THE ETIOLOGY, CONCEPT, AND PROPHYLAXIS OF CHILDBED FEVER

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AUTOBIOGRAPHICAL INTRODUCTION

Medicine's highest duty is saving threatened human life, and obstetrics is the branch of medicine in which this duty is most obviously fulfilled. Frequently it is necessary to deliver a child in transverse lie. Mother and child will probably die if the birth is left to nature, while the obstetrician's timely helping hand, almost painlessly and taking only a few minutes, can save both.

I was already familiar with this prerogative of obstetrics from the theoretical lectures on the specialty. I found it perfectly confirmed as I had the opportunity to learn the practical aspects of obstetrics in the large Viennese maternity hospital. But unfortunately the number of cases in which the obstetrician achieves such blessings vanishes in comparison with the number of victims to whom his help is of no avail. This dark side of obstetrics is childbed fever. Each year I saw ten or fifteen crises in which the salvation of mother and child could be achieved. I also saw many hundreds of maternity patients treated unsuccessfully for childbed fever. Not only was therapy unsuccessful, the etiology seemed deficient. The accepted etiology of childbed fever, on the basis of which I saw so many hundreds of maternity patients treated unsuccessfully, cannot contain the actual causal factor of the disease.

The large gratis Viennese maternity hospital is divided into two clinics; one is called the first, the other the second. By Imperial Decree of 10 October 1840, Court Commission for Education Decree of 17 October 1840, and Administrative Ordinance of 27 October 1840, all male students were assigned to the first clinic and all female students to the second. Before this time student obstetricians and midwives received training in equal numbers in both clinics.

The admission of maternity patients was regulated as follows: Monday afternoon at four o'clock admissions began in the first clinic and continued until Tuesday afternoon at four. Admissions then began in the second clinic and continued until Wednesday afternoon at four o'clock. At that time admissions were resumed in the first clinic until Thursday afternoon, etc. On Friday afternoon at four o'clock admissions began in the first clinic and continued through forty-eight hours until Sunday afternoon, at which time admissions began again in the second clinic. Admissions alternated between the two clinics through twenty-four hour periods, and only once a week did admissions continue in the first clinic for forty-eight hours. Thus the first clinic admitted patients four days a week, whereas the second clinic admitted for only three days. The first clinic, thereby, had fifty-two more days of admissions [each year] than the second.

From the time the first clinic began training only obstetricians until June 1847, the mortality rate in the first clinic was consistently greater than in the second clinic, where only midwives were trained. Indeed, in the year 1846, the mortality rate in the first clinic was five times as great as in the second, and through a six-year period it was, on the average, three times as great. This is shown in Table 1.

The difference in mortality between the clinics was actually larger than the table suggests, because occasionally, for reasons to be examined later,[1] during times of high mortality all ill maternity patients in the first clinic were transferred to the general hospital. When these patients died, they were included in the mortality figures for the general hospital rather than for the maternity hospital. When the transfers were undertaken, the reports show reduced mortality, since only those who could not

Source: Excerpted from Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever,* trans. K. Codell Carter. Madison, The University of Wisconsin Press, 1983.

We have omitted the bracketed page references to the original German version which appeared in the above-cited source. Ed.

[1]See below, pages 50ff.

Table 1. Annual births, deaths, and mortality rates for all patients at the two clinics of the Vienna maternity hospital from 1841 to 1846.

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1841	3036	237	7.7	2442	86	3.5
1842	3287	518	15.8	2659	202	7.5
1843	3060	274	8.9	2739	164	5.9
1844	3157	260	8.2	2956	68	2.3
1845	3492	241	6.8	3241	66	2.03
1846	4010	459	11.4	3754	105	2.7
Total	20 042	1989		17 791	691	
Avg.			9.92			3.38

be transferred because of the rapid course of their illness were included. In reality, many additional victims should be included. In the second clinic such transfers were never undertaken. Only isolated patients were transferred whose condition might endanger the other patients. . . .

. . . What is the origin, then, of the difference in mortality between the clinics? Hyperinosis [excessive fibrin in the blood], hydremia [excessive water in the blood], plethora [an excessive quantity of blood], disturbances caused by the pregnant uterus, stagnation of the circulation, inopexia [spontaneous coagulation of the blood], delivery itself, decreased weight caused by the emptying of the uterus, protracted labor, wounding of the inner surface of the uterus in delivery, imperfect contractions, faulty involutions of the uterus during maternity, scanty and discontinued secretion and excretion of lochia [a vaginal discharge during the first few weeks after delivery], the weight of secreted milk, death of the fetus, and the individuality of patients are causes to which may be ascribed much or little influence in the generation of childbed fever. But in both clinics these must be equally harmful or harmless and they cannot, therefore, explain the appalling difference in mortality between the clinics.

While I was still unable to find a cause for the increased mortality rate in the first clinic, I became aware of other inexplicable circumstances. Those whose period of dilation was extended over twenty-four hours or more almost invariably became ill either immediately during birth or within the first twenty-four or thirty-six hours after delivery. They died quickly of rapidly developing childbed fever. An equally extended period of dilation in the second clinic did not prove dangerous. Because dilation was usually extended during first deliveries, those delivering for the first time usually died. I often pointed out to my students that because these blossoming, vigorously healthy young women had extended periods of dilation, they would die quickly from puerperal fever either during delivery or immediately thereafter. My prognoses were fulfilled. I did not know why, but I often saw it happen. This circumstance was inexplicable, since it was not repeated in the second clinic. I speak here of the period of dilation, not of delivery; thus the trauma of delivery is not under consideration.

Not only these mothers but also their newborn infants, both male and female, died of childbed fever. I am not alone in speaking of puerperal fever of the newborn.[2] With the exception of the genital areas, the anatomical lesions in the corpses of such newborn infants are the same as the lesions in the corpses of women who die of puerperal fever. To recognize these findings as the consequence of puerperal fever in maternity patients but to deny that identical findings in the corpses of the newborn are the consequence of the same disease is to reject pathological anatomy.

