The Etiology, Concept, and Prophylaxis of Childbed Fever

by

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Preface

After twice completing the course in practical obstetrics at the first maternity clinic in Vienna, I presented myself on 1 July 1844 to the director of the clinic, the late Professor Dr. [Johann] Klein,[1] as a candidate for a future post of assistant physician at the clinic. I was provisionally appointed to this post by a decree dated 27 February 1846. On 1 July 1846 I officially assumed the position of assistant at the first maternity clinic. On 20 October of the same year, however, I was obliged to withdraw in favor of my predecessor, Dr. [Franz] Breit, because Dr. Breit had in the meantime received a two-year extension of his service. That I may be understood, in the course of this essay I will refer to these four months, namely July, August, September, and October of 1846, as my first period of service. *[iv]* In Vienna, the period of service of the assistants in all departments was fixed at two years. In every other department it was customary, after the expiration of two years, for the appointment to be extended for an additional two years. In the obstetrical department this had not been the practice, however, and the assistants were regularly changed every two years. Dr. Breit was the first to enjoy this privilege [of a two-year extension]. Then Dr. Breit was named Professor of Obstetrics at the medical school at Tübingen, and for the second time I officially assumed the position of assistant, on 20 March 1847. I functioned as such for two years, until 20 March 1849. I refer to these two years as my second period of service.

The object of this essay is this: to present historically to the medical instructor observations that I made at this clinic in this period, to demonstrate how I began to doubt existing teachings concerning the origin and the concept of childbed fever, and how

1. Johann Klein (1788–1856) was Professor of Obstetrics at the University of Salzburg and at the University of Vienna. As Semmelweis's chief in the maternity clinic, he seems to have felt threatened by the younger members of the faculty, and he opposed both Semmelweis and his theories.

I was irresistibly forced to my present conviction, in order that he also, for the welfare of mankind, may derive the same convictions.

[v] By nature I am averse to all polemics. This is proven by my having left numerous attacks unanswered. I believed that I could leave it to time to break a path for the truth. However, for thirteen years my expectations have not been fulfilled to the degree that is essential for the well-being of mankind. An additional misfortune was that in the school years of 1856–57 and 1857–58, maternity patients died in such numbers at my own obstetrical clinic in Pest that my opponents could use these deaths as evidence against me. It must be shown that these two unfortunate years provide tragic and unintentional yet direct confirmation of my views.

To my aversion to all polemics must be added my innate aversion to every form of writing. Fate has chosen me as the representative of those truths that are laid down in this essay. It is my inescapable obligation to support them. I have given up the hope that the importance and the truth of the matter would make all controversy unnecessary. Rather than my inclinations, people's lives must be considered, people who do not even participate in the conflict over whether my opponents or I have the truth. *[vi]* Since silence has proven futile, I must forcibly restrain my inclinations and step once more before the public as though uncautioned by the many bitter hours I have endured. I have made the best of these hours; for those yet to come I find consolation in knowing that I advocate only that which is firmly grounded in my own convictions.

Pest, 30 August 1860

Chapter 1

Autobiographical Introduction

[1] 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. *[2]* 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.

[3] TABLE 1

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1841	3,036	237	7.7	2,442	86	3.5
1842	3,287	518	15.8	2,659	202	7.5
1843	3,060	274	8.9	2,739	164	5.9
1844	3,157	260	8.2	2,956	68	2.3
1845	3,492	241	6.8	3,241	66	2.03
1846	4,010	459	11.4	3,754	105	2.7
Total	20,042	1,989		17,791	691	
Avg.			9.92			3.38

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.

[3] 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 be transferred because of the rapid course of their illness were included. In reality, many additional victims should be included. *[4]* In the second clinic such transfers were never undertaken. Only isolated patients were transferred whose condition might endanger the other patients.

The additional mortality in the first clinic consisted of many hundreds of maternity patients, some of whom I saw die from puerperal processes, but for whose deaths I could find no explanation in the existing etiology. To convince the reader that this additional mortality cannot be explained by the existing etiology, we must examine more carefully the previously acknowledged causes of childbed fever that have been used in attempting to explain this additional mortality.

It has not been questioned and has been expressed thousands of times that the horrible ravages of childbed fever are caused by epidemic influences. By epidemic influences one understands atmospheric-cosmic-terrestrial changes,[2] as yet not precisely defined, that often extend over whole countrysides, and by which childbed fever is generated in persons predisposed by the puerperal state. But if the atmospheric-cosmic-terrestrial conditions of Vienna cause puerperal fever in predisposed persons, how is it that for many years these conditions have affected persons in the first clinic while sparing similarly predisposed persons in the second? *[5]* To me there appears no doubt that if the ravages of childbed fever in the first clinic are caused by epidemic influ-

1. See below, pp. 73, 83f.; German edition, pp. 35, 48.

2. Physicians actually used the phrase "atmospheric-cosmic-terrestrial [or telluric] influences." See, for example, p. 343 of Eduard Caspar Jakob von Siebold, "Betrachtungen über das Kindbettfieber," *Monatsschrift für Geburtskunde und Frauenkrankheiten* 17(1861), 335–57, 401–17, and 18, 19–39; and pp. 255–58 of Anselm Martin, "Zur Erforschung der Ursachen des epidemischen Puerperalfiebers," ibid. 10(1857), 253–73.

ences, the same conditions must operate with minimal variation in the second clinic. Otherwise, one is forced to the unreasonable assumption that lethal epidemic influences undergo twenty-four-hour remissions and exacerbations and that the remissions, through a series of years, have exactly coincided with admissions to the second clinic, while the exacerbations begin precisely at the time of admission to the first clinic.

However, even with such unreasonable assumptions, epidemic influences cannot explain the differences in mortality. The exacerbated influences must affect individuals either before they are admitted to the maternity hospital or during their stay. If they operate outside the hospital, certainly those who are admitted to the first clinic will be no more subject to them than those admitted to the second. No significant difference in mortality between the equally exposed patients admitted to the two clinics would then exist. On the other hand, if epidemic influences operate on individuals during their stay in the hospital, there could be no difference in the mortality rate, since two clinics so near one another that they share a common anteroom must necessarily be subject to the same atmospheric-cosmic-terrestrial influences. *[6]* These considerations alone forced me to the unshakable conviction that epidemic influences were not responsible for the horrible devastations of the maternity patients in the first clinic.

Once I had come to this conviction, other supporting considerations occurred to me. If the atmospheric influences of Vienna occasion an epidemic in the maternity hospital, then necessarily there must be an epidemic among maternity patients throughout Vienna because the entire population is subject to the same influences. But in fact, while the puerperal disease rages most furiously in the maternity hospital, it is only infrequently observed either in Vienna at large or in the surrounding countryside. During a cholera epidemic, people in general are affected, not just those in a particular hospital. A common and successful expedient for halting an epidemic of childbed fever is to close the maternity hospitals. Hospitals are closed not to force maternity patients to die somewhere else, but because of the belief that if patients deliver in the hospital they are subject to epidemic influences, whereas if they deliver elsewhere they will remain healthy. However, this proves one is not dealing with a disease dependent on atmospheric influences, because these influences would extend beyond the hospital into every part of the city. *[7]* This proves that the disease is endemic—a disease due to causes limited by the boundaries of the hospital. What would defenders of the epidemic conception say if someone proposed to control cholera by closing cholera hospitals? Puerperal fever that originates traumatically, for example, in a forced delivery by forceps, is entirely the same in its course and in its anatomical manifestations as the so-called epidemic form. Can any other epidemic disease be generated by trauma? Epidemics exhibit intermissions perhaps years in duration; childbed fever has ravaged the first clinic for years without even minimal intermissions. Do cholera epidemics occur annually? If so-called childbed fever epidemics were really due to atmospheric influences, then they could not occur in opposing seasons and climates. In actual fact, however, the disease is observed in all seasons, in the most different climates, and under all weather conditions.

To prove numerically that seasons have no influence on the incidence of childbed fever, I will utilize the time interval represented in Table 1, together with the first five months of 1847. This will prove that every month of the year can be either favorable or unfavorable for the health of patients in the first clinic. I omit only December 1841, since I have lost the figures for this month. *[8]* However, this month may belong to those in which many patients died, since it is between other months in which patients were unhealthy. In November 1841, 53 of 235 patients died (22.55 percent), and in January 1842, 64 of 307 died (20.84 percent). This is shown in Table 2.

[9–10] The reader will observe that epidemic influences are so powerful that their pernicious activity cannot be restricted to a particular season. They rage with equal violence in the bitterest cold of winter and in the oppressive heat of summer, but they do not scourge all maternity hospitals equally; they spare some while raging all the more furiously in others. Indeed, they are so partial that they afflict differently the different divisions of the same institution.

[9] TABLE 2

	Lowest Mortality				Highest Mortality			
	Year	Births	Deaths	Rate	Year	Births	Deaths	Rate
Jan.	1847	311	10	3.21	1842	307	64	20.84
Feb.	1847	312	6	1.92	1846	293	53	18.08
Mar.	1847	305	11	3.60	1846	311	48	15.43
Apr.	1841	255	4	1.57	1846	253	48	18.97
May	1841	255	2	0.74	1846	305	41	13.44
Jun.	1844	224	6	2.67	1846	266	27	10.15
Jul.	1843	191	1	0.52	1842	231	48	20.79
Aug.	1841	222	3	1.35	1842	216	55	25.46
Sept.	1844	245	3	1.22	1842	223	41	18.38
Oct.	1844	248	8	3.22	1842	242	71	29.33
Nov.	1843	252	18	7.14	1841	235	53	22.55
Dec.	1846	298	16	5.37	1842	239	75	31.38

With a few exceptions, maternity hospitals that are not teaching institutions or that train only midwives are more favorable than institutions that train obstetricians. Table 1 shows the different mortality rates of two divisions of one institution; a similar difference occurred in the two divisions of the maternity hospital at Strasbourg. Later we will speak more of these circumstances.[3]

As explained before, these considerations strengthened my conviction that the great mortality of the first clinic was not due to epidemic influences but rather to harmful endemic factors (i.e., to a cause manifested so horribly only within the first clinic). However, when we examine the previously acknowledged endemic causes in reference to the comparative mortality rates of the two clinics, we see either that no difference in mortality could exist or that the second clinic must have the larger rate. *[11]* If overcrowding were the cause of death, mortality in the second clinic would have been larger, because the second clinic was more crowded than the first. Because of the bad reputation of the first clinic, everyone sought admission to the second clinic. For this reason, the second clinic was often unable to resume admissions at the specified time because it was impossible to accommodate new arrivals. Or if the second clinic began to admit, within a few hours it was necessary to resume admitting patients to the first clinic because the passageway was crowded with such a great number of persons awaiting admission to the second clinic. In a short time all the free places were taken. In the five years I was associated with the first clinic, not once did overcrowding make it necessary to reopen admission to the second clinic. This was true even though once each week the first clinic admitted continuously for a period of forty-eight hours. In spite of this overcrowding, the mortality rate in the second clinic was strikingly smaller.

Each year, the first clinic recorded hundreds more births than the second. This, however, was because each week it had one more day of admissions and had, therefore, a larger assigned area. In spite of its smaller number of births with respect to its

3. See below, pp. 126–28; German edition, pp. 131–35.

capacity, the second clinic was more crowded. *[12]* This is verified by the fact that the second clinic was often unable to resume admissions or had to discontinue admissions early, which never happened at the first clinic. Had the second clinic been large enough to admit all who sought admission, it would have had significantly more births each year than the first clinic, even though it had fifty-two fewer days of admissions. If we disregard the comparative overcrowding within the two clinics, and consider only the degree of overcrowding within the first clinic as determined by the number of patients treated within a given month, it is apparent than the favorable or unfavorable health of the patients was not due to the degree of overcrowding. Again I use the time interval of Table 1, together with the first five months of 1847. Mortality in these months is shown in Table 3. [Semmelweis gives eighteen pages of further tables presenting this information in different arrangements. He then continues:]

[13–32] One may believe that a location in which so many thousands of individuals have given birth, contracted childbed fever, and died must inevitably be so infested that the presence of childbed fever is no surprise. If this were the case, however, the mortality in the second clinic would be greater, since in the location of the second clinic, even in [Rogers Lucas Johann] Boër's times,[4] serious epidemics of puerperal fever raged. At that time, the building now occupied by the first clinic was not even built.

It has been proposed that the evil reputation of the institution, with its great annual contingent of deaths, so frightens the newly admitted patients that they become ill and die. The patients really do fear the first clinic. Frequently one must witness moving scenes in which patients, kneeling and wringing their hands, beg to be released in order to seek admission to the second clinic. Such persons have usually been admitted because they are ignorant of the reputation of the first clinic, but they soon become suspicious because of the large number of doctors present. *[33]* One

4. Rogers Lucas Johann Boër (1751–1835) was the first professor of obstetrics at the Viennese maternity hospital; he served from 1789 until 1822. He was an outstanding advocate of the conservative trend in obstetrics. He strongly discouraged use of forceps and other instruments and advocated the practice of natural parturition.

sees maternity patients with abnormally high pulse rates, bloated stomachs, and dry tongues (in other words, very ill with puerperal fever), still insisting only hours before death that they are perfectly healthy, because they know that treatment by the physicians is the forerunner of death. Nevertheless, I could not convince myself that fear was the cause of the high mortality rate in the first clinic. As a physician, I could not understand how fear, a psychological condition, could bring about such physical changes as occur in childbed fever. Moreover, it would have required a long period of time with consistently unequal mortality rates for ordinary people, who did not have access to hospital reports, to become aware that one clinic had a much greater mortality rate than the other. Fear could not account for the initial difference.

