomic changes. If, therefore, in the case of Professor Kolletschka a general sepsis [contamination of the blood] arose from the inoculation of cadaver particles, then puerperal fever must originate from the same source. Now it was only necessary to decide from where and by what means the putrid cadaver particles were introduced to the delivery cases. The fact of the matter is the transmitting source of those cadaver particles was to be found in the hands of the students and attending physicians.\(^5\)

That one little “ah ha” moment, laid several new bits of data on the table. One was an overlooked difference between the two wards.

\(e_9\). Medical students and their teachers in the First Division regularly conducted autopsies. The midwifery students and their teachers did not.

Another had to do with regular medical practice at the time.

\(e_{10}\). Doctors and students would routinely transition from autopsies to gynecological examinations and procedures, including childbirth, with only the most cursory rinsing of their hands.

Added to this, of course, was the information garnered from Kolletschka’s death.

\(e_{11}\). Details regarding the accident, subsequent progression of the disease, and ultimate death of Kolletschka.

All of this led to a completely new and original theory about the cause of childbed fever.
Childbed fever is caused by the introduction to the blood of cadaver particles.

**AN EXPERIMENT AND A TREATMENT**

Semmelweis was, above all, a good and compassionate doctor. His first responsibility was to the patients entrusted to his care. It’s not surprising, therefore, that his instincts upon forming his new hypothesis were all directed at putting this information immediately to work in the interests of his patients. The reasoning was straightforward. If childbed fever was being spread by the introduction of cadaver particles from the hands of the doctors and students, something had to be done to stop this from happening in the future. Although nothing was known of the true nature of infectious disease, Semmelweis and his colleagues knew that chloride solutions had long been used to rid objects of the noxious odor of putrid materials. Semmelweis reasoned that a chloride solution would be the ideal substance to destroy the foul-smelling cadaver particles.

In the middle of May 1847, he ordered that a bowl of *chlorine liquida*, a dilute concentration of the disinfectant, be placed at the entrance to the First Division, and he insisted that every entering medical attendant wash in it before touching a woman in labor. Small, stiff brushes were kept nearby, to be used for cleaning under fingernails.  

Although medical research was not his primary goal at this juncture, it is quite natural to interpret Semmelweis’s actions as an interesting experiment designed to test his new hypothesis. One can imagine bizarre and evil experiment that could have been used to test the cadaver particles theory. He might have randomly chosen twenty pregnant women and separated them into two groups. To ten he might have intentionally introduced cadaver particles into their blood streams; the other ten, the “control,” he would have scrupulously allowed no entry of cadaver.
particles. He would have then waited to see if the ten he predicted would contract childbed fever did, while the other ten did not. Thankfully, this was not his experimental procedure. He might, a little more sanely, have also conducted a similar experiment with laboratory animals, but again, his focus was on saving lives.

His order of chlorinated lime, though, produced some stunning new data.

\[ e_{12}. \text{Semmelweis ordered the chlorinated lime procedure in May of 1847.} \]

\[ e_{13}. \text{By 1848 the death rate in the First Division from childbed fever had fallen to 1.2 percent, just a tick less than the Second Division, at 1.3 percent.} \]

**Semmelweis's Evidence**

Let’s pause for a moment, and use inference to the best explanation to assess the quality of Semmelweis’s evidence. There is a great deal of evidence to schematize.

\[
e_1 \quad : \quad e_{13} \quad === \quad t_0
\]

There are also a number of rival explanations that had been discussed and partially tested.

\[
t_1 \quad : \quad t_5
\]
When we now add $t_0$ to this list and rank order all of them in terms of explanatory, we would all agree, I trust, that $t_0$ is by far the best explanation, and that Semmelweis’s evidence was quite overwhelming.

I can imagine some of you seeing things differently. You are sophisticated about the true nature of infectious diseases like childbed fever and know that their cause is certain kinds of bacteria. You might argue, therefore, that a better explanation would be:

$$t_6. \text{ Childbed fever is caused by the introduction to the blood of a certain strain of bacteria.}$$

Indeed, we would now say that all of the evidence, including a lot that was yet to come, strongly supports exactly such a theory. We would probably even say that we “know for certain” that childbed fever is caused by a bacterial infection. But all of this is how the evidence stands at the beginning of the twenty-first century. Bacteria were completely unknown in Semmelweis’s day, and what he called “cadaver particles” was a pretty accurate placeholder for their existence and causal role in childbed fever.

**The Tragedy of Semmelweis**

The story of Ignác Semmelweis should have ended in glory. He singlehandedly solved a terrible medical mystery, and saved countless lives. But glory was not to be his fate. He became so obsessed, first with solving the problem of childbed fever, and then with insisting that colleagues immediately adopt his new methods, that he became a little hard to live with. He actually accused skeptical colleagues of murder for not disinfecting their hands. To make matters worse, his direct superior was part of the entrenched older generation, and never accepted the theory, or the empirical methodology that led to its discovery. In 1848 when his
assistantship was up, he was denied reappointment to his position at the Vienna General Hospital. He took all of this very badly, and, despite the support of Rokitansky, Skoda, and Herba, he abruptly departed Vienna and returned to Budapest.

He still might have garnered the fame and prestige he so richly deserved had he only published his results shortly after the discovery. As we discussed earlier, he was never comfortable with his speaking abilities, and even less so with his writing. He simply did not alert the European medical community to what he had discovered. His friend, Herba, did make a couple of short reports that were eventually published, but all of this was distinctly second hand. When he finally did write a book about childbed fever, it was much later, and consisted as much in character assassination of those colleagues who disagreed with his theory, as it did of the clinical and experimental findings.

Semmelweis, like all of us, was a prisoner of his times, his personality, and his training and interests. He was pretty much ignorant of good experimental technique. Although he and a medical student did conduct one inconclusive set of experiments with rabbits, he did not pursue the systematic animal experimentation that would have strongly supported his theory. And, although microscopes had been invented and were being used by medical researchers, it apparently never occurred to him to look and see if he could observe those cadaver particles first hand. One can only imagine the course of medical history had he done so.

Semmelweis went to an early grave an embittered and disappointed man. He continued to practice in Hungary, but never attained the recognition he craved. In early middle age he began to behave erratically, and was ultimately institutionalized. He died shortly thereafter. There is common ironic story about his end. Some have suggested
that just like his friend Kolletschka, Semmelweis became infected with childbed fever, and that the behavioral changes and death were a result of the infection. Professor Nuland, whose two books on Semmelweis I have used so freely, argues persuasively that Semmelweis, in fact, developed Alzheimer’s disease, and died from beating in the mental hospital.

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1 Quoted in Nuland (2003), pp. 77-8.
3 Hempel p. ?
4 Ibid, p. ?
5 Quoted in, Nuland, op. cit., pp. 99-100.