But if the maternity patients and the newborn die from the same disease, then the etiology that accounts for the deaths of the

[2] Semmelweis was not alone, but he was in the minority. There was a discussion of infant puerperal fever in French medical literature in 1855. The discussion was reviewed in the *Monatsschrift für Geburtshülfe* 7(1856): 152f., and in the *Wiener medizinische Wochenschrift, Journal Revue,* no. 3 (1856):22f. Carl Braun also mentioned that "the unmistakable influence of puerperal fever epidemics on the mortality of fetuses has been recognized in the Viennese maternity hospital for years"; he then notes that the French refer to such cases as puerperal fever of fetuses. Carl Braun, *Lehrbuch der Geburtshülfe* (Vienna: Braumüller, 1857), pp. 589f.

Table 2. Annual births, deaths, and mortality rates for newborns at the two clinics of the Vienna maternity hospital from 1841 to 1846.

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1841	2813	177	6.2	2252	91	4.04
1842	3037	279	9.1	2414	113	4.06
1843	2828	195	6.8	2570	130	5.05
1844	2917	251	8.6	2739	100	3.06
1845	3201	260	8.1	3017	97	3.02
1846	3533	235	6.5	3398	86	2.05

mothers must also account for the deaths of the newborn. Since the difference in mortality between the maternity patients in the two clinics was reflected in the mortality rates for the newborn, the accepted etiology for childbed fever no more accounts for the deaths of the newborn than for the deaths of the maternity patients. Table 2 gives the mortality rates of the newborn at the two clinics.

Because their mothers died or were otherwise unable to nurse, many of the newborn were sent directly to the foundling home. Later we will consider their fate.[3]

The occurrence of childbed fever among the newborn can be explained in two ways. Childbed fever may be caused by factors operating on the mother during the intrauterine life of the fetus, and the mother can then impart the disease to the infant. Alternatively, the causes may affect the infant itself after birth, in which case the mother may or may not be affected. Thus the infant dies, not because the disease has been imparted, as in the first case, but rather because childbed fever originates in the infant itself. If the mother imparts childbed fever to the infant during intrauterine life, then the difference in infant mortality between the two clinics cannot be explained by the accepted etiology, because this etiology inadequately explains the origin of the disease in mothers. If the cause of childbed fever operates directly on the infant independently of the mother, then it is still impossible for the accepted etiology to explain the difference in infant mortality rates. [Given the accepted theories], one would expect the mortality in the second clinic to have been either equal to or greater than that of the first. Of course, many of the causal factors that purportedly explain childbed fever among maternity patients are simply impossible with regard to infants—infants would not, in all probability, fear the evil reputation of the first clinic, their modesty would not be offended by having been delivered in the presence of men, etc.

Childbed fever is defined as a disease characteristic of and limited to maternity patients, for whose origin the puerperal state and a specific causal moment are necessary.[4] Thus when this cause operates on a person who is predisposed by the puerperal state, childbed fever results. However, if this same cause operates on persons who are not puerperae, some disease other than puerperal fever is generated. For example, some believe that maternity patients in the first clinic, knowing of the countless deaths occurring there each year, are so frightened that they contract the disease. Thus the disposing factor is the puerperal state, and the precipitating factor is fear of death. We can assume that many soldiers engaged in murderous battle must also fear death. However, these soldiers do not contract childbed fever, because they are not puerperae and so they lack the disposing factor.

If an individual is openly examined for the instruction of males, her modesty is offended and, because she is predisposed by the puerperal state, she contracts childbed fever. But female modesty can be offended in many ways, and if the offended young woman is not in the puerperal state, she does not contract childbed fever because she is not predisposed. Something else occurs; for example, she may swoon. Chilling brings childbed fever in puerperae, but in

[3] See below, pages 57ff.

[4] Among Semmelweis's contemporaries the causal explanation of a specific instance of some disease was usually divided into predisposing and exciting factors. Different diseases were believed to result from the operation of a constant, exciting cause if the persons on whom that cause operated had been differently predisposed. In this and the following two paragraphs Semmelweis is subjecting this doctrine to ironic criticism.

other persons it causes rheumatic fever. In puerperae, mistakes in diet induce childbed fever. In others, similar mistakes cause only gastric fever.

Becoming convinced that childbed fever is not restricted to puerperae and that it can begin during birth or even in pregnancy, one may ignore the puerperal state and focus on the unique composition of the blood during pregnancy. But even if we adopt such an approach, what predisposes the newborn to puerperal disease? Surely not the puerperal condition of their genitals. Do both male and female have the blood composition uniquely characteristic of pregnancy? The occurrence of childbed fever among the newborn shows that the very conception of puerperal fever is erroneous.

Because Vienna is so large, women in labor often deliver on the street, on the glacis,[5] or in front of the gates of houses before they can reach the hospital. It is then necessary for the woman, carrying her infant in her skirts, and often in very bad weather, to walk to the maternity hospital. Such births are referred to as street births. Admission to the maternity clinic and to the foundling home is gratis, on the condition that those admitted be available for open instructional purposes, and that those fit to do so serve as wet nurses for the foundling home. Infants not born in the maternity clinic are not admitted gratis to the foundling home because their mothers have not been available for instruction. However, in order that those who had the intention of delivering in the maternity hospital but who delivered on the way would not innocently lose their privilege, street births were counted as hospital deliveries. This, however, led to the following abuse: women in somewhat better circumstances, seeking to avoid the unpleasantness of open examination without losing the benefit of having their infants accepted gratis to the foundling home, would be delivered by midwives in the city and then be taken quickly by coach to the clinic where they claimed that the birth had occurred unexpectedly while they were on their way to the clinic. If the child had not been christened and if the umbilical cord was still fresh, these cases were treated as street births, and the mother received charity exactly like those who delivered at the hospital. The number of these cases was high; frequently in a single month between the two clinics there were as many as one hundred cases.

As I have noted, women who delivered on the street contracted childbed fever at a significantly lower rate than those who delivered in the hospital. This was in spite of the less favorable conditions in which such births took place. Of course, in most of these cases delivery occurred in a bed with the assistance of a midwife. Moreover, after three hours our patients were obliged to walk to their beds by way of the glass-enclosed passageway. However, such inconvenience is certainly less dangerous than being delivered by a midwife, then immediately having to arise, walk down many flights of stairs to the waiting carriage, travel in all weather conditions and over horribly rough pavement to the maternity hospital, and there having to climb up another flight of stairs. For those who really gave birth on the street, the conditions would have been even more difficult.