Even religious practices did not escape attention. The hospital chapel was so located that when the priest was summoned to administer last rites in the second clinic, he could go directly to the room set aside for ill patients. On the other hand, when he was summoned to the first clinic he had to pass through five other rooms because the room containing ill patients was sixth in line from the chapel. According to accepted Catholic practice, when visiting the sick to administer last rites, the priest generally arrived in ornate vestments and was preceded by a sacristan who rang a bell. This was supposed to occur only once in twenty-four hours. Yet twenty-four hours is a long time for someone suffering from childbed fever. Many who appeared tolerably healthy at the time of the priest's visit, and who therefore did not require last rites, were so ill a few hours later that the priest had to be summoned again. *[34]* One can imagine the impression that was created on the other patients when the priest came several times a day, each time accompanied by the clearly audible bell. Even to me it was very demoralizing to hear the bell hurry past my door. I groaned within for the victim who had fallen to an unknown cause. The bell was a painful admonition to seek this unknown cause with all my powers. It had been proposed that even this difference in the two clinics explained the different mortality rates. During my first period of service, I appealed to the compassion of the servant of God and arranged for him to come by a less direct route, without bells, and without passing

[13] TABLE 3

	1841			1842			1843		
	Births	Deaths	Rate	Births	Deaths	Rate	Births	Deaths	Rate
Jan.	254	37	14.56	307	64	20.84	272	52	19.11
Feb.	239	18	7.53	311	38	12.21	263	42	15.96
Mar.	277	12	4.33	264	27	10.23	266	33	12.40
Apr.	255	4	1.57	242	26	10.74	285	34	11.93
May	255	2	0.78	310	10	3.22	246	15	6.10
Jun.	200	10	5.00	273	18	6.60	196	8	4.08
Jul.	190	16	8.42	231	48	20.79	191	1	0.52
Aug.	222	3	1.35	216	55	25.46	193	3	1.55
Sept.	213	4	1.87	223	41	18.38	221	5	2.26
Oct.	236	26	11.00	242	71	29.33	250	44	17.60
Nov.	235	53	22.55	209	48	22.96	252	18	7.14
Dec.				239	75	31.38	236	19	8.05

	1844			1845			1846		
	Births	Deaths	Rate	Births	Deaths	Rate	Births	Deaths	Rate
Jan.	244	37	15.16	303	23	7.59	336	45	13.39
Feb.	257	29	11.28	274	13	5.11	293	53	18.08
Mar.	276	47	17.03	292	13	4.45	311	48	15.43
Apr.	208	36	17.80	260	11	4.23	253	48	18.97
May	240	14	5.83	296	13	4.39	305	41	13.44
Jun.	224	6	2.67	280	20	7.14	266	27	10.15
Jul.	206	9	4.37	245	15	6.12	252	33	13.10
Aug.	269	17	6.32	251	9	3.58	216	39	18.05
Sept.	245	3	1.22	237	25	10.55	271	39	14.39
Oct.	248	8	3.22	283	42	14.84	254	38	14.98
Nov.	245	27	11.00	265	29	10.94	297	32	10.77
Dec.	256	27	10.55	267	28	10.48	298	16	5.37

1847		
Births	Deaths	Rate
311	10	3.21
312	6	1.92
305	11	3.60
312	57	18.27
294	36	12.24
268	6	2.38
250	3	1.20
264	5	1.89
262	12	5.23
278	11	3.95
246	11	4.47
273	8	2.93

through the other clinic rooms. Thus, no one outside the room containing the ill patients knew of the priest's presence. The two clinics were made identical in this respect as well, but the mortality rate was unaffected.

The high mortality was also attributed to the clinic's practice of admitting only single women in desperate circumstances. These women had been obliged throughout their pregnancies to support themselves by working hard. They were miserable and in great need, often malnourished, and may have attempted to induce miscarriages. But if these conditions constituted the cause, the mortality rate in the second clinic should have been the same, since the same type of women were admitted there.

It had also been proposed that the high mortality rate in the first clinic resulted from the obstetricians examining the patients in a rougher manner than did the student midwives. *[35]* If inserting the finger, however roughly, into the vagina and to the adjacent parts of the uterus—already widened and extended by pregnancy—was sufficient to cause damages leading to so horrible a condition, then surely the passage of the baby's body through the birth canal must cause damage so much worse that every birth would end in the death of the mother.

It had also been suggested that the mortality rate in the first clinic resulted from the offense to modesty incurred through the presence of males at delivery. As those familiar with the Viennese maternity hospital realize, patients are troubled by fear but not by offended modesty. Moreover, it is not clear how this offended modesty would bring about the exudative mortal processes of the disease.

Since they were the same in both clinics, medical procedures were not responsible for the increased death rate. Also, as part of an experiment, all diseased maternity patients were occasionally transferred to the general hospital. They succumbed there after very different treatments. It was not that equal numbers became ill and fewer recovered in the first clinic than in the second. Rather, more patients became ill in the first clinic. The recovery rate in both clinics was the same. *[36]* However, obstetrical practices, numerous and rough procedures, etc., did not cause a great number of illnesses in the first clinic, because the

majority of those who became ill had not been subjected to special obstetrical procedures. In both divisions, patients were treated according to Boër's principles.[5]

In the first clinic it was the custom that three hours after giving birth, patients would get up from the delivery bed and walk through a passageway to their own beds. The passageway was enclosed in glass and was heated in the winter. This was a considerable distance for those whose beds were furthest from the delivery room. Nevertheless, only those who were weak or ill or had had special operations were carried.[6] But this inconvenience was not responsible for the greater mortality rate, since the second clinic was subject to the same drawback. Indeed, circumstances were worse there, since the second clinic was divided by a common unheated anteroom and patients whose beds were in rooms beyond the anteroom had to walk through that as well.

In the first clinic, a large maternity room on the third floor was reached through a glass-enclosed stairway. Since one could not expect newly delivered patients to go there on foot, it was necessary for healthy patients to be transferred there seven or eight days after delivery. At that time they would normally have been allowed to leave their beds in any case. This second move did not cause the greater mortality rate, since after seven or eight

5. "In 1723 forceps had come into general use. Delighted with the beneficial invention, French and German obstetricians (Osiander) applied them on every occasion, even when there was no need for them. One might have thought that 'Nature had abandoned its delivery job and left it to the obstetrician's instrument' (Boër). Against this busy use of instruments Boër employed his patient procedure of waiting and relying on the natural forces of the organism. In limiting instrumental and manual intervention to the cases in which such assistance was indicated and in 'restoring the delivery power of Nature to its rights,' he became the founder of the new obstetrical natural method." Erna Lesky, *The Vienna Medical School of the Nineteenth Century* (Baltimore: Johns Hopkins University Press, 1976), p. 52.

6. Lajos Markusovszky, Semmelweis's close friend and associate, recorded that Semmelweis had newly delivered patients carried back to their beds so that they would not be obliged to walk. György Gortvay and Imre Zoltán, *Semmelweis: His Life and Work* (Budapest: Akadémiai Kiadó, 1968), p. 48.

days patients seldom became ill. Moreover, the second clinic followed exactly the same procedure.

[37] Poor ventilation in the first clinic, which even in winter was for the most part accomplished by opening windows, was also proposed as an explanation for the more numerous deaths in the first clinic. Those making this proposal overlooked the fact that the second clinic was ventilated in the same way. The laundry process was blamed because the contractor mixed the laundry with that of the general hospital. Yet laundry from the second clinic was mixed in just the same way. The disadvantage of being in close contact with so large a hospital as the Viennese Imperial General Hospital was also shared by both clinics. Indeed, the clinics were so close that they shared a common anteroom and were constructed in a similar fashion. Other disadvantages, such as continuous use for instructional purposes, free passage between wards for sick and well patients, unrestricted contact between attendants for the sick and those for the healthy, were also shared by both clinics. Neither catching a cold nor errors in diet could explain the differences in mortality between the two clinics. The possibility of contracting colds was the same in both, and the food in both clinics conformed to the same dietary standards and was prepared by the same contractor.

These are the endemic causes to which was attributed the greater mortality among maternity patients in the first clinic. *[38]* With the exception of one circumstance which will be discussed later, I fully agree that these do not adequately explain the greater mortality in the first clinic. We have shown that these harmful endemic factors were either equally operative in both clinics, in which case mortality would have been the same, or they were more pronounced in the second clinic than in the first, in which case the second clinic should have had more deaths. However, precisely the opposite has regularly occurred. Since the first clinic has been used exclusively for training obstetricians, its mortality rate has consistently been significantly greater than that of the second clinic.

Since neither epidemic influences nor the previously acknowledged endemic factors can explain the greater mortality rate of

the first clinic, we must consider other factors that have been proposed as causes of childbed fever.

Recent investigators blame the disease on the most remote of all possible causes—conception itself. Supposedly, the penetration of sperm occasions a manifold series of alterations, including partially unknown changes in the blood. But I suspect that I am not misinformed in claiming that those who delivered in the second clinic must also have conceived. What is the origin, then, of the difference in mortality between the clinics? *[39]* 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. *[40]* 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.[7] 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 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 4 gives the mortality rates of the newborn at the two clinics.

[41] 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.[8]

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,

7. 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.

8. See below, pp. 99f.; German edition, pp. 67–69.

[41] TABLE 4

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1841	2,813	177	6.2	2,252	91	4.04
1842	3,037	279	9.1	2,414	113	4.06
1843	2,828	195	6.8	2,570	130	5.05
1844	2,917	251	8.6	2,739	100	3.06
1845	3,201	260	8.1	3,017	97	3.02
1846	3,533	235	6.5	3,398	86	2.05

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. *[42]* 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.[9] 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. How-

9. 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.

ever, these soldiers do not contract childbed fever, because they are not puerperae and so they lack the disposing factor.

[43] 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 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,[10] 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. *[44]* 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.

10. 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.

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. *[45]* 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. *[46]* 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, however, this difference is undeniable. In 1848 Professor [Josef] Skoda[11] 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.[12]

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

11. 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.

12. 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.

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.

[47] 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.[13] 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.

[48] 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

13. See below, pp. 117f., 147f.; German edition, pp. 107f., 193f.

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. *[49]* As a result of 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 5 shows the mortality figures for 1846 and for the first five months of 1847. *[50]* 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.[14]

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

14. See below, pp. 101–5; German edition, pp. 71–75.

[49] TABLE 5

	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

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. *[51]* 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 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. *[52]* Everything was in question; everything seemed inexplicable; everything was doubtful. Only the large number of deaths was an unquestionable reality.

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 the 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.

[53] 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. *[54]* 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 contact 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. *[55]* 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 6).

[56] In these seven months, of the 1,841 maternity patients cared for, 56 died (3.04 percent). In 1846, before washing with chlorine was introduced, of 4,010 patients cared for in the first clinic, 459 died (11.4 percent). In the second clinic in 1846, of 3,754 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 3,490 patients, 176 died (5 percent). In the second clinic of 3,306 patients, 32 died (0.9 percent). In 1848, chlorine washings were employed throughout the year and of 3,556 patients, 45 died (1.27 percent). In the second clinic in the year 1848, of 3,219 patients 43 died (1.33 percent). The mortality rates for the individual months of 1848 are shown in Table 7.

[57] 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

[56] TABLE 6

	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	1,841	56	3.04
Avg.			

[57] TABLE 7

	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	3,556	45	
Avg.			1.27

there were 20 deaths (4.9 percent). On 20 March Dr. Carl Braun[15] 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. *[58]* Thus one could infer that the 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. *[59]* 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.

15. 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.

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. *[60]* 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.

[61] 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 3,066 patients only 26 died (.84 percent). In 1841, after the Viennese medical school adopted an anatomical orientation, of 3,036 patients 237 died (7.7 percent). In 1843 of 3,060 patients 274 died (8.9 percent). In 1827, of 3,294 patients 55 died (1.66 percent). In 1842 of 3,287 patients 518 died (15.8 percent).[16] 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 8.

[62–63] 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.

[64] Table 1 indicates the difference in mortality rates between

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

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 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.[17] 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 9). The variation in mortality for both clinics can be traced to the activities of those in the process of becoming physicians. *[65]* 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[18] 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

17. On page 130; German edition, page 139, 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.

18. 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.