To me, it appeared logical that patients who experienced street births would become ill at least as frequently as those who delivered in the clinic. I have already expressed my firm conviction that the deaths in the first clinic were not caused by epidemic influences but by endemic and as yet unknown factors, that is, factors whose harmful influences were limited to the first clinic. What protected those who delivered outside the clinic from these destructive unknown endemic influences? In the second clinic, the health of the patients who underwent street births was as good as in the first clinic, but there the difference was not so striking, since the health of the patients was generally much better.

This would be the place to exhibit a table showing that the mortality rate among those who delivered on the street was lower than among those who delivered in the first clinic. While I had access to the records of the first clinic I felt that such a table was unnecessary because no one denied these facts. Thus I neglected to complete a table. Later when I was no longer assistant, these facts were denied, as was the existence of a significant difference in mortality between the clinics. Because of Table 1,

[5] While Semmelweis was in the first clinic, Vienna was still surrounded by medieval fortifications. The glacis was a broad earthwork that sloped away from the city and that constituted part of the fortifications. Between 1857 and 1865 the city walls were demolished and were replaced by gardens, boulevards, and public buildings.

however, this difference is undeniable. In 1848 Professor [Josef] Skoda[6] proposed that the faculty of the Viennese medical school nominate a commission that, among other things, would construct such a table. The proposal was adopted by a great majority, and the commission was immediately named. However, as a result of protests by the Professor of Obstetrics, higher authorities intervened and the commission was unable to begin its activity.[7]

In addition to those who delivered on the street, those who delivered prematurely also became ill much less frequently than ordinary patients. Those who delivered prematurely were not only exposed to all the same endemic influences as patients who went full-term, they also suffered the additional harm of whatever caused the premature delivery. Under these circumstances, how could their superior health be explained? One explanation was that the earlier the birth, the less developed the puerperal condition and therefore the smaller the predisposition for the disease. Yet puerperal fever can begin during birth or even during pregnancy; indeed, even at these times it can be fatal. The better health of patients who delivered prematurely in the second clinic conformed to the general superior health of full-term patients in the clinic.

Patients often became ill sporadically. One diseased patient would be surrounded by healthy patients. But very often whole rows would become ill without a single patient in the row remaining healthy. The beds in the maternity wards were arranged along the length of the rooms and were separated by equal spaces. Depending on their location, rooms in the clinic extended either north-south or east-west. If patients in beds along the north walls became ill we were often inclined to regard chilling as a significant factor. However, on the next occasion those along the south wall would become ill. Many times those on the east and west walls would become diseased. Often the disease spread from one side to the other, so that no one location seemed better or worse. How could these events be explained, given that the same patterns did not appear in the second clinic where one encountered the disease only sporadically?

It was my firm conviction that childbed fever was not contagious and did not spread from bed to bed. Later we will consider the proof for this conviction. For now, it is sufficient to note that the disease appeared only sporadically in the second clinic. If childbed fever were contagious, from the sporadic cases whole rows would become ill as the disease spread from bed to bed.

The authorities did not remain indifferent to the disturbing difference in mortality rates between the two clinics. Commissions repeatedly investigated and conducted hearings to determine the cause of the difference, and to decide whether it was possible to save a larger number of those patients who became ill. To achieve this last goal, from time to time all the diseased patients were transferred to the general hospital. But in spite of the change in physicians, rooms, and medical procedures, etc., the patients died almost without exception. The commissions would conclude that the cause of the great mortality rate was one or another or several of the endemic factors previously discussed. Various suitable measures were adopted, but none succeeded in bringing the death rate within the limits set by the second clinic. The failure of these measures proved that the factors identified were not, in fact, the relevant causes.

Toward the end of 1846 an opinion prevailed in one commission that the disease originated from damage to the birth canal inflicted during the examinations that were part of the instructional process. However, since similar examinations were part of the instruction of midwives, the increased incidence of disease in the clinic for physicians was made intelligible by assuming that male students, particularly foreigners, were too rough in their examinations. As a result of

[6] Josef Skoda (1805–81) was head of the department for thoracic diseases, and from 1846 until 1871 he was Professor of Medicine at the University of Vienna. Skoda pioneered auscultation and repercussion as diagnostic techniques, and popularized the use of the stethoscope. He supported Semmelweis at the beginning, but seems never to have accepted Semmelweis's strategy of characterizing diseases etiologically. After Semmelweis left Vienna for Budapest in 1850, Skoda apparently never again mentioned Semmelweis or his works—not even in lectures on puerperal diseases.

[7] The Professor of Obstetrics was Johann Klein. The proposal was, in fact, adopted unanimously, which means that even Klein approved of having a commission investigate Semmelweis's findings. But when the commission was named, Klein was not included. Thus, he would not have been a member of the commission that was to investigate work done in his own clinic. This may have led him to protest to the ministry. Erna Lesky, *Ignaz Philipp Semmelweis und die Wiener medizinische Schule* (Vienna: Hermann Böhlaus, 1964), pp. 11–35.

this opinion the number of students was reduced from forty-two to twenty. Foreigners were almost entirely excluded, and examinations were reduced to a minimum. The mortality rate did decline significantly in December 1846, and in January, February, and March of 1847. But in spite of these measures, fifty-seven patients died in April and thirty-six more in May. This demonstrated to everyone that the view was groundless. To further the reader's understanding, Table 3 shows the mortality figures for 1846 and for the first five months of 1847. We will come back later to the fact that from December 1846 through March 1847 the mortality rate declined, and that it climbed back up again in April and May 1847.[8]

Recommendations based on studies of the cause of the great mortality in the first clinic all involved one inexplicable contradiction: given the concept of an epidemic, and given that the commissions did not have the power to change the atmospheric-cosmic-terrestrial conditions of Vienna, they should have concluded that no remedies were possible. But they did not draw this conclusion, even though they considered the deaths an epidemic. What does one do to shorten the duration or to prevent the recurrence of a cholera epidemic? They attributed the disease to one or more of the previously identified endemic causes. They did not, however, identify it as an endemic disease, which would have been appropriate, but rather as an epidemic. In general, the unfortunate confusion in the concepts of epidemic and endemic disease delayed discovery of the true cause of childbed fever.

In classifying puerperal disease as epidemic or endemic, one must disregard entirely the number of patients who become ill or die. The cause of the illness or death determines whether the disease is epidemic or endemic. Epidemic puerperal fever is induced by atmospheric-cosmic-terrestrial influences; the concept of an epidemic does not stipulate whether one or one hundred persons become ill. If puerperal fever is caused by endemic factors—that is, by factors whose operation is limited to a specific location—then puerperal fever is endemic, and it is immaterial whether one or one hundred individuals become ill. This follows

Table 3. Monthly births, deaths, and mortality rates for all patients at the first clinic of the Vienna maternity hospital from January 1846 to May 1847.

	Births	Deaths	Rate
1846			
Jan.	336	45	13.39
Feb.	293	53	18.08
Mar.	311	48	15.43
Apr.	253	48	18.97
May	305	41	13.44
Jun.	266	27	10.15
Jul.	252	33	13.10
Aug.	216	39	18.05
Sept.	271	39	14.39
Oct.	254	38	14.98
Nov.	297	32	10.77
Dec.	298	16	5.37
1847			
Jan.	311	10	3.21
Feb.	912	6	1.92
Mar.	305	11	3.60
Apr.	312	57	18.27
May	294	36	12.24

from the concepts of epidemic and endemic disease. In classifying the disease one way or the other, however, the commissions did not consider the purported cause but only the number of cases. Because many patients became ill and died, it was identified as an epidemic.

I was convinced that the greater mortality rate at the first clinic was due to an endemic but as yet unknown cause. That the newborn, whether female or male, also contracted childbed fever convinced me that the disease was misconceived. I was aware of many facts for which I had no explanation. Delivery with prolonged dilation almost inevitably led to death. Patients who delivered prematurely or on the street almost never became ill, and this contradicted my conviction that the deaths were due to endemic causes. The disease appeared sequentially among patients in the first clinic. Patients in the second clinic were healthier, although individuals working there were no more skillful or conscientious in their duties. The disrespect displayed by the employees toward the personnel of the first clinic made me so miserable that life seemed worthless. Everything was in question; everything seemed inexplicable; everything was doubtful. Only the large number of deaths was an unquestionable reality.

[8] See below, pages 58 and 59.

The reader can appreciate my perplexity during my first period of service when I, like a drowning person grasping at a straw, discontinued supine deliveries, which had been customary in the first clinic, in favor of deliveries from a lateral position. I did this for no other reason than that the latter were customary in the second clinic. I did not believe that the supine position was so detrimental that additional deaths could be attributed to its use. But in the second clinic deliveries were performed from a lateral position and the patients were healthier. Consequently, we also delivered from the lateral position, so that everything would be exactly as in the second clinic.

I spent the winter of 1846–47 studying English. I did this because my predecessor, Dr. Breit, resumed the position of assistant, and I wanted to spend time in the large Dublin maternity hospital. Then, at the end of February 1847, Dr. Breit was named Professor of Obstetrics at the medical school in Tübingen. I changed my travel plans and, in the company of two friends, departed for Venice on 2 March 1847. I hoped that Venetian art treasures would revive my mind and spirits, which had been so seriously affected by my experiences in the maternity hospital.

On 20 March of the same year, a few hours after returning to Vienna, I resumed, with rejuvenated vigor, the position of assistant in the first clinic. I was immediately overwhelmed by the sad news that Professor [Jakob] Kolletschka, whom I greatly admired, had died in the interim.

The case history went as follows: Kolletschka, Professor of Forensic Medicine, often conducted autopsies for legal purposes in the company of students. During one such exercise, his finger was pricked by a student with the same knife that was being used in the autopsy. I do not recall which finger was cut. Professor Kolletschka contracted lymphangitis and phlebitis [inflammation of the lymphatic vessels and of the veins respectively] in the upper extremity. Then, while I was still in Venice, he died of bilateral pleurisy, pericarditis, peritonitis, and meningitis [inflammation of the membranes of the lungs and thoracic cavity, of the fibroserous sac surrounding the heart, of the membranes of the abdomen and pelvic cavity, and of the membranes surrounding the brain, respectively]. A few days before he died, a metastasis also formed in one eye. I was still animated by the art treasures of Venice, but the news of Kolletschka's death agitated me still more. In this excited condition I could see clearly that the disease from which Kolletschka died was identical to that from which so many hundred maternity patients had also died. The maternity patients also had lymphangitis, peritonitis, pericarditis, pleurisy, and meningitis, and metastases also formed in many of them. Day and night I was haunted by the image of Kolletschka's disease and was forced to recognize, ever more decisively, that the disease from which Kolletschka died was identical to that from which so many maternity patients died.

Earlier, I pointed out that autopsies of the newborn disclosed results identical to those obtained in autopsies of patients dying from childbed fever. I concluded that the newborn died of childbed fever, or in other words, that they died from the same disease as the maternity patients. Since the identical results were found in Kolletschka's autopsy, the inference that Kolletschka died from the same disease was confirmed. The exciting cause of Professor Kolletschka's death was known; it was the wound by the autopsy knife that had been contaminated by cadaverous particles. Not the wound, but contamination of the wound by the cadaverous particles caused his death. Kolletschka was not the first to have died in this way. I was forced to admit that if his disease was identical with the disease that killed so many maternity patients, then it must have originated from the same cause that brought it on in Kolletschka. In Kolletschka, the specific causal factor was the cadaverous particles that were introduced into his vascular system. I was compelled to ask whether cadaverous particles had been introduced into the vascular systems of those patients whom I had seen die of this identical disease. I was forced to answer affirmatively.

Because of the anatomical orientation of the Viennese medical school, professors, assistants, and students have frequent opportunity to contact cadavers. Ordinary washing with soap is not sufficient to remove all adhering cadaverous particles. This is proven by the cadaverous smell that the hands retain for a longer or shorter time. In the examination of pregnant or delivering maternity patients, the hands, contaminated with cadaverous particles, are brought into con-

tact with the genitals of these individuals, creating the possibility of resorption. With resorption, the cadaverous particles are introduced into the vascular system of the patient. In this way, maternity patients contract the same disease that was found in Kolletschka.