[62] TABLE 8

	Births	Deaths	Rate	Year	Births	Deaths	Rate
1784	284	6	2.11	1817	2,735	25	0.91
1785	899	13	1.44	1818	2,568	56	2.18
1786	1,151	5	0.43	1819	3,089	154	4.98
1787	1,407	5	0.35	1820	2,998	75	2.50
1788	1,425	5	0.35	1821	3,294	55	1.66
1789	1,246	7	0.56	1822	3,066	26	0.84
1790	1,326	10	0.75	1823	2,872	214	7.45
1791	1,395	8	0.57	1824	2,911	144	4.94
1792	1,574	14	0.89	1825	2,594	229	4.82
1793	1,684	44	2.61	1826	2,359	192	8.12
1794	1,768	7	0.39	1827	2,367	51	2.15
1795	1,798	38	2.11	1828	2,833	101	3.56
1796	1,904	22	1.16	1829	3,012	140	4.64
1797	2,012	5	0.24	1830	2,797	111	3.97
1798	2,046	5	0.24	1831	3,353	222	6.62

1799	2,067	20	0.96	1832	3,331	105	3.15
1800	2,070	41	1.98	1833	3,907	205	5.25
1801	2,106	17	0.80	1834	4,218	355	8.41
1802	2,346	9	0.38	1835	4,040	227	5.61
1803	2,215	16	0.72	1836	4,144	331	7.98
1804	2,022	8	0.39	1837	4,363	375	8.59
1805	2,112	9	0.40	1838	4,560	179	3.92
1806	1,875	13	0.73	1839	4,992	248	4.96
1807	925	6	0.64	1840	5,166	328	6.44
1808	855	7	0.81	1841	5,454	330	6.05
1809	912	13	1.42	1842	6,024	730	12.11
1810	744	6	0.80	1843	5,914	457	7.72
1811	1,050	20	1.90	1844	6,244	336	5.38
1812	1,419	9	0.63	1845	6,756	313	4.63
1813	1,945	21	1.08	1846	7,027	567	8.06
1814	2,062	66	3.20	1847	7,039	210	2.98
1815	2,591	19	0.73	1848	7,095	91	1.28
1816	2,410	12	0.49				

[64] TABLE 9

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1839	2,781	151	5.4	2,010	91	4.5
1840	2,889	267	9.5	2,073	55	2.6

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.

[66] 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 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. *[67]* 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. *[68]* 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 3,533 infants in the first clinic, 235 died (6 percent). In the second clinic, of 3,398 infants 86 died (2.5 percent). In 1847, during the last seven months of which we washed with chlorine, of 3,322 infants 167 died (5.02 percent). In the second clinic, of 3,139 infants 90 died (2.8 percent). In 1848, when chlorine washings were practiced during the entire year, of 3,496 infants 147 died in the first clinic (4.2 percent). In the second clinic 100 infants died, out of 3,089 (3.2 percent). *[69]* 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."[19] 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

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

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. *[70]* Also, women who delivered prematurely became ill less often because they were not examined either. 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. *[71]* 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 5 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. *[72]* 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). *[73]* 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. *[74]* 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. *[75]* The reason 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.

[76] In order to confirm my views directly, I felt it was necessary to conduct animal experiments. With my friend, Dr. [George Maria] Lautner, assistant to Professor Rokitansky, I carried out experiments on rabbits. [Semmelweis devotes several pages to technical descriptions of the experiments, we continue with his summary.] *[77–80]* . . . It is hardly necessary to mention that the changes discovered in dissecting the rabbits are the same as those found in human bodies in consequence of puerperal diseases and, in general, in consequence of pyemia.

At the end of my two-year period of service I requested an extension of two additional years as had been awarded to Dr. Breit, my predecessor. Such an extension would have enabled me to further support my opinions on the origin of childbed fever; these opinions had occasioned numerous denials. My request was not granted, although at the same time my colleague in the second clinic [Franz Zipfl] was awarded a similar opportunity. *[81]* Indeed, my successor [Carl Braun] was also given a two-year extension of his period of service.

On 20 March 1849, after being released from the position of assistant, I petitioned to be made private docent of obstetrics.[20] My attempt met with no success. After a second petition and an eight-month wait, on 10 October 1850 I was named private docent of theoretical obstetrics, but I was limited to demonstrating and practicing on manikins. Such a limited docentship was of no use to me. While the law required equally encompassing in-

20. A private docent was a private lecturer or teacher who taught university students, but who was inferior in rank to a professor. A docent was often paid directly by the students with whom he worked.

struction for licensing a docent as for a professor, professors were permitted to demonstrate and to practice on cadavers. In October 1850, therefore, I returned to my native city of Pest.

One of the first evenings after I returned to Pest was spent in the company of a large number of physicians. Because of my presence, the conversation turned to childbed fever. Objections to my opinions regarding the origin of the disease were expressed. It was claimed that at that very time in the maternity ward of St. Rochus Hospital in Pest an epidemic of childbed fever was raging. Since the hospital was not a teaching institution, however, no students were examining patients with hands contaminated by decaying animal-organic matter.

On the following morning, in order to convince myself, I visited the maternity hospital. There I found a corpse, not yet removed, of a person who had just died of puerperal fever, another patient in severe agony, and four others seriously ill with the disease. The other persons present were not maternity patients but suffered from various disorders. *[82]* Thus the unhealthy condition of the maternity patients was clearly established, but this did not contradict but rather confirmed my opinion on the origin of childbed fever. Closer inspection disclosed that the obstetrical ward was not independent but was assigned to a surgical ward. The head physician of obstetrics was simultaneously a head surgeon and a juridical anatomist. Moreover, lacking a coroner, the various division physicians were obliged to perform autopsies. The head physician first visited the surgical wards and then the maternity ward. Thus, while the obstetrical ward of the St. Rochus Hospital had no student examiners whose hands were contaminated with decaying animal-organic matter, the head physician and the other physicians assigned to him, having visited the surgical ward, did examine with contaminated hands.

I have shown that the great mortality in the first clinic derived from the cadaverous particles adhering to the hands of the examiners. I have shown that in October 1847, ichorous particles from a discharging medullary carcinoma of the uterus brought on childbed fever. I have also shown that in November 1847, exuded ichorous particles from a carious knee brought on the disease. At the maternity ward of St. Rochus Hospital, the causal

factor of childbed fever was the decaying animal-organic substances that are found so abundantly in a surgical ward. *[83]* It may be necessary to say a few words about the maternity ward in the St. Rochus Hospital.

The St. Rochus Hospital belongs to the community of Pest and has a capacity of 600 patients, with three medical and two surgical head physicians. As already mentioned, the maternity ward was assigned to a head surgeon. As long as the obstetrical clinic of the Pest medical faculty is open, the St. Rochus Hospital does not admit maternity patients. This is in order that the clinic will not lack cases for instructional purposes. Only during the major vacation in August and September, when the obstetrical clinic of the Pest medical faculty is closed, are maternity patients admitted to St. Rochus Hospital. For the remaining ten months of the year, the area in which the maternity ward is located is used as a surgical ward. During the school year, only those women delivered who happened to go into labor while they were in St. Rochus Hospital suffering from miscellaneous illnesses. The obstetrical ward is located on the third floor of the building, and consists of a labor room and two maternity rooms. All six windows of these rooms overlook the morgue. Running beside the building in which the morgue is located is a wide street which partially dissipates the noxious exhalations of the morgue.

On 20 May 1851, I assumed the position of unpaid, honorary head physician of the obstetrical ward of the St. Rochus Hospital, thus dissolving the connection with the surgical wards. I functioned in this capacity for six years, until June 1857. During the school year, the location of the maternity ward became a gynecological rather than a surgical ward. *[84]* This removed the causal factor that had previously occasioned childbed fever, namely the degenerating animal-organic matter from the surgical ward. Consequently, childbed fever declined significantly.

We did not ordinarily employ chlorine washings, because we had no need to purify our hands from decaying animal-organic material. After the few autopsies that I was obliged to perform, I cleaned my hands with chloride of lime. In the vacation months of the school year 1850–51, there were 121 births recorded in

the maternity ward at St. Rochus Hospital. In succeeding years there were 189, 142, 156, 199, and 126 births. By 1855–56 there had been, therefore, 933 births. From these, 24 patients died, and of these only 8 from childbed fever (.85 percent). The remaining 16 died of the various diseases for which they were being treated while pregnant. At the onset of labor they had been transferred to the maternity ward. Of the 8 patients who died of childbed fever, 1 contracted the disease as follows: because of the breech position of the fetus, the mother was examined by an assistant surgeon. And this occurred just after he had performed an autopsy on a man who died of a gangrenous leg. Thus, in the maternity clinic of the St. Rochus Hospital over a period of six years, less than 1 percent of the maternity patients died of childbed fever. Formerly each year childbed fever had claimed many lives.

[85] On 18 July 1855, I was named Professor of Theoretical and Practical Obstetrics at the University of Pest. I began my activities in the obstetrical clinic in October of the same year. The obstetrical clinic is located on the third floor of the faculty building and consists of a labor room and four maternity rooms. In order to acquaint the reader with the conditions of this clinic, I will quote part of a petition I placed with the appropriate authorities for permission to leave this highly unsanitary and inconvenient location:

> The following considerations show that the obstetrical clinic is highly unsanitary: imperial ordinances stipulate that hospitals will have 16 square yards [*vier Quadratklafter* = 4 square fathoms] for each maternity bed. Since the obstetrical clinic has twenty-six beds, this ordinance requires 416 square yards. However, the clinic has only 164 square yards. Moreover, it lacks the space that is required for a large number of students. Three rooms are so small that not even half the students can gain admission at one time. The other two rooms are barely large enough to accommodate all the students without pressing them together so much that they are immobile. The air becomes so stale that it is dangerous to the patients; every disinterested person recognizes this. *[86]* Three smokestacks from the chemical laboratory are in the window columns of two of the rooms. When there are fires in the corresponding hearths, the temperature in these rooms becomes unbearable. The obstetrical clinic is so restricted in area that no room can be reserved for those who are ill. Thus, ill patients remain scattered among the healthy, and in this way childbed fever is spread. Childbed fever is not a contagious disease, but it can, under certain circumstances, spread from one person to another.
>
> Two of the clinic's windows open onto the northern light well and six windows onto the western. The northern shaft is 8 yards wide and a fire wall encloses it to the height of the windows of the maternity ward. The lavatories for the first three floors are in this light well. On the ground floor, next to the lavatory, is the building garbage pit. This decaying mass exudes a penetrating stench. Facilities for elementary and pathological anatomy are on the first floor. Drains from these facilities, through which all their liquid wastes are discharged, are immediately below the windows of the maternity clinic. The second floor contains the chemistry department. The morgue is between the light wells. The west light well is 2 yards wide and is enclosed by a wall 6 yards high. On the opposite side of this wall is an open field. Part of the morgue is in this shaft, and again, on the first floor are elementary and pathological anatomy, with chemistry on the second floor.
>
> This is not the place for an exposition of the petitioner's views on childbed fever. *[87]* It is sufficient to note that he believes that every case of childbed fever without exception is due to the incorporation of decaying animal-organic matter. The honorable college of professors can well imagine the undesirable position of a professor of obstetrics who has this conviction and who is obliged to choose either to have the windows hermetically sealed, and so to allow his patients to grow worse by breathing air befouled by throngs of students, or to leave the windows open and to admit air saturated with corrupted organic matter.
>
> But however dark the present situation may be, if the clinic remains in the same location it has an even more dismal future. A three-story building is to be erected on the empty lot opposite the western light well. This will totally obstruct light from the windows of the obstetrical clinic. Moreover, the exhalations from the narrow light well will no longer be dissipated over the empty lot; they will be conducted in a highly dangerous concentration from the three-story building to the windows of the obstetrical clinic.
>
> Whether the petitioner's patients enjoy good health or die from

childbed fever is not only of importance for those cared for in this clinic. The consequences of the petitioner's efforts regarding the health of his patients is of significance to all humanity.

[88] That childbed fever causes significantly more deaths in maternity hospitals than outside them is known to both physicians and laymen. In official documents, maternity hospitals are termed "deathtraps" not only by physicians but also by officials. Because childbed fever rages in the hospitals the question has been discussed repeatedly whether it would not be in the interests of humanity to close them. Only a detestable dilemma saves the maternity hospitals from destruction: if individuals deliver in a maternity hospital, childbed fever rages horribly, and many in the prime of life descend into an early grave. However, if the maternity hospitals are closed, larger numbers of women certainly remain healthy, but they become concerned about themselves and the care of their infants. Thus because of their need, the crimes of abortion, abandonment, and infanticide take place. The maternity hospitals are endured because it is believed to be better for patients to risk childbed fever inside them than to risk misery outside that may lead them to prison.

The petitioner has found the cause of childbed fever and has shown how to prevent it. The attention of advocates and opponents of these doctrines is focused on the health of the patients he treats. If their health becomes unfavorable, then the advocates of these opinions are weakened, and the opponents are strengthened in their doubts. *[89]* In this way, the spread of the petitioner's teachings will be hindered and humanity will continue to endure this plague. . . .

[90–95] [Semmelweis concludes the petition by quoting other authorities who had criticized the unsanitary conditions of the obstetrical clinic in Pest. He then notes:] In this location within ten months, 500 maternity patients are cared for, and 60 to 70 obstetricians and 180–90 midwives are trained. For obstetricians the course in practical obstetrics lasts two months; for student midwives the course lasts five months. Thus at any given time, the teacher is surrounded by at least 100 students. In the school year 1855–56, 514 maternity patients were cared for; 5 died, 2 of childbed fever (.19 percent) and 3 of other diseases. In 1856–57, 558 patients were cared for, 31 died, 16 of childbed fever (2.9 percent) and 15 of other diseases. In 1857–58, 457 patients were

cared for, 23 died, 18 of childbed fever (4.05 percent) and 5 of other diseases.

The mortality in 1856–58 resulted in an official correspondence. This is partially quoted here in order that the reader may become familiar with the cause of these deaths:

Confidential reports have been made concerning various inappropriate circumstances in the obstetrical clinic of the Imperial University. For example, through the carelessness of the head midwife, not only is the bed linen seldom changed, but indeed, linen that is still befouled with the blood of earlier deceased patients is spread under newly admitted patients. As a result of this, mortality at the beginning of this year was so high that as many as ten patients died on a single day.

[96] This fact is all the more striking in that during the previous year, in a period with a much lower mortality rate, this situation was noted by the Professor and a larger allocation of bed linen was sought. So much was immediately obtained that a store of bed linen was provided consisting of several hundred more sheets than were needed. Also, as requested, a quantity of bedding and personal linen was provided during the vacation. The costs were so great that they did not escape the notice of the Minister of Culture and Education.

The Professor appears, therefore, to be in agreement with others who have access to the clinic in recognizing that the increase of sickness and death is not due to lack of linen, or to irregular deliveries from the laundress, but to inattention and irregularity in managing the linen.

I responded as follows:

I am certainly in agreement with others who have access to the clinic in recognizing that the greater mortality rate of 1857–58 is not due to a lack of linen or to irregular deliveries from the laundress, but to inattention and irregularity in managing the linen. *[97]* However, an attendant, not the head midwife, was responsible. This attendant has subsequently been discharged.

In 1856–57, thirty-one patients died. Sixteen of these died from childbed fever because of a lack of linen and because of irregular deliveries from the laundress. In the school year 1857–58, twenty-four patients died. Of these, eighteen died of childbed fever be-

cause of inattention and irregularity in managing the linen.

Never more than two individuals died on a given day. If one says, therefore, that in 1856–57 a much smaller mortality rate prevailed, and that at the beginning of the school year 1857–58 as many as ten patients died on a single day, one is simply not telling the truth.

Linen still befouled with the blood of dead patients was never spread under new patients. This charge must, therefore, have reference to linen that we received from the laundry in the school year 1856–57 that was supposed to have been laundered but that, in fact, was still befouled with blood and lochial discharge. I personally demonstrated, in a report, that this linen occasioned childbed fever in the clinic.[21]

From the first medical writers on, from Hippocrates until the most recent times, it was the unchallenged conviction of all physicians of all times that the horrible ravages of childbed fever among maternity patients were epidemic—that is, they were due to atmospheric influences. Influences that, notwithstanding every possible precaution of the physician, ravaged unabated and without remission. In 1847, in the great Viennese maternity hospital, I succeeded in proving that this opinion is false and that every single case of childbed fever is occasioned by infection. *[98]* In consequence of the measures suggested by my views, during twenty-one months in Vienna, six years in the St. Rochus Hospital, and one year at the clinic at Pest, I have had no epidemics. Previously all these institutions were devastated by horrible epidemics. I regard the two disastrous years that followed as unintentional, tragic, but direct proof of the accuracy of the opinion published in my report.

The beneficial consequences of my opinion regarding the generation of childbed fever have been compared with those of Jenner's cowpox inoculations. I realize how presumptuous it is to assert such a thing of myself; only the fact that precisely my clinic has been accused of greater mortality forces me to make such an assertion. Given that I am not responsible for the increased mortality at the clinic at Pest, perhaps my nine years of shining success can be seen in a more favorable light.

21. The report was "A gyermekágyi láz kóroktana [The Etiology of Childbed Fever]" published in *Orvosi hetilap* 2(1858); a translation is included in Tiberius von György, *Semmelweis' gesammelte Werke* (Jena: Gustav Fischer, 1905), pp. 61–83. This was Semmelweis's first publication on the subject of puerperal fever. According to György the substance of the report was contained in lectures delivered before the Budapester königliche Ärzteverein in the spring of 1858. Ibid., p. 601.

From this official correspondence, the reader can easily determine that mortality among maternity patients during these two years was caused by unclean bed linen in combination with the generally unsanitary conditions of the clinic.

The laundry is given out to a contractor who is required to exchange the linen weekly. During the school year 1856–57, the responsible officials felt that too much was paid for the laundry service, and it was released to the minimum bidder. Of course the minimum bidder does not guarantee the best work—only the cheapest. *[99]* The price was so reduced that it was impossible, especially in winter, to furnish clean linen. Childbed fever was caused by the use of such poorly laundered linen. After complaints were registered, the earlier contractor was given the laundry again at the original price and the disease became less frequent. In the school year 1857–58 it was again unclean linen that occasioned the greater mortality rate, but the linen was not delivered dirty from the contractor. Rather, the attendant neglected to change the linen regularly. Blood and lochial discharge decayed to such a degree that childbed fever was again encountered. Through thorough cleaning of the bed linen and the dismissal of the attendant, the mortality was again reduced. . . .

[Semmelweis repeats the various ways infection can occur, as well as certain other conclusions. The chapter ends with this paragraph:] *[100–101]* In the school year 1857–58, the external genitals of two patients became gangrenous. Because of a shortage of space, they were obliged to remain among the other patients. To isolate them as much as possible, they were cared for by two student midwives in twelve-hour shifts. These midwives had orders not to touch any other individual. Nevertheless, one of them was caught as she prepared to examine a newly admitted patient.[22]

22. In another essay Semmelweis reports that the patient became ill from this examination but later recovered. György, op. cit., note 21 above, p. 74. He also notes that one of the attendants had previously pricked her finger with a pin. From the gangrenous patients she contracted puerperal fever and nearly died. See below, pp. 148–49; German edition, p. 195.

Chapter 2

The Concept of Childbed Fever

[102] Based on experience of over fifteen years in three different institutions, all of which were severely afflicted with childbed fever, I regard the disease, without a single exception, as a resorption fever dependent on the resorption of decaying animal-organic matter. Resorption first causes disintegration of the blood. This is followed by exudation. In the overwhelming majority of cases the decaying animal-organic matter is conveyed to the individual from external sources. These are the cases represented as epidemics of childbed fever; these are the cases that can be prevented. Occasionally, decaying animal-organic matter is generated within the attacked organism. This is self-infection and cannot always be prevented.

The source of decaying animal-organic matter can be a corpse of any age, of either sex, regardless of the preceding disease, regardless whether the corpse is a pregnant woman or not. Only the degree of decomposition of the corpse should be taken into consideration. *[103]* These assorted corpses are the ones which people who practiced in the first clinic examined. The source of decaying animal-organic matter can be a diseased person of any age, of either sex, regardless whether the individual suffers from childbed fever; only whether the decaying animal-organic matter is a result of the disease comes into question. In the first clinic in October 1847 childbed fever was caused by a discharging medullary carcinoma of the uterus, and in November 1847 by the exhalations of a carious knee. In the maternity ward of the St. Rochus Hospital childbed fever was caused by the ichorous products of various surgical disorders. The source of the decaying animal-organic matter is every physiological animal-organic structure that, having been withdrawn from the vital order, has

reached a specific degree of decay. Not the nature of the structure but only the degree of decomposition comes into question. In the obstetrical clinic at Pest during the school years 1856–57 and 1857–58 childbed fever was caused by physiological blood and normal lochial discharge that adhered to the bed linen and began to decay.[1]

Decaying animal-organic matter is carried by examining fingers, operating hands, instruments, bed linen, the atmosphere, sponges, basins, hands of midwives and attendants. *[104]* In other words, anything that is contaminated by decaying animal-organic matter and that comes into contact with the genitals of patients.

The decaying animal-organic matter is resorbed at the inner surface of the uterus from the external orifice upward. As a result of pregnancy, this surface is denuded of its mucous membrane and is therefore unusually resorbant. The remaining undamaged parts of the genitals, which also lack a mucous membrane, do not resorb because of the thick layer of epithelium [cells joined with cementing substances that cover all the interior and exterior surfaces of the body]. When injured, each part of the genitals can become a site of resorption. Infections seldom occur during pregnancy because, since the orifice of the uterus is closed, the resorbant inner surface is inaccessible. External infection can occur during pregnancy when the orifice of the uterus is open and the resorbant inner surface is accessible. The rarity of external infections during pregnancy is also explained by the fact that even when the orifice is open, it is seldom necessary in manual examinations to touch these inner surfaces. I have neglected to record how frequently in the first clinic childbed fever occurred during pregnancy. I believe it happened approximately twenty times a year. Childbed fever always terminates pregnancy. Only one patient died of childbed fever while

1. In their early accounts of Semmelweis's work, both C. H. F. Routh and Josef Skoda suggested that corpses were the only source of infection that was involved in puerperal fever. Consequently, many of Semmelweis's critics attacked this indefensible position, and his views seemed to be much weaker than they really were. This misunderstanding certainly delayed comprehension and adoption of Semmelweis's prophylactic measures. In his book Semmelweis continually stresses the other sources of contamination.

still pregnant; she was delivered by me, postmortem, by Caesarean section, in order to save the child.

[105] Infection occurs most often during dilation. Not only is the inner surface of the uterus accessible at that time but it is also frequently necessary to penetrate the uterus in manual examination to determine the location and position of the fetus. Thus, before chlorine washings, almost every patient whose period of dilation was extended died of childbed fever. During delivery the body of the fetus, as it pushes down, renders the inner surface of the uterus inaccessible. During delivery, therefore, infection seldom occurs. Immediately after birth and thereafter, the inner surface of the uterus is again accessible, and during this period infection occurs, especially when air saturated with decaying animal-organic matter penetrates the genitals. In November 1847, air in the maternity rooms of the first clinic became permeated with the exudations of a carious knee. This air penetrated the lacerated genitals of the patients and caused childbed fever. After birth, infection can also occur when the genitals, injured by the passage of the fetus, are brought into contact with bed linen that is contaminated with decaying animal-organic matter. During the school years 1856–57 and 1857–58 childbed fever was caused by unclean linen at the obstetrical clinic in Pest.

[106] In rare cases, decaying animal-organic matter originates inside the affected person. This occurs when organic matter that should be discharged during delivery begins to decay before being discharged. In being resorbed, it causes childbed fever by self-infection. The organic matter can be lochia, decidual remnants, clotted blood that is retained in the uterus, etc. Alternatively, the decaying animal-organic matter can be a product of pathological processes. For example, as a result of a difficult operation with forceps, portions of the genitals may be crushed and become gangrenous. Upon resorption, the gangrenous particles cause childbed fever by self-infection.

Suppose we explain childbed fever as a fever of resorption in which the introduction of decaying animal-organic matter leads to disintegration of the blood and to exudation. Then childbed fever is not a disease unique to and appearing only in the newly delivered, because as a result of resorption the disease may arise

during pregnancy or when giving birth. The disease can be conveyed to infants, whether male or female. In consequence of resorption of decaying matter it can also be found among anatomists, surgeons, in operative cases in surgical wards, etc. Kolletschka also had this disease. Thus childbed fever is not a species of disease; rather it is a type of pyemia.

Various concepts are associated with the expression 'pyemia' and it is necessary, therefore, to explain that I understand this term as referring to disintegration of the blood through decaying animal-organic matter. *[107]* One type of pyemia I call childbed fever, because in pyemia of maternity patients one finds phenomena in the genital region that are not found in other people. Anatomists and surgeons who die of pyemia do not have endometritis [serious infection of the mucous membrane of the uterus], etc.

Childbed fever is not a contagious disease. A contagious disease is one that produces the contagion by which the disease is spread. This contagion brings about only the same disease in other individuals. Smallpox is a contagious disease because smallpox generates the contagion that causes smallpox in others. Smallpox causes only smallpox and no other disease. Scarlet fever cannot be contracted from someone suffering from smallpox. Conversely, another disease can never bring about smallpox. For example, a person suffering from scarlet fever can never cause smallpox in another person. Childbed fever is different. This fever can be caused in healthy patients through other diseases. In the first clinic it was caused by a discharging medullary carcinoma of the uterus, by exhalations from a carious knee, and by cadaverous particles from heterogeneous corpses. In the maternity ward of the St. Rochus Hospital it originated because of decaying matter from the surgical ward, etc. However, childbed fever cannot be transmitted to a healthy maternity patient unless decaying animal-organic matter is conveyed. *[108]* For example, suppose a patient is seriously ill with a form of childbed fever in which no decaying matter is produced. Then the disease cannot be transmitted to healthy patients. On the other hand, if the patient with childbed fever has septic endometritis or discharging metastases, then her disease can be conveyed to healthy patients.

This explains why the conflict over whether childbed fever is or is not contagious could never be conclusively resolved. Those who believe in contagion cite cases in which childbed fever had undeniably spread from an ill patient to a healthy one. Their opponents cite cases in which the disease did not spread as it would have done if it had been contagious. Childbed fever is not a contagious disease, but it can be conveyed from diseased to healthy patients by decaying animal-organic matter. After death, the corpses of puerperae, like all corpses, are sources of the decaying matter that causes the disease.