Suppose cadaverous particles adhering to hands cause the same disease among maternity patients that cadaverous particles adhering to the knife caused in Kolletschka. Then if those particles are destroyed chemically, so that in examinations patients are touched by fingers but not by cadaverous particles, the disease must be reduced. This seemed all the more likely, since I knew that when decomposing organic material is brought into contact with living organisms it may bring on decomposition.

To destroy cadaverous matter adhering to hands I used *chlorina liquida.* This practice began in the middle of May 1847; I no longer remember the specific day. Both the students and I were required to wash before examinations. After a time I ceased to use *chlorina liquida* because of its high price, and I adopted the less expensive chlorinated lime. In May 1847, during the second half of which chlorine washings were first introduced, 36 patients died—this was 12.24 percent of 294 deliveries. In the remaining seven months of 1847, the mortality rate was below that of the patients in the second clinic (see Table 4).

In these seven months, of the 1841 maternity patients cared for, 56 died (3.04 percent). In 1846, before washing with chlorine was introduced, of 4010 patients cared for in the first clinic, 459 died (11.4 percent). In the second clinic in 1846, of 3754 patients, 105 died (2.7 percent). In 1847, when in approximately the middle of May I instituted washing with chlorine, in the first clinic of 3490 patients, 176 died (5 percent). In the second clinic of 3306 patients, 32 died (0.9 percent). In 1848, chlorine washings were employed throughout the year and of 3556 patients, 45 died (1.27 percent). In the second clinic in the year 1848, of 3219 patients 43 died (1.33 percent). The mortality rates for the individual months of 1848 are shown in Table 5.

Table 4. Monthly births, deaths, and mortality rates for all patients at the first clinic of the Vienna maternity hospital from June to December 1847.

	Births	Deaths	Rate
1847			
Jun.	268	6	2.38
Jul.	250	3	1.20
Aug.	264	5	1.89
Sept.	262	12	5.23
Oct.	278	11	3.95
Nov.	246	11	4.47
Dec.	273	8	2.93
Total	1841	56	3.04

In March and August 1848 not a single patient died. In January 1849, of 403 births 9 died (2.23 percent). In February, of 389 births 12 died (3.08 percent). March had 406 births, and there were 20 deaths (4.9 percent). On 20 March Dr. Carl Braun[9] succeeded me as assistant.

As mentioned, the commissions identified various endemic factors as causes of the greater mortality rate in the first clinic. Accordingly, various measures were instituted, but none brought the mortality rate within that of the second clinic. Thus one could infer that the

Table 5. Monthly births, deaths, and mortality rates for all patients at the second clinic of the Vienna maternity hospital from January to December 1848.

	Births	Deaths	Rate
1848			
Jan.	283	10	3.53
Feb.	291	2	0.68
Mar.	276	0	0.00
Apr.	305	2	0.65
May	313	3	0.99
Jun.	264	3	1.13
Jul.	269	1	0.37
Aug.	261	0	0.00
Sept.	312	3	0.96
Oct.	299	7	2.34
Nov.	310	9	2.90
Dec.	373	5	1.34
Total	3556	45	
Avg.			1.27

[9] Carl Braun (1822–91) was Klein's assistant from 1849 until 1853. He succeeded Klein as Professor of Obstetrics at the University of Vienna and became Rector of the University. Braun was consistently hostile to Semmelweis; he was not conscientious in using the prophylactic measures necessary to prevent childbed fever, and he did not accept Semmelweis's etiological characterization of the disease.

factors identified by the commissions were not causally responsible for the greater mortality in the first clinic. I assumed that the cause of the greater mortality rate was cadaverous particles adhering to the hands of examining obstetricians. I removed this cause by chlorine washings. Consequently, mortality in the first clinic fell below that of the second. I therefore concluded that cadaverous matter adhering to the hands of the physicians was, in reality, the cause of the increased mortality rate in the first clinic. Since the chlorine washings were instituted with such dramatic success, not even the smallest additional changes in the procedures of the first clinic were adopted to which the decline in mortality could be even partially attributed. The instruction system for midwives is so instituted that pupils and instructors have less frequent occasion to contaminate their hands with cadaverous matter than is the case in the first clinic. Thus, the unknown endemic cause of the horrible devastations in the first clinic was the cadaverous particles adhering to the hands of the examiners.

In order to destroy the cadaverous material, it was necessary that every examiner wash in chlorinated lime upon entry into the labor room. Because students in the labor room had no opportunity to contaminate their hands anew, I believed one washing was sufficient. Because of the large number who gave birth each year in the first clinic, patients were seldom alone in the labor room; as a rule several were there simultaneously. For purposes of instruction, those in labor were arranged and examined sequentially. I regarded it as sufficient that after each examination the hands were washed with soap and water only. Within the labor room, it seemed unnecessary for the hands to be washed with chlorine water between examinations. Once the hands had been cleaned of cadaverous particles, they could not become contaminated again.

In October 1847, a patient was admitted with discharging medullary carcinoma [cancer of the innermost part] of the uterus. She was assigned the bed at which the rounds were always initiated. After examining this patient, those conducting the examination washed their hands with soap only. The consequence was that of twelve patients then delivering, eleven died. The ichor from the discharging medullary carcinoma was not destroyed by soap and water. In the examinations, ichor was transferred to the remaining patients, and so childbed fever multiplied. Thus, childbed fever is caused not only by cadaverous particles adhering to hands but also by ichor from living organisms. It is necessary to clean the hands with chlorine water, not only when one has been handling cadavers but also after examinations in which the hands could become contaminated with ichor. This rule, originating from this tragic experience, was followed thereafter. Childbed fever was no longer spread by ichor carried on the hands of examiners from one patient to another.

A new tragic experience persuaded me that air could also carry decaying organic matter. In November of the same year, an individual was admitted with a discharging carious left knee. In the genital region this person was completely healthy. Thus the examiners' hands presented no danger to the other patients. But the ichorous exhalations of the carious knee completely saturated the air of her ward. In this way the other patients were exposed and nearly all the patients in that room died. The reports of the first clinic indicate that eleven patients died in November and eight more in December. These deaths were largely due to ichorous exhalations from this individual. The ichorous particles that saturated the air of the maternity ward penetrated the uteruses already lacerated in the birth process. The particles were resorbed, and childbed fever resulted. Thereafter, such individuals were isolated to prevent similar tragedies.