I assert that in the overwhelming majority of cases childbed fever is caused by external infection and that these cases can be prevented. If we disregard all these cases and also all deaths during maternity that are not due to childbed fever, how many patients are left that die from childbed fever through self-infection? */109/* As yet, I am not able to provide numbers with which to answer this question. The three institutions in which I made my observations did not adopt those prophylactic measures that are necessary to avoid all cases of external infection. The petition, part of which I quoted earlier, was intended to help me procure a new maternity hospital that would avoid all such cases. Should my petition prove fruitful, and should I have the opportunity over a longer period of years to make observations in such a hospital, it would be possible to determine the number of cases of unavoidable self-infection. Otherwise I must leave it to another more fortunate colleague to determine.

/110/ For the present, I accept as a standard the mortality rate of the Viennese maternity hospital from the time before adoption of the anatomical orientation. In the eighteenth century, and in the first decades of the nineteenth century, there were twenty-five years in which not even one patient in one hundred died. [Semmelweis provides a list of twenty-five years between 1786 and 1822 in which the mortality rate ranged from one in one hundred to one in four hundred. After summarizing the list he continues:] */111/* This small mortality rate may not be the smallest possible. Some of the patients may have died from diseases other than childbed fever. Moreover, even then there could have been infection from external sources; for example, ichor from

diseased patients could have been conveyed to healthy patients. This did in fact occur even before Viennese medicine adopted an anatomical orientation, since even then mortality occasionally reached the rate of 4 percent. The Viennese maternity hospital opened in 1784. Of its first thirty-nine years there were twenty-five during which less than 1 percent died, seven during which 1 percent died, five during which 2 percent died, one year (1814) in which 3 percent and one year (1819) in which 4 percent died. Subject to the above reservations, the twenty-five years within which less than 1 percent died can be adopted as a standard for the frequency of self-infection.

/112/ If we judge my results against this standard, it becomes apparent that I was not always successful in restricting the disease to unavoidable cases of self-infection. Occasionally there were cases of external infection. In the last seven months of 1847, in spite of chlorine washings, 56 of 1,841 patients died (3.04 percent). In 1848, when chlorine washings were used throughout the year, 45 of 3,780 patients died (1.19 percent). In January and February of 1849, 21 of 801 patients died (2.62 percent). If we consider individual months, then during only seven months did we successfully restrict deaths to cases of self-infection. Of 276 patients in March 1848 and of 261 patients in August 1848 not a single patient died; in five other months less than 1 percent died. The requirements for the prophylaxis of childbed fever were certainly not met by the construction of the first clinic; this prevented me from limiting deaths to cases of self-infection. */113/* Moreover, at the time I first arrived at my new convictions, I was myself so inexperienced that in October 1847 I did not wash with chlorine after examining a discharging medullary carcinoma of the uterus, and in November 1847 I did not isolate a patient with a discharging carious knee. [Semmelweis reviews other incidents that caused childbed fever in his practice.]

Chapter 3

Etiology

[114] In discussing the concept of childbed fever, I asserted that every case of the disease, without exception, comes about by the resorption of decaying animal-organic matter. I claimed that in the majority of cases this matter is brought to the individual from external sources and that in only a few cases it is produced internally. For me, therefore, the etiological factors of childbed fever include only those which convey decomposing animal-organic matter to the individual from without or which generate this matter internally. Earlier, we examined the accepted etiological factors of childbed fever as an explanation for the increased mortality in the first clinic. We must now determine to what extent these factors can either bring decaying animal-organic matter to the individual or generate it internally. *[115]* We will admit as causes only those factors that do one of these; other factors must be rejected.

Today, the commonest opinion among physicians is that childbed fever consists in a disintegration of the blood and that the anatomical products of childbed fever are excretions from the disintegrated blood. I share this conviction. Epidemic and endemic influences, emotional disturbances, mistakes in diet, and catching a cold have all been claimed to cause the blood to disintegrate. We will now examine these claims. . . .

[116] We begin with epidemic influences. My unshakable conviction is that there are no epidemic influences sufficient to cause childbed fever, that there never have been epidemic causes, and that the endless series of epidemics recorded in medical literature consists entirely of instances of infection from external sources which could have been prevented. The considerations that give me the courage to contradict an opinion so many centuries old

are the following: the ground, the unshakable rock, on which I erect my teaching is the fact that from May 1847 until the present day, 19 April 1859, that is for over twelve years, in three different institutions, I succeeded in limiting childbed fever to isolated cases. Even the most recalcitrant advocates of the epidemic concept are unable to call this an epidemic. And even when the number of deaths increased, it could be proven that the increased mortality was not an epidemic, due to atmospheric-cosmic-terrestrial influences, that is, but rather that it was always due to decaying animal-organic matter transmitted to individuals in spite of my precautions. *[117]* There can be no defense against childbed fever that is due to atmospheric-cosmic-terrestrial influences. Advocates of the epidemic theory secure themselves behind this indefensibility; they thereby escape all responsibility for the devastations of the disease. I myself am impotent against such influences. I do not know what can be done to remove patients from destructive atmospheric-cosmic-terrestrial influences. Thus, if I am successful in preventing a disease long regarded as unpreventable, this proves that the disease is not due to inescapable atmospheric-cosmic-terrestrial influences, and that the cause of the disease is preventable.

The great mortality that is cited by advocates of the epidemic concept does not prove the existence of epidemic influences. . . . *[118]* Suppose that in a specific time many individuals become ill and die of the same disease. As I have indicated, this does not warrant calling it an epidemic. Otherwise, every battle would be an epidemic; in every battle many individuals suffer and die from the same disorder. The concept of an epidemic involves the cause of childbed fever and is completely independent of numbers. Only those cases of childbed fever that are due to atmospheric-cosmic-terrestrial influences are epidemic. . . . It is obvious that epidemic influences bring no decaying animal-organic matter to individuals. However, it is conceivable that atmospheric-cosmic-terrestrial influences result in the production of such matter in many individuals at a given time, that this is then resorbed, and that childbed fever results through self-infection. Such childbed fever would indeed be epidemic. . . . However, if childbed fever were caused by epidemic influences, then, as is

observed with other epidemics, it must be limited to a particular season. Opposing atmospheric influences could not have the same results. *[119]* We have proven that childbed fever is not associated with a particular season. Childbed fever occurs in both large and small numbers during each month of the year. . . .

[120–21] The prevailing opinion is that winter is the season most conducive to outbreaks of childbed fever. [Semmelweis refers to earlier tables that prove that] winter really is more frequently an unhealthy time for patients. But this is not to be explained by winter atmospheric influences, since then childbed fever would never occur in larger numbers in summer. It is rather to be explained by the different activities of those who visit the maternity hospital. These activities are determined by the season. After the long vacation in August and September, students resume their studies, including obstetrics, with renewed diligence. In winter the influx of students into the maternity hospital is so great that individuals must wait weeks and even months for their turn to study. In summer, during vacation, half or even two-thirds of the places are vacant. In winter, the pathological and forensic autopsies and the medical and surgical wards in the Imperial Hospital are visited industriously by those who also visit the maternity hospital. In summer, the diligence is noticeably less. The charming surroundings of Vienna are more attractive than the reeking morgue or the sultry wards of the hospital. In winter the assistant of obstetrics holds practical operative exercises on cadavers before the afternoon rounds at four o'clock, because in the mornings students are otherwise engaged, and following the afternoon rounds, at five o'clock, it is already too dark. *[122]* In summer the heat is too oppressive before the afternoon visit, and the operative exercises are held in the evening following afternoon rounds. Does it make any difference to the health of examined patients whether students make rounds before or immediately after they dissect cadavers? These circumstances generally make winter unhealthy and summer healthy for the patients. Suppose we assume that the atmospheric influences of winter really make winter an unhealthy time for patients. Then, assuming that over the twenty-five years when the mortality rate was below 1 percent there were no winter epidemics, must one not suspect that through twenty-five years Vienna had no winter? . . .

[123] Just as, if childbed fever were dependent on atmospheric influences, it would be limited to certain seasons, so too the disease would appear only in those climates typified by the weather of that season. In reality it appears in large numbers in all climates. Moreover, in all climates there are maternity hospitals in which it does not appear in large numbers. This appearance and nonappearance of large numbers of cases in different climatic areas cannot be explained by atmospheric influences. It can only be explained by decaying animal-organic matter. In maternity hospitals in different climatic zones where individuals are infected from external sources, childbed fever appears in large numbers and is then falsely called an epidemic. . . .

Irrespective of the climate, favorable health among maternity patients indicates that the hospital is not a teaching institution. The reason for this is obvious. Hospitals that are not teaching institutions but in which individuals are, nevertheless, infected from external sources constitute an exception. . . . *[124]* The Vienna paid maternity ward, a ward for confinement births, belongs in this category. Not only is this ward not used for teaching, but to achieve its purpose it is hermetically closed to all persons other than the physicians employed there. One can, therefore, assume that in this ward there would be no cases of infection from external sources, and as a result, there would be less than 1 percent mortality. However, a glance at the mortality rates for this ward in Table 10 indicates something different. The mortality rate was actually larger than this. A few hours or days after delivery, patients were often released healthy, or occasionally even unhealthy, so that they could return home or be admitted to the general hospital. After I adopted chlorine washings, the health of maternity patients even in the disparaged first clinic was superior to that of patients in the paid ward.

[125] This will cease to be mysterious once I explain these circumstances: the directors of this ward were [Eduard] Mikschik and [Baptist Johann] Chiari.[1] The reader who is familiar

1. Baptist Johann Chiari (1817–54) was Professor of Obstetrics in the Univer-

[124] TABLE 10

	Births	Deaths	Rate
1839	202	6	2.9
1840	204	6	2.9
1841	249	7	2.8
1842	358	10	2.7
1843	367	19	5.2
1844	362	8	2.2
1845	311	6	1.9
1846	315	3	0.9
1847	258	3	1.1
1848	213	4	1.8
Total	2,839	72	
Avg.			2.5

with medical literature will know what these physicians have accomplished. But these accomplishments were possible only through activities in which their hands must have become contaminated with decaying animal-organic matter. Both physicians were simultaneously directors of the gynecological ward of the general hospital. How dangerous a gynecological ward can become for a maternity ward was proven by the discharging medullary carcinoma of the uterus that in October 1847 led to devastation in the first clinic. In the Viennese general hospital between 600 and 800 forensic autopsies are performed each year. The custom is that on alternate weeks one or the other of the two youngest head physicians has to be present as a legal witness. When Mikschik was named chief physician, he was the youngest. After his departure, Chiari was the youngest. Thus, each was obliged to attend the forensic autopsies every other week. Is the unhealthy condition of the maternity patients in the paid ward still mysterious?

Patients in maternity hospitals that are simultaneously teaching institutions are less healthy than those not in teaching institutions. Among teaching institutions, those restricted to educat-

sity of Prague and later for a short time in the Josephinum of Vienna. He was the son-in-law of Johann Klein. He supported Semmelweis from the beginning, but also expressed reservations. Semmelweis was generally favorable in his comments about Chiari.

ing midwives have more favorable mortality rates than those restricted to educating physicians. *[126]* This is because the curricula are so arranged that student midwives do not contaminate their hands with decaying matter as often as medical students do.

The Maternité in Paris is an exception. It is exclusively for the education of midwives, but it has a mortality rate as great as [Paul-Antoine] Dubois's Paris Clinic for the education of physicians. Dr. [Franz Hektor] Arneth[2] has said of the location of Dubois's Clinic, "It is regrettable that the autopsy room of the hospital is so near."[3] The Maternité has a mortality rate comparable to that of Dubois's Clinic. [Semmelweis gives tables showing the mortality rates of the two institutions.] *[127–28]* In the Maternité, the curriculum is such that midwives contaminate their hands with decaying matter as frequently as would be the case elsewhere only among physicians. [Johann Friedrich Osiander describes the Maternité as follows:]

> The hospital midwives and some of their students accompanied the physician on his daily rounds through the infirmary for maternity patients. Each student was assigned a diseased patient for particular observation and was expected to prepare a short case history of the birth and of the physician's treatment. . . . *[129]* . . . Autopsies were conducted in a building in the garden somewhat removed from the maternity hospital; these were usually attended by student midwives. I was often astonished to see the active part some of the young women took in the dissection of corpses. With bare and bloody arms, holding large knives in their hands, laughing and quarreling, they cut the pelvis apart, having received permission from the physician to prepare the corpse for him.[4]. . .

2. Franz Hektor Arneth (1818–1907) was assistant in the second clinic while Semmelweis was assistant in the first clinic. Arneth gave favorable lectures on Semmelweis in Paris and in London. After spending some time in Russia, Arneth became a successful practitioner in Vienna.

3. Franz Hektor Arneth, *Über Geburtshülfe und Gynäkologie in Frankreich, Grossbritannien und Irland* (Vienna [Braumüller], 1853), [p. 51; author's note].

4. Johann Friedrich Osiander, *Bemerkungen über die französische Geburtshülfe, nebst einer ausführlichen Beschreibung der Maternité in Paris* (Hannover: Hahn, 1813), pp. 33, 46 [author's note].