The maternity hospital in Vienna was opened on 16 August 1784. In the eighteenth century and in the early decades of the nineteenth century, medicine was concerned with theoretical speculation, and the anatomical foundations were neglected. Thus in 1822, of 3066 patients only 26 died (.84 percent). In 1841, after the Viennese medical school adopted an anatomical orientation, of 3036 patients 237 died (7.7 percent). In 1843 of 3060 patients 274 died (8.9 percent). In 1827, of 3294 patients 55 died (1.66 percent). In 1842 of 3287 patients 518 died (15.8 percent).[10] From 1784 until 1823, over a period of twenty-five years, less than 1 percent of the patients cared for in the maternity hospital died. This is shown in Table 6.

[10] The figures for 1841, 1842, and 1843 are for the first clinic only, see Table 1.

Table 6. Annual births, deaths, and mortality rate for all patients at the Vienna maternity hospital from 1784 to 1848.

Year	Births	Deaths	Rate	Year	Births	Deaths	Rate
1784	284	6	2.11	1817	2735	25	0.91
1785	899	13	1.44	1818	2568	56	2.18
1786	1151	5	0.43	1819	3089	154	4.98
1787	1407	5	0.35	1820	2998	75	2.50
1788	1425	5	0.35	1821	3294	55	1.66
1789	1246	7	0.56	1822	3066	26	0.84
1790	1326	10	0.75	1823	2872	214	7.45
1791	1395	8	0.57	1824	2911	144	4.94
1792	1574	14	0.89	1825	2594	229	4.82
1793	1684	44	2.61	1826	2359	192	8.12
1794	1768	7	0.39	1827	2367	51	2.15
1795	1798	38	2.11	1828	2833	101	3.56
1796	1904	22	1.16	1829	3012	140	4.64
1797	2012	5	0.24	1830	2797	111	3.97
1798	2046	5	0.24	1831	3353	222	6.62
1799	2067	20	0.96	1832	3331	105	3.15
1800	2070	41	1.98	1833	3907	205	5.25
1801	2106	17	0.80	1834	4218	355	8.41
1802	2346	9	0.38	1835	4040	227	5.61
1803	2215	16	0.72	1836	4144	331	7.98
1804	2022	8	0.39	1837	4363	375	8.59
1805	2112	9	0.40	1838	4560	179	3.92
1806	1875	13	0.73	1839	4992	248	4.96
1807	925	6	0.64	1840	5166	328	6.44
1808	855	7	0.81	1841	5454	330	6.05
1809	912	13	1.42	1842	6024	730	12.11
1810	744	6	0.80	1843	5914	457	7.72
1812	1419	9	0.63	1844	6244	336	5.38
1811	1050	20	1.90	1845	6756	313	4.63
1813	1945	21	1.08	1846	7027	567	8.06
1814	2062	66	3.20	1847	7039	210	2.98
1815	2591	19	0.73	1848	7095	91	1.28
1816	2410	12	0.49				

This table provides unchallengeable proof for my opinion that childbed fever originates with the spread of animal-organic matter. At the time when the educational system limited opportunities for spreading decaying animal-organic matter, the patients cared for in the maternity hospital were much healthier.

As the Viennese medical school adopted an anatomical orientation, the health of the maternity patients worsened. When the number of births and of students became so great that one professor could not supervise the births and give instruction, the maternity hospital was divided into two clinics. At that time the same number of male and female students were assigned to each clinic. On 10 October 1840, by imperial decree, all males were assigned to the first clinic and all female students to the second. I am not able to say in which year the maternity hospital was divided. Colleagues who taught obstetrics in the second clinic when male students were still admitted report that there was, at that time, no significant difference in mortality between the clinics. The consistently unfavorable health of patients in the first clinic dates from 1840, when all male students were assigned to the first clinic and all female students to the second. After what has been reported, it would be superfluous to explain these facts further.

Table 1 indicates the difference in mortality rates between the patients of the two clinics after the first was devoted exclusively to training obstetricians and the second to training midwives. This would be the place to provide a similar table for the years during which female and male students were divided equally between both clinics. It would show that during this time the mortality rate was not consistently larger in the first clinic. However, I do not have

Table 7. Annual births, deaths, and mortality rates for all patients at the two clinics of the Vienna maternity hospital for 1839 and 1840.

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1839	2781	151	5.4	2010	91	4.5
1840	2889	267	9.5	2073	55	2.6

access to the necessary data. The reports were prepared in triplicate in both clinics. One copy remained in the institution; one copy was sent to the governmental administration. Those who now have these reports would do a service to science if they would release them to the public.[11] I possess the reports of both clinics only for 1840, when the male and female students were separated, and for the preceding year (see Table 7). The variation in mortality for both clinics can be traced to the activities of those in the process of becoming physicians. I was obstructed in disclosing this information because at the time it was construed as a basis for personal denunciation.

Professor Skoda assigned various responsibilities to the above mentioned commission of the Viennese medical college. Among these were the construction of a table showing, as far as the data was available, the number of deliveries and of deaths month by month, and a list of the assistants and students in the sequential order in which they served and practiced in the maternity hospital. Professor [Karl] Rokitansky[12] has directed the pathological-anatomical division since 1828. From his recollections, and from autopsy reports, and with the help of other physicians and of the assistants and students who participated in the examination of corpses, it would be possible to determine whether the number of diseased patients corresponded to the activities of assistants and students in the autopsy room. As mentioned above, higher authorities prevented the commission from carrying out this assignment.

In consequence of my conviction I must affirm that only God knows the number of patients who went prematurely to their graves because of me. I have examined corpses to an extent equaled by few other obstetricians. If I say this also of another physician, my intention is only to bring to consciousness a truth that, to humanity's great misfortune, has remained unknown through so many centuries. No matter how painful and oppressive such a recognition may be, the remedy does not lie in suppression. If the misfortune is not to persist forever, then this truth must be made known to everyone concerned.