[130] . . . The reader can understand how the midwives in the Maternité contaminate their hands with decaying matter.[5]

If childbed fever were due to atmospheric-cosmic-terrestrial influences, then it would be impossible for there to be a so-called epidemic of childbed fever in some hospitals and for other hospitals in the same climatic zone to be spared. Over a long period of time, it would be even more unlikely for these influences to devastate, in varying degrees, the two divisions of one hospital. *[131]* Table 1 shows that through six years the maternity patients in the first clinic in Vienna died at a rate consistently three times greater than the patients in the second clinic. This observation first made me doubt the epidemic concept of childbed fever.

The same inequality of mortality between two divisions of one institution occurred in Strasbourg. Dr. Arneth writes that in Strasbourg,

> The maternity hospital consists of two divisions, the clinic for physicians and the division where midwives are trained. Until the end of 1845 the two divisions were under different directors; they were next to one another, separated only by a thin wall. Admissions were arranged so that maternity patients went alternately into the two divisions. During the vacation all were accepted into the clinic. Since [Charles Henri] Ehrmann left, both divisions have been directed by [Joseph-Alexis] Stoltz. It is not possible to determine the precise mortality rates, but both professors agree that the clinic for male students has consistently more deaths.[6]

For more precise information I wrote to Dr. [Friedrich] Wieger[7] and Professor Stoltz in Strasbourg, and received the following replies. Dr. Wieger wrote:

5. It was always possible for midwives to become contaminated with decaying matter. In a lecture in 1846 Kolletschka is reputed to have said, "It is here no uncommon thing for midwives, especially in the commencement of their practice, to pull off legs and arms of infants, and even to pull away the entire body and leave the head in the uterus. Such occurrences are not altogether uncommon; they often happen." *Lancet* 2(1855): 503.

6. Arneth, op. cit., note 3 above, p. 30.

7. Wieger had been Semmelweis's student in the first clinic and was, in his own words, an eyewitness to Semmelweis's discovery; he also wrote a positive report on Semmelweis's work that appeared in a French medical periodical. Judging by Stoltz's comments below, however, Wieger either failed to grasp the significance of Semmelweis's work or he did not have a significant influence on his associates.

8. Strasbourg, 19 May 1858 [author's note].

9. Strasbourg, 26 March 1858 [author's note].

> *[132]* . . . What Arneth has told you is correct. During the time that the midwife school was under Professor Ehrmann, puerperal fever was relatively unknown. At that time both divisions were housed in wings in the third floor of the large hospital, separated only by a room containing the beds of students who lived in the building. When Professor Stoltz took over both divisions, the disease became common in both, and it remains so. The divisions are now united in an attractive, newly erected pavilion.[8]

Professor Stoltz writes:

> . . . The passage in Arneth's book . . . is accurate, but I have always attributed the difference in mortality to the sanitation of the two divisions. In fact, the rooms of the faculty clinic are inferior, less spacious, and always overcrowded, while the midwives' rooms are well-ventilated, well-appointed, and contain proportionately fewer beds. They are also kept cleaner, and in the course of a year they contain fewer pregnant and diseased patients. *[133]* The more difficult cases are always assigned to the faculty clinic.
>
> Until 1856 both divisions were part of the general hospital. Last year they were moved into a single, well-situated building facing south and west, and surrounded by courtyards and a garden. The two clinics are separated by a lecture hall and a room for instruments. Pregnant women are given places on the ground floor. The division for midwives is still more conveniently arranged than that for the faculty. Nevertheless, in the winter of 1856–57, here as in Munich, there was an equally fatal and devastating epidemic in both divisions. This is in spite of the fact that the faculty clinic employed chlorine for disinfecting hands.
>
> You see, honored colleague, that our observations of your theory of the etiology of puerperal fever have not been favorable. I will, nevertheless, read with great interest your work on these matters and attempt to follow painstakingly all your prescriptions.
>
> I am delighted to have entered into this scientific correspondence with you, and I will be pleased if it is not limited to this one occasion.[9] . . .

[134] . . . Does it not contradict good sense to insist that childbed fever, which before the unification of the two divisions was limited to the division for physicians, is epidemic, that is, dependent on atmospheric-cosmic-terrestrial influences? Professor Stoltz himself sought the cause of childbed fever in endemic noxious influences, particularly in the difference in sanitary conditions between the divisions. However, the more favorable conditions of the division for midwives did not protect it from childbed fever, because those conditions were nullified once it ceased to be exclusively for midwives. Also, in the new building, the more favorable conditions of the division for midwives did not protect it from childbed fever. . . . *[135]* Judgment about the ineffectiveness of the chlorine washing will be reserved for another part of this work.[10]

Before the Strasbourg school for midwives was unified with the division for physicians, and from the time the Viennese second clinic was devoted exclusively to midwives until adoption of chlorine washing in May 1847, these two proved that patients are healthier in teaching institutions where only midwives are trained than in institutions for training physicians.

The great mortality rate in maternity hospitals is not dependent on atmospheric conditions but rather on decaying animal-organic matter. This must be true, since so-called epidemic childbed fever began in various hospitals when decaying matter was first conveyed to patients in a specific, regular way. Osiander relates that the Maternité in Paris was horribly ravaged by childbed fever in 1803 and in 1808. We find in the educational system of the Maternité a sufficient etiology. Between 1803 and 1808 in Vienna less than 1 percent died from childbed fever; there the so-called epidemic of childbed fever began in 1823. This was the time when Viennese medicine adopted an anatomical orientation. *[136]* Professor Rokitansky has functioned at the Institute for Pathological Anatomy since 1828. For twenty-four years, from 1823 until chlorine washings began in 1847, mortality al-

10. In discussing the reaction to his work, Semmelweis later points out that opponents to his theories are not likely to use chlorine conscientiously and that their unfavorable results, therefore, are not a reliable indication of the usefulness of his methods. See below, pp. 194f.; German edition, pp. 330–32.

ways exceeded 2 percent and was sometimes as high as 12 percent. During the thirty-nine years from 1784 through 1822 mortality never exceeded 4 percent, and in twenty-five years it was less than 1 percent. In a letter that I will later quote in full, the late [Gustav Adolf] Michaelis wrote from the Kiel maternity hospital, "Puerperal fever first appeared here in 1834. That is, however, approximately the time when I first became active in instruction and when students began examining regularly." Epidemic childbed fever first appeared in the Strasbourg school for midwives in 1845, the year in which it was united with the division for physicians. In the St. Rochus Hospital in Pest the health of maternity patients has been continuously unfavorable since the existence of the obstetrical division. This is because the maternity ward was an appendage to the surgical division. However, the health of the maternity patients of the medical faculty of Pest was always good until the 1840s. In Pest, medicine adopted an anatomical orientation at that time.

My predecessor, Councilor [Ede Flórián] Birly,[11] once Boër's assistant, believed that his maternity patients in Pest were healthier than those in Vienna because of his extensive use of purges. He believed that childbed fever was caused by uncleanliness of the bowel. At the opening of his clinic each October he regularly directed a stinging oration against Vienna and claimed that the great mortality rate in Vienna was due to their negligence in the administration of purgatives. *[137]* However, as soon as medicine in Pest became anatomically oriented, the purgatives lost their prophylactic powers. At a time when I was not yet a member, the College of Medical Professors in Pest was repeatedly obliged to close the clinic during the school year because childbed fever became so overwhelming. I cannot provide figures, because the records were lost in the [1848] revolution. I live in the city about which I speak, and this is sufficient guarantee that I speak the truth.

The great mortality in the maternity hospitals is not depen-

11. Ede Flórián Birly (1787–1854) preceded Semmelweis as Professor of Theoretical and Practical Obstetrics at the University of Pest. He never accepted Semmelweis's opinions although he and Semmelweis worked together closely for four years.

dent on atmospheric influences, but rather on decaying matter that is regularly transmitted to patients from external sources. This follows from the fact that when circumstances are changed so that decaying matter is less frequently transmitted, mortality declines. . . . If the spread of decaying matter is completely halted, epidemic childbed fever is also halted. An example is the maternity ward in St. Rochus Hospital in Pest. It was separated from the surgical division and placed under my direction. *[138]* For six years I had no epidemic, even without chlorine washing. . . . If measures are adopted to destroy decaying matter, the so-called epidemics of childbed fever cease, even though they may have been raging for years. Examples are the first clinic in Vienna and the obstetrical clinic in Pest. Foreign experience supporting this will be discussed later.[12]. . .

[139] The mortality for the two clinics in the eight years from 1833 until 1841, during which male and female students were divided equally between both clinics, is shown in Table 11. I regret that I am late in obtaining the facts reported in this table and that I could not include this information where I first needed it. The reader may want to reread [pages 94 and 95 above; German edition,] pages 63 and 64.[13] . . .

In the years following adoption of the chlorine washings, the mortality rates in the two clinics are as given in Table 12.

[140] Even I have not achieved the smallest possible mortality rate. This will be discussed in the appropriate place below.[14] Evaluation of the increased mortality [in the years after I left the clinic] will be delayed until we discuss the failure of chlorine washings as attempted by other obstetricians.[15] *[141]* It is sufficient here to note that all the physicians officially functioning at this time in both clinics were and are opposed to my opinion regarding the origin of childbed fever. My successor, Carl Braun, wrote against my opinion. Carl Braun's successor, his brother

12. In discussing reaction to his work Semmelweis mentions several foreign cases in which adoption of chlorine washing reduced mortality. See below, pp. 174f., 186, 188–90; German edition, pp. 283–88, 307, 310–13.

13. See above, chap. 1, footnote 17.

14. See below, pp. 163f.; German edition, pp. 266f.

15. See below, pp. 193–95; German edition, pp. 330–32.

[139] TABLE 11

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1833	3,737	197	5.29	353	8	2.26
1834	2,657	205	7.71	1,744	150	8.60
1835	2,573	143	5.55	1,682	84	4.99
1836	2,677	200	7.47	1,670	131	7.84
1837	2,765	251	9.09	1,784	124	6.99
1838	2,987	91	3.04	1,779	88	4.94
1839	2,781	151	5.04	2,010	91	4.05
1840	2,889	267	9.05	2,073	55	2.06
Total	23,066	1,505		13,095	731	
Avg.			6.56			5.58

[140] TABLE 12

	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
1847	3,490	176	5.00	3,306	32	0.09
1848	3,556	45	1.27	3,319	43	1.33
1849	3,858	103	2.06	3,371	87	2.05
1850	3,745	74	1.09	3,261	54	1.06
1851	4,194	75	1.07	3,395	121	3.05
1852	4,471	181	4.00	3,360	192	5.07
1853	4,221	94	2.02	3,480	67	1.09
1854	4,393	400	9.10	3,396	210	6.18
1855	3,659	198	5.41	2,938	174	5.92
1856	3,925	156	3.97	3,070	125	4.07
1857	4,220	124	2.92	3,795	83	2.18
1858	4,203	86	2.04	4,179	60	1.43
Total	47,935	1,712		40,770	1,248	
Avg.			3.57			3.06

Gustav, has disclosed his opinions by 400 deaths in 1854. In the seventy-five years of the maternity hospital even the combined deaths of both clinics exceeded this figure only three times—in 1842 with 730 deaths, in 1843 with 457 deaths, and in 1846 with 567 deaths. . . .

To provide a clear overview of the health of the patients cared

[142] TABLE 13

	Before Separation of Clinics		
	Births	Deaths	Rate
Before pathological anatomy	71,395	897	1.25
After pathological anatomy	28,429	1,509	5.30

	After Separation of Clinics					
	First Clinic			Second Clinic		
	Births	Deaths	Rate	Births	Deaths	Rate
Male and female students Equally divided between clinics	23,059	1,505	6.56	13,097	731	5.58
Students divided by sex, before chlorine washings	20,042	1,989	9.92	17,791	691	3.38
After chlorine washings	47,938	1,712	3.57	40,770	1,248	3.06
Total	91,043	5,206		71,656	2,670	
Avg.			5.71			3.72

	Births	Deaths	Rate
Total for all seventy-five years	262,523	10,282	
Avg. for all seventy-five years			3.91

for in the Viennese maternity hospital, in Table 13 I show the most important figures for the crucial time periods.

[142–45] Table 13 must persuade every disinterested person that deaths in the Viennese maternity hospital were dependent on decaying animal-organic matter. This table shows clearly that variations in mortality were determined by the frequency with which decaying matter was conveyed from external sources. Since the laws of nature are the same throughout the entire world, I conclude that atmospheric-cosmic-terrestrial influences are never capable of inducing childbed fever. The endless series of epidemics reported in medical literature consists of cases of preventable infection from without; in other words, all these cases of the disease originate through the introduction of decaying animal-organic matter.

So-called epidemics in the maternity hospitals are not due to atmospheric influences but rather to decaying animal-organic matter. This is proven by the healthier condition of patients in countries where English opinions dominate, as in Ireland and Scotland. There is no basis for assuming that the atmospheric influences that supposedly destroy patients in German and French hospitals do not also exist in England, Scotland, and Ireland. *[146]* Thus atmospheric influences cannot explain the difference in health of the patients in these lands. However, English physicians are significantly different from the French and Germans in their opinions regarding the origin of childbed fever. The English regard childbed fever as contagious; in France and Germany, childbed fever is not believed to be contagious. I do not believe that childbed fever is contagious; however, healthy patients can be infected by decaying matter that is generated in diseased patients. Childbed fever is not conveyed from all diseased maternity patients, only from those who generate decaying matter. After death, childbed fever can be conveyed from every corpse to a healthy patient providing that the corpse has decayed sufficiently.

The English, proceeding from the opinion that childbed fever is contagious, will not visit a healthy maternity patient if they have earlier visited one who is ill unless they first wash with chlorine and change their clothing. If the number of diseased patients increases, they travel or give up their practice for a time. After an autopsy of a puerperal corpse, English physicians will not visit healthy maternity patients without first washing in chlorine and changing their clothing. *[147]* In those cases where ill patients do not generate decaying matter, English physicians take superfluous steps. But after treating ill patients who do generate such matter, English physicians, seeking to destroy a contagium, destroy that which, if conveyed to healthy patients, would cause childbed fever. . . .

In a significant essay Chiari claimed that in many cases decaying matter can be drawn from these sources.

[148] I call attention to a condition that, although frequently discussed, nevertheless requires more explanation. This is the origin and prevention of so-called puerperal epidemics. I say so-called because it has been established that this disease does not occur in many cases simultaneously over a large district, but usually only in institutions for delivery, and also not equally in different divisions of the same institution. I will not return to the different opinions regarding the causal origin of this really horrible disease. However, I will offer a few observations regarding the cause of many cases of the disease among maternity patients. These observations were made in connection with my responsibilities in Prague.

Because of an unusually thick cervix, a first delivery was protracted from 23 through 27 January 1853. Gangrene ensued. After useless baths, douches, antiphlogistic measures, and incisions in the swollen, cartilaginous and finger thick cervix, the dead fetus was cut in order to reduce its size and to end the four-day birth process. During the last two days, secretions from the vagina were brownish and very bad smelling. The patient became seriously ill with septic endometritis and died on 1 February. While this patient was in the delivery room, nine other patients became ill; all but one died. They had all been in the delivery room at the same time. *[149]* From the end of January many cases of illness followed until, from May through October, the health of the patients improved.

I became convinced that in this concrete case the cause of the frequent illnesses was the spread of gangrenous matter from the diseased patient to healthy ones. Naturally, all possible caution was observed in order to avoid spreading harmful matter through examinations. Nevertheless, the simultaneous residence of one ill patient and of several healthy ones in the same relatively small locality makes it possible for harmful matter to be spread in various ways. Given that several persons became ill, it is conceivable that in an institution where space is inadequate for numerous births the disease may be propagated in this way. I do not wish to imply that all so-called puerperal epidemics must arise in this way. However, I wish to call attention to a condition that may and will frequently occur in large institutions for delivery.

In confirmation of my opinion I had a second tragic experience. In October 1853, a few days before my return to Prague after a vacation, it became necessary to perform surgery on a woman who had been in labor for several days because of a narrow pelvis. This patient died of septic endometritis with inflamed synchondrosis [certain cartilaginous joints, in this case probably those located in the pubic region (symphysis pubica)]. From then on several serious cases of disease were encountered; they ceased only in the middle of November. From then until the end of my stay in Prague, namely until the end of August of the next year, I was fortunate enough not to observe more cases of this horrible disease.

[150] By these observations I intend to show only that by careful attention one is sometimes able to establish the mode of origin of numerous cases of disease in maternity hospitals. Moreover, this method of origin was already identified by Semmelweis. Also this autumn, in the clinic for midwives, a similar observation was made and privately communicated to me by my friend Dr. [Joseph] Späth.[16]

I regard it a matter of conscience to publish these observations. For even though I have not said that this is the only way in which this plague can originate, nevertheless, these retrospective observations can have great practical significance in the organization of maternity institutions. It is imperative that in larger maternity institutions several delivery rooms be held ready in order that cases of protracted delivery can be isolated from ordinary deliveries. It is self-evident that this isolation is necessary also in the organization of instruction. In consequence of my opinion that the spread of maternity diseases in large institutions depends on transferrence of foul harmful matter, I sought opportunities to avoid this cause. I employed the following preventive measures: first, I divided instruction so that individual patients were never examined by more than five students, after which every participant was required to wash his hands with chloride of lime. *[151]*

16. Joseph Späth (1823–96) was Professor of Obstetrics in Vienna and from 1873 until 1886 he was director of the Second Obstetrical Clinic. Semmelweis regarded him as a principal opponent. By 1864, however, he had adopted Semmelweis's view, and observed that virtually all obstetricians were convinced that Semmelweis was correct, although few admitted it openly. Frank P. Murphy, "Ignaz Philipp Semmelweis (1818–65): An Annotated Bibliography," *Bulletin of the History of Medicine* 20(1946), 653–707: 669f.

Second, so that students could not easily come to the clinic from anatomical work, I conducted the clinic both summer and winter in the morning hours between seven and nine. Third, I watched very closely that the laundry was cleaned conscientiously. Because of the second epidemic, vulvar compresses were washed outside the hospital. Fourth, it was easily comprehendible, for example, that by sponging the ulcerated genitals of a patient, this condition could be transmitted to other patients. Thus I adopted the policy of cleansing the genitals of patients only by sprays rather than by sponges. Although sponges do come into contact with the genitals, it is much less likely that sprays will. Fifth, I sought to remove the seriously ill from the maternity wards by transferring them into the general hospital. This measure was also necessary because space was limited. Everyone must see that it is very much to the purpose, from both physical and moral considerations, to prevent the accumulation of such diseased patients in the maternity institution. Sixth, from the opinions expressed above, it also followed that when many people became diseased in a maternity institution, it became necessary to change location. Even the furnishings of a hospital could provide the means by which disease was spread.

It therefore appeared advisable that new institutions should be so constructed that, for example, each obstetrical clinic would be located in its own building and, even in respect to the laundry, completely isolated from other clinics. *[152]* Since as a result of utilizing these measures insofar as possible frequent cases of puerperal disease ceased after one or two months, I believe myself able to recommend them most enthusiastically.[17]

From Chiari's observations the reader sees that many cases of disease can be caused by the decaying matter generated by one diseased patient. Decaying matter generated by patients is not the only source of so-called puerperal epidemics, however. This follows from what I have said about the sources of this matter.

Patients are healthier in maternity hospitals where it is believed that childbed fever is contagious and where steps are taken to destroy the contagion. This follows from a report published by Professor [Karl Edouard Marius] Levy of Copenhagen. The

17. [Baptist Johann] Chiari, "Winke zur Vorbeugung der Puerperal-Epidemie," *Wochenblatt der Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* [11] (1855): [117–21; author's note. Semmelweis quotes the entire article.]

report concerns the maternity hospitals and the teaching of practical obstetrics in London and Dublin. Professor Michaelis in Kiel has translated the report. I cannot resist reproducing the introductory remarks of the translator.

[153] On a recent trip I had the opportunity to convince myself of the truth of the present report. The industriousness and thoroughness of the report must also convince every reader.

The author's main object is to explore the conditions under which puerperal fever appears and the techniques that have been successfully used to control it. English institutions offer the most important results in this matter, for whereas they were formerly devastated by this plague, mortality in London and Dublin institutions has been reduced to just over 1 percent through the adoption of health measures in the last decades. Unfortunately, on the continent we are still a long way from such favorable results. . . . With the advance of education and of human sensitivity [*Humanität*] public opinion is being forcefully aroused—even in places where formerly there was indifference to this horrible offering of human life. The goal is clear: one must make the institutions healthy, or close them. . . .

[154] For those with experience in the matter, no proof is required for the assertion that this plague will not be extinguished through therapeutic treatment. Thorough and strictly observed measures of cleanliness, ventilation, etc. offer more hope. Indeed, according to the experience of the British, this approach will certainly lead to success. . . .

Fortunately, in Vienna last year it was discovered that the disease could be reduced significantly by cleaning the hands with chlorine before examining. With time, use of this remedy reduced the number of deaths to nearly one-tenth the normal rate; an obviously striking result. We owe this discovery to Dr. Semmelweis, who will certainly soon publish the details. Apparently, the use of this remedy, together with universal disinfection, will usher in more fortunate times for our maternity hospitals. *[155]* I must publicly thank Dr. [Heinrich] Hermann Schwartz of Holstein for bringing the Viennese experience to my attention.—Kiel, 17 April 1848.[18] . . .

18. The original essay by Karl Edouard Marius Levy appeared in *Bibliothek for Laeger*, the German translation [entitled "Gebärhäuser und der praktischen Unterricht in der Geburtshülfe"] by [Gustav Adolf] Michaelis was published in *Neue Zeitschrift für Geburtskunde* 27 [(1850):, 392–449; author's note].

[Next come six pages of tables showing that three British maternity hospitals had mortality rates usually ranging between .5 and 2 percent. Semmelweis observes that a midwife, Mrs. Widgen, attributed a higher mortality rate of 4.93 percent in one hospital to an autopsy that was performed in the hospital that year. Next, there is a table of annual mortality rates for another British hospital—in this case mortality averaged over 3 percent over several years, and then, through 1844, 1845, and 1846, it was reduced to zero. Semmelweis quotes Levy's discussion of this change.]

[156–61] . . . In the immediate neighborhood of the building, not 30 feet from the wall, an open pit was found that was more than 1,500 feet in area. This pit received the refuge from the adjacent poor and densely built part of the city. The contents of the pit were stagnant, and boiled constantly through the production of gas. In 1838 after numerous difficulties and debates with the water commission, the hospital administration finally succeeded in having a 644-foot stretch of the pit cleaned and covered. In the process, however, one mistake occurred—instead of carrying away an incredible hulk of black, stinking slime, it was simply spread nearby on the ground. This, of course, greatly increased the surface of evaporation. Within twenty-four hours two cases of puerperal fever occurred in the hospital; Dr. [Edward] Rigby believed that they could be traced directly to this irresponsible procedure. . . . *[162]* [In 1838, while all this was going on, the hospital had a mortality rate of 26.76 percent. Then, while the mortality rate remained high, a new ventilation system was installed. The mortality rate declined significantly, and some of the physicians were inclined to attribute the decline to this new system. However, Levy notes, doubts remained because] now and then a stinking mass of liquid had been observed to surface in the cellar near the hearth of the smokestack. Investigation disclosed that the water was coming from the drainage system. The building's drains were examined. The main drain was found to be so firmly blocked by some pieces of wood that it seemed to have been done intentionally. The whole surrounding area of the cellar was abundantly strewn and saturated with filth and it was impossible to determine how long this condition had existed.[19]

[Semmelweis quotes more of Levy's discussion of whether the

19. Ibid., pp. 418–20.

decline in mortality was due to a change in the ventilation system or to the correction of the unsanitary conditions. Semmelweis remarks that given either possibility, the disease was not due to atmospheric-cosmic-terrestrial influences. Semmelweis reproduces several more of Levy's tables showing favorable mortality rates for British, Irish, and Scottish hospitals. Semmelweis then observes] *[163–68]* . . . We have examined eight maternity hospitals throughout three countries. In seven of these, mortality just exceeded 1 percent; in the eighth it was 3 percent. We do not find the causal moment for this larger mortality in atmospheric influences but rather in the harmful matter of the drainage canals that surrounded the building.

Why should atmospheric influences so strikingly spare maternity hospitals in the United Kingdom while so many patients in German and French hospitals are sacrificed? The answer is that there are no atmospheric influences to which German and French patients are falling victim; rather decaying animal-organic matter is being brought to the individuals from external sources. This causes the mortality in the hospitals of the United Kingdom and of France and Germany. Because of the circumstances of the French and German hospitals, decaying matter is much more frequently transmitted to patients in those hospitals, and they have a greater mortality rate. *[169]* In the United Kingdom, individuals are much less often exposed to decaying matter from without, thus their mortality rates are much lower. The English regard childbed fever as contagious. Thus they employ chlorine washings and destroy the decaying matter taken from ill patients and from corpses. In German and French maternity hospitals this matter is not destroyed. As Chiari has shown, this can cause many cases of illness. In German and French hospitals decaying matter is frequently taken from corpses and from ill persons who are unaffected by childbed fever. As a rule German and French maternity hospitals are associated with large general hospitals. Therefore their students occupy themselves in morgues, and in medical and surgical wards, as well as in maternity wards. In this way they become carriers of the decaying matter that initiates so much misfortune. Maternity hospitals in the United Kingdom are independent institutions; because they are re-

moved from general hospitals, the students are forced to concern themselves exclusively with obstetrics.

Some may attribute the favorable health of the London maternity hospital to the fact that there are never more than two students training at one time. But I must observe that students do not constitute atmospheric influences and that childbed fever caused by students with contaminated hands is not, therefore, epidemic childbed fever. *[170]* It clearly matters whether many or just a few students with contaminated hands examine patients, but it does not matter whether many or a few students with clean hands examine patients. This is proven by the Dublin maternity hospital; Levy writes: ". . . A practical school has been established where, in the course of time, several thousand young physicians from all parts of England have sought a practical education in obstetrics. This finally proves that it is only cowardly superstition to discount the needs of education and science and to assert that a horrible death rate is the unavoidable result of large maternity institutions."[20] This is also proven by the first clinic in Vienna where, in April 1847, without chlorine washing and with only twenty students, 57 of 312 patients died (18.27 percent), while in 1848, with chlorine washings and with forty-two students, 45 of 3,556 died (1.27 percent). In order to clarify the difference in mortality rates between maternity hospitals in which individuals contaminate themselves with decaying matter at different rates, in Table 14 we will compare the reports of the Dublin and Viennese maternity hospitals for a period of sixty-six years. Both are institutions for the education of physicians.