After it was realized that the additional deaths in the first clinic were explained by cadaverous and ichorous particles on the examiners' contaminated hands, various unexplained phenomena could be accounted for quite naturally. In the morning hours the professor and the students made general rounds; in the afternoons the assistant and the students made rounds. As part of their instruction, the students examined all patients who were pregnant or in labor. The assistant was also obliged, before the morning visit of the professor, to examine those in labor and to report on them to the professor. Between these visits the assistant and the students would assume responsibility for necessary examinations. When, therefore, dilation extended over a long period and the patient spent one or more days in the labor room, she was certain to be examined repeatedly by persons whose hands were contaminated with cadaverous and ichorous particles. In this way childbed fever was induced, and as I have mentioned, these individuals died almost without exception. Once the chlorine washings were adopted and the patients were examined only by persons with clean hands, patients with

[11] On page 139, German edition, . . . Semmelweis reports that he has just obtained this information and proceeds to give the table that he here omits. He refers back to this page and apologizes for not including the information where it was first needed. The figures for 1839 and 1840 were made public in Carl Haller's report on the operation of the Vienna General Hospital published in the *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien*, 5, no. 2 (1849): 535–46.

[12] Karl Rokitansky (1804–1878) was Professor of Pathological Anatomy at the University of Vienna from 1844 until 1875 and was Rector of the University in 1853. He was one of the outstanding anatomists of the century—he is said to have performed more than 30 000 autopsies. Rokitansky also supported Semmelweis against the older members of the faculty until Semmelweis left Vienna in 1850.

extended periods of dilation stopped dying, and extended labor was no more dangerous than in the second clinic.

In order to make my next point intelligible, I must partially explain how I conceive of childbed fever. For now it is sufficient to observe that foul animal-organic particles are resorbed, and that in consequence of this resorption, disintegration of the blood [*Blutentmischung*] sets in. We have already noted that those with extended periods of dilation contracted rapidly developing childbed fever either during birth or directly thereafter. In other words, the resorption of foul animal-organic particles and the resulting disintegration of the mother's blood occurred at a time when the fetal blood was in organic exchange through the placenta with the blood of the mother. In this way, blood disintegration, from which the mother was suffering, was transmitted to the child. In consequence the newborn, whether female or male, died from a disease identical to that of the mother and in numbers equal to the mothers. Childbed fever originates in the mother because foul animal-organic matter is resorbed and leads to blood disintegration. In the infant the situation is somewhat different. The fetus, as yet unborn and in the birth canal, does not resorb foul animal-organic matter when it is touched by the examiner's contaminated fingers, but only when its blood is organically mixed with the mother's blood that has already become contaminated. This explains why an infant never dies of childbed fever while the mother remains healthy; childbed fever does not arise in the newborn through direct resorption. Both become ill while the child and mother are in organic interchange through the placenta and when the blood of the mother has disintegrated through the resorption of foul animal-organic matter. The mother can become ill while the child remains healthy if the organic interchange between them is ended by the birth process before disintegration of the mother's blood has begun.

As I have said, cadaverous particles adhering to the hands were destroyed by chlorine washings. In this way, the incidence of disease among maternity patients was brought within the limits set in the second clinic. Chlorine washings had the same effect on the incidence of disease among the newborn. Healthy mothers could no longer impart childbed fever to their infants.

In 1846, without chlorine washing, of 3533 infants in the first clinic, 235 died (6 percent). In the second clinic, of 3398 infants 86 died (2.5 percent). In 1847, during the last seven months of which we washed with chlorine, of 3322 infants 167 died (5.02 percent). In the second clinic, of 3139 infants 90 died (2.8 percent). In 1848, when chlorine washings were practiced during the entire year, of 3496 infants 147 died in the first clinic (4.2 percent). In the second clinic 100 infants died, out of 3089 (3.2 percent). These infant deaths were not from childbed fever.

If a mother died before her child, or if a mother, for whatever reason, could not nurse her child, the child was taken to the foundling home. In the foundling home, many nursing infants died of childbed fever. After the introduction of chlorine washings, nursing infants in the foundling home ceased to die of childbed fever. Dr. [Alois] Bednar, then head physician of the Imperial Foundling Home in Vienna, wrote: "Sepsis of the blood of newborns has become a great rarity. For this we must thank the consequential and most noteworthy discovery of Dr. Semmelweis, emeritus assistant of the Viennese first maternity clinic. His work fortunately explained the cause and the prevention of the formerly murderous ravages of puerperal fever."[13] Where I speak of childbed fever of the newborn, Dr. Bednar correctly speaks of sepsis of the blood; he thus remains consistent with ordinary usage.

Once the cause of the increased mortality in the first clinic was identified as cadaverous particles adhering to the hands of the examiners, it was easy to explain why women who delivered in the street had a strikingly lower mortality rate than those who delivered in the clinic. This was so because once the infant was born and the placenta separated, there was generally no longer opportunity for instruction; thus there were no examinations. A bed was assigned to such patients, and they generally left it in good health. There was no reason for their genitals to be touched by contaminated hands; therefore they did not contract childbed fever. Also, women who delivered prematurely became ill less often because they were not examined ei-

[13] [Alois] Bednar, *Die Krankheiten der Neugeborenen und Säuglinge vom klinischen und pathologisch-anatomischen Standpunkte bearbeitet* (Vienna: Gerold, 1850), p. 198 [author's note].

ther. The first requirement in premature births is to delay birth if possible. Consequently, these persons were not used for open instruction, and decaying organic matter was not conveyed to their genitals.

The sequential appearance of disease was also easy to explain. Because of the large number of births in the first clinic, several individuals were often in the labor room simultaneously. These persons were examined at least twice a day—during the morning rounds of the professor, and during the afternoon rounds of the assistant. Everyone in labor was examined for instruction sequentially in the order of their beds. When, therefore, the examiners' hands were contaminated with cadaverous particles, the genitals of several individuals were simultaneously brought into contact with cadaverous particles. This meant that the germ [*Keim*] for childbed fever was planted through resorption in several individuals at once. The patients were placed back in the maternity ward in the order in which they had delivered. Thus it often happened that those who were together in the labor room delivered at about the same time and thereafter remained in the same sequential order in the maternity clinic. In the labor room they were examined in rows by persons whose hands were contaminated with cadaverous particles, the germ of the future puerperal fever, and the disease occurred among them sequentially. After chlorine washing was instituted, sequential cases of the disease ceased.

I mentioned that toward the end of 1846, because of the prevalence of childbed fever in the first clinic, yet another commission was instituted—I have no idea how many times this had already been done—in order to identify the cause of these deaths. This commission identified the cause as injury to genitals inflicted during instructional examinations. But because the same examinations were conducted for the instruction of midwives, the commission explained that male students, particularly foreigners, examined too roughly. Consequently, the number of students was reduced to a minimum. Table 3 shows how great the mortality was before this measure was adopted, how it then declined, and how, in the months of April and May, it increased again in spite of the preventive measures. I will now explain these phenomena. Before I do, however, one item must be discussed.

As an aspirant for the position of assistant in the first clinic, later as provisional assistant and then, finally, as actual assistant, it was not possible for me to study gynecology at the gynecological division of the Imperial Hospital. However, such study was highly desirable for an obstetrician. As a substitute, as soon as I had decided to devote my life to obstetrics I examined all the female corpses in the morgue of the Imperial General Hospital. From 1844 until I moved to Pest in 1850, I devoted nearly every morning before the professor's rounds in the obstetrical clinic to these studies. I very much appreciate having enjoyed the friendship of Professor Rokitansky. Through his kindness I secured permission to dissect all female corpses, including those not already set aside for autopsy, in order to correlate the results of my examinations with autopsies.

For reasons that do not concern us here, the assistant of the first clinic seldom visited the morgue in the months of December 1846 and January, February, and March 1847. The Austrian students, whose number was reduced to eighteen, followed his example. The opportunity for them to contaminate their hands with cadaverous particles was thereby greatly reduced. Restricting examinations to the minimum also reduced the opportunity for the genitals of patients to be touched by contaminated hands. For these reasons, mortality in the first clinic was reduced during these months.

On 20 March 1847, I reassumed the position of assistant in the first clinic. Early that morning I conducted my gynecological studies in the morgue. I then went to the labor room and began to examine all the patients, as my predecessors and I were obliged to do, so that I could report on each patient during the professor's morning rounds. My hands, contaminated by cadaverous particles, were thereby brought into contact with the genitals of so many women in labor that in April, from 312 deliveries, there were 57 deaths (18.26 percent). In May, from 294 deliveries there were 36 deaths (12.24 percent). In the middle of May, without noting the exact day, I instituted chlorine washings. Thus, the great mortality in the first clinic was not caused by injuries in rough examinations—a completely false assumption—but by contaminated fingers that contacted the genitals of the patients. During April and May, when again so many died, the clinic remained the same as in earlier months, yet the mortality rate increased

significantly because I intervened, my fingers contaminated with cadaverous particles.

After chlorine washings were conducted for a longer period with such beneficial results, the number of students was again increased to forty-two. One no longer took account of whether they were Austrian or foreign. The examinations were resumed as was expedient for instruction. Nevertheless, the first clinic lost the dismal distinction of having the greater mortality rate. In December 1846 and in January, February, and March of 1847, I functioned as provisional assistant and simultaneously conducted gynecological studies in the morgue, yet in these months the mortality rate remained low. The reason is that as provisional assistant I had the right, but not the duty, to examine all patients in labor. After three years in so large a maternity hospital, it was no longer instructive for me to examine all the patients. I examined only exceptional cases—that is, I examined very seldom. When I became the actual assistant, it was my duty to conduct all examinations before the professor's morning rounds. Thereafter, it was necessary for me to examine nearly all the women in labor for the purpose of instructing the students. This occasioned the great mortality rates in April and May of 1847.

Native students are those who completed their education at an Austrian university [*Hochschule*]. Foreign students are those who were educated elsewhere and who then did further work at the great University of Vienna. In Vienna one can meet physicians from all the countries of the civilized world. The course in practical obstetrics lasted two months. The influx of students into this, the largest maternity hospital in the world, was so great that to accept simultaneously all who sought admission would have excessively disrupted the patients. Applicants were assigned numbers, and were accepted sequentially to replace departing students, regardless of whether they were native or foreign. Each student was free to repeat the course as often as he felt it necessary for his own obstetrical training. However, in order that those who wished to repeat the course would not remain constantly enrolled, precluding others from taking it at all, it was necessary that one wait three months after completing the course before enrolling again. The commission charged the foreigners with being more dangerous than the natives because they were rough in examinations and, consequently, at any one time only two foreigners were allowed to attend the course in practical obstetrics. Everyone, even those who do not share my opinion, will agree that the commission acted groundlessly in imputing guilt to the foreigners. In fact, I alone held that foreigners *were* more dangerous than natives, but not because they examined more roughly. The reasons that foreigners were more dangerous than natives lies in the following considerations.

Foreigners come to Vienna to perfect medical training already begun in their own universities. They visit pathological and forensic autopsies in the general hospital. They take courses in pathological anatomy, in surgery, obstetrics, microscopic surgery of cadavers, they visit the medical and surgical wards of the hospital, etc. In a word, they utilize their time as efficiently and educationally as possible. They have, therefore, many opportunities for their hands to become contaminated with foul animal-organic matter. Thus, it is no wonder that foreigners, busy in the maternity hospital at the same time, are more dangerous for patients. Natives take the course in practical obstetrics after completing two difficult examinations in order to attain the degree of Doctor of Medicine. The law stipulates that the minimum preparation time for these examinations is six months. Thus the natives have already toiled excessively before they are admitted into the maternity hospital, and they regard the time there as a rest. While enrolled in practical obstetrics, natives do not concern themselves with other activities that would contaminate their hands. Indeed, while working at the maternity hospital, they concern themselves even less with other aspects of medicine because, after completing the course, they can perfect their knowledge of medicine to the highest possible degree. Since the foreigners are generally able to remain in Vienna only a few months, they are compelled to work simultaneously in more than one aspect of medicine. Even so, one cannot impute guilt to the foreigners any more than to me or to all the others who undertook examinations with contaminated hands. None of us knew that we were causing the numerous deaths.