[172–78] . . . The great mortality in the maternity hospitals is not dependent on atmospheric influences, since delivering women among the general population do not simultaneously suffer from childbed fever. The maternity hospital and the surrounding area must be subject to the same atmospheric influences. *[179]* However, while patients in the hospital are decimated, other women enjoy good health. This is proven by the measure of closing the maternity hospitals; after a hospital is closed, births do not cease,

20. Ibid., p. 449

but women give birth in various locations rather than being admitted to a hospital. Those who deliver remain healthy; in nearby maternity hospitals they would be killed by atmospheric influences. Certainly many women giving birth die outside maternity hospitals. But these deaths cannot be attributed to atmospheric influences. This is true, since increased mortality outside the hospitals is not always simultaneous with that in the hospitals. The mortality in the hospitals is often high while it is low outside, and high mortality outside the hospitals is very rarely observed.

Childbed fever outside the maternity hospitals—exactly like that which rages inside—is, without exception, a resorption fever dependent on the resorption of decaying animal-organic matter. In exceptional cases, both inside and outside the maternity hospitals, the decaying matter is created within the attacked individual and childbed fever originates through self-infection. In the overwhelming majority of cases, however, the decaying matter is transmitted to the individual from external sources; childbed fever then originates as external infection, whether in or out of the hospitals. *[180]* Both inside and outside maternity hospitals, physicians who practice obstetrics perform autopsies and treat patients who are generating decaying matter. . . . Midwives cleanse patients who are producing decaying matter. . . . *[181]* . . . However, private physicians have fewer opportunities to contaminate their hands, so childbed fever is less common outside maternity hospitals. . . . The busy physician may only care for a few obstetrical cases each day. In the Viennese maternity hospital, there are often thirty or forty births within twenty-four hours. Therefore, a private physician whose hands are contaminated cannot cause as many cases of childbed fever as can a physician in a large maternity hospital. Moreover, outside the hospital, individuals are generally examined by only one physician; in the hospital, a person may be examined by many. Although one unclean hand is sufficient to cause many deaths, with many examining hands it is more likely that one hand or another will be contaminated.

Arneth has published a very informative collection of experi-

[171–78] TABLE 14

	Dublin Maternity Hospital			Viennese Maternity Hospital		
	Births	Deaths	Rate	Births	Deaths	Rate
				BEFORE SEPARATION OF CLINICS		
				Before Pathological Anatomy		
1784	1,261	11	0.87	284	6	2.11
1785	1,292	8	0.61	899	13	1.44
1786	1,351	8	0.59	1,151	5	0.43
1787	1,347	10	0.74	1,407	5	0.35
1788	1,469	23	1.56	1,425	5	0.35
1789	1,435	25	1.74	1,246	7	0.56
1790	1,546	12	0.77	1,326	10	0.75
1791	1,602	25	1.56	1,395	8	0.57
1792	1,631	10	0.61	1,579	14	0.89
1793	1,747	19	1.08	1,684	44	2.61
1794	1,543	20	1.29	1,768	7	0.39
1795	1,503	7	0.46	1,798	38	2.11
1796	1,621	10	0.61	1,904	22	1.16
1797	1,712	13	0.75	2,012	5	0.24
1798	1,604	8	0.49	2,046	5	0.24
1799	1,537	10	0.65	2,067	20	0.96
1800	1,837	18	0.97	2,070	41	1.98
1801	1,725	30	1.74	2,106	17	0.80
1802	1,985	26	1.30	2,346	9	0.38
1803	2,028	44	2.16	2,215	16	0.72
1804	1,915	16	0.83	2,022	8	0.39
1805	2,220	12	0.54	2,112	9	0.40
1806	2,406	23	0.95	1,875	13	0.73
1807	2,511	12	0.47	925	6	0.64
1808	2,665	13	0.48	855	7	0.81
1809	2,889	21	0.72	912	13	1.42
1810	2,854	29	1.01	744	6	0.80
1811	2,561	24	0.98	1,050	20	1.90
1812	2,676	43	1.69	1,419	9	0.63
1813	2,484	62	2.49	1,945	21	1.08
1814	2,508	25	0.99	2,062	66	3.20
1815	3,075	17	0.51	2,591	19	0.73
1816	3,314	18	0.54	2,410	12	0.49
1817	3,473	32	0.92	2,735	25	0.91
1818	3,539	56	1.58	2,568	56	2.18
1819	3,197	94	2.94	3,089	154	4.98
1820	2,458	70	2.84	2,998	75	2.50
1821	2,849	22	0.77	3,294	55	1.66
1822	2,675	12	0.44	3,066	26	0.84

[171–78] TABLE 14 (continued)

	Dublin Maternity Hospital			Viennese Maternity Hospital		
	Births	Deaths	Rate	Births	Deaths	Rate
				After Pathological Anatomy		
1823	2,584	59	2.28	2,872	214	7.45
1824	2,446	20	0.81	2,911	144	4.98
1825	2,740	26	0.64	2,594	229	8.82
1826	2,440	81	3.33	2,359	192	8.12
1827	2,550	33	1.29	2,367	51	2.15
1828	2,856	43	1.50	2,833	101	3.56
1829	2,141	34	1.59	3,012	140	4.64
1830	2,288	12	0.52	2,797	111	3.97
1831	2,176	12	0.55	3,353	222	6.62
1832	2,242	12	0.58	3,331	105	3.15
				AFTER SEPARATION OF CLINICS		
				Males and Females in Both		
1833	2,138	12	0.56	3,737	197	5.29
1834	2,024	34	1.67	2,657	205	7.71
1835	1,902	34	1.78	2,573	143	5.55
1836	1,810	36	1.98	2,677	200	7.47
1837	1,833	24	1.30	2,765	251	9.09
1838	2,126	45	2.11	2,987	91	3.04
1839	1,951	25	1.23	2,781	151	5.04
1840	1,521	26	1.70	2,889	267	9.05
				Males in First Clinic Only		
1841	2,003	23	1.14	3,036	237	7.07
1842	2,171	21	0.96	3,287	518	15.08
1843	2,210	22	0.99	3,060	274	8.09
1844	2,288	14	0.61	3,157	260	8.02
1845	1,411	35	2.48	3,492	241	6.08
1846	2,025	17	0.83	4,010	459	11.04
				Chlorine Washings Used in Physicians' Clinic		
1847	1,703	47	2.75	3,490	176	5.00
1848	1,816	35	1.92	3,556	45	1.27
1849	2,063	38	1.84	3,858	103	2.06
Total	141,903	1,758		153,841	6,224	
Avg.			1.21			4.04

ences of the English regarding the origin of childbed fever outside the hospital.

[182] Puerperal fever is such a frightful disease that we must be interested in what English physicians think of the disease in general, and especially how they treat patients, and what they think about its most mysterious aspect—its etiology.

During a non-epidemic period, the experienced [John] Roberton classified women according to the frequency with which they were attacked by puerperal fever. According to his experience, those who care for a household are visited by the disease much less frequently than those who are served. In the industrial city of Hulme, only a small number of the 40,000 inhabitants retain servants. The far greater number of the female inhabitants are working women who arise at five in the morning, send the older children to work, and either accompany their husbands to the factories or are busy from early in the morning until late at night conducting the household and caring for children. Throughout pregnancy, even as the first phrase of delivery approaches and is endured, such women must continue the same activities until serious labor pains force them to stop. In spite of such deprivations, official records for the decade 1839–49 indicate that only 1 of 196.5 deaths was due to childbed fever. In four small neighboring villages whose inhabitants were much more leisured, 1 of 84 deaths was due to this cause. *[183]* Of course the situation is quite different during an epidemic; crowded together in small rooms with numerous other local inhabitants, poor maternity patients frequently die. The prosperous, living in greater comfort and cleanliness and with conscientious care, enjoy much greater hope. In Roberton's opinion the unhealthy aspects of both social classes apparently unite in the wives of shopkeepers and small merchants. These pass their days in poorly constructed houses and in spite of their better training, coddling, and amusements are not able to enjoy the advantages of the more prosperous classes.

We will investigate numerous experiences in England which suggest that puerperal fever can be induced in delivering women by the transmission of gangrenous foul matter in general, and of particles from corpses in particular. However, as we will see, these cases have usually been interpreted quite differently. Among the various articles and editorials that have appeared in England, none showed more insight than a journal article by Robert Storrs. Storrs

asked various colleagues for their experiences and opinions. The results of his queries included the following: [G.] Reedal in Sheffield treated a young man who suffered and finally died from an open tumor and an erysipelatous infection.[21] *[184]* . . . While Reedal cared for the patient, five women whose deliveries he attended between 26 October and 3 November 1843 contracted puerperal fever and died. He visited these unfortunate women almost immediately after attending to the wounds of the former patient. Two other women, at whose deliveries he also assisted, did not become ill, but he did not visit them until a few hours after the dangerous call. After these deaths Reedal ceased visiting the young man, because he recognized that he himself was the carrier of the disease. Since then he has had no more cases of puerperal fever than he had before treating the case of erysipelas. Mr. [R. P.] Sleight of Hull records that while treating a patient for erysipelas, he was called to attend a delivery. The delivery proceeded normally; nevertheless, within twenty hours the woman succumbed to puerperal fever, and she died eighteen hours later. [Semmelweis, still quoting Arneth, relates several more similar stories.]

[185–87] . . . Storrs hopes to have proved: *[188]* (i) that puerperal fever is communicable through physical contact, (ii) that puerperal fever derives from an animal poison and, in particular, from erysipelas and its sequelae, and occasionally from typhoid, (iii) that without differences in the circumstances of the patient, childbed fever brings about erysipelas, typhoid, and in men a fever that sometimes is remarkably similar to puerperal fever, and (iv) that the most rapid, conscientious, and informed treatment remains without success. Storrs recommends that to avoid mishaps, obstetricians should never visit persons in labor while wearing the same clothing formerly worn when attending other patients. This concerns especially the outer clothing that usually carries the matter which generates the disease. Once erysipelas or typhoid is encountered, the same measures must be adopted for all maternity patients. After autopsy or an operation on a person suffering from erysipelas or typhoid, the surgeon must always

21. Erysipelas is a contagious disease of the skin and subcutaneous tissues now known to be an infection by a particular streptococcus. It was particularly common following surgery and often accompanied the other symptoms of puerperal fever. The British regarded it as related to puerperal fever and, therefore, as particularly dangerous to puerperae.

wash as carefully as possible and change his clothing completely before attending a delivery. One must not disregard gloves because the hands and arms are the parts of the body most likely to convey the poison. *[189]* Once the disease is established in a physician's practice, he should leave his residence for two or three weeks, change his entire wardrobe, wash everything conscientiously, and avoid every case of illness that could serve as a source of the animal poison.

A similar communication from Roberton generated a great sensation in England. A midwife who had a considerable practice among beneficiaries of a particular charitable society had the misfortune to be present when a woman she delivered died of puerperal fever. In the following month, December 1830, she attended deliveries of 30 women from different parts of the city, and 16 died of childbed fever. This circumstance was particularly striking because 380 other deliveries were performed successfully at the same time by other midwives from the same organization. The physicians of the institution persuaded the midwife to discontinue her practice for a time and to go into the country. Within a short time puerperal fever was encountered in the practices of other midwives and physicians at various points throughout the city. By June the disease raged with a distribution and fury that had never been known in Manchester. . . .

[190–91] . . . In contrast to Semmelweis and Skoda, the English do not conclude that putrid matter has been conveyed to the sexual organs of the women. They conclude that the disease *qua talis* is conveyed from one woman to another. Thus [Fleetwood] Churchill remarks, "After carefully examining the facts, I cannot doubt that the disease spreads through infection and contact, i.e., that from one person suffering from puerperal fever it can be conveyed to another whom the first touches or has as a neighbor."

Deciding which interpretation is correct may be of great practical importance. Given the opinion common in England, one need not proscribe contacting corpses of persons who die from diseases other than puerperal fever. On the other hand, given the assumption that a healthy person cannot convey the disease without having touched an ill one, one can feeely go from an ill maternity patient to others without changing clothing as is required in England. The English assume that it is possible to convey the disease for a fairly long time; thus they frequently recommend

that a physician who encounters numerous cases of puerperal fever should temporarily give up assisting at births and should change his entire wardrobe. *[192]* This is justified by citing occurrences where numerous cases of puerperal fever arise in the practice of one physician or midwife while other physicians have no similar cases. This circumstance can be explained more easily if one assumes that these physicians have been involved either with autopsies or with other putrescent substances, the opening of abscesses, cleaning and binding of wounds, cleaning or examining maternity patients, extracting placentas, etc. Many physicians have temporarily interrupted their obstetrical practices after losing several women to puerperal fever. Upon resuming their practice after a period of several weeks they were not more fortunate; this proves that the cause they identify is not responsible.[22]

[193] I am also convinced that the activities of physicians cause puerperal fever. I include this information to demonstrate that activities outside the maternity hospital can also cause childbed fever. However, I draw conclusions different from those drawn by English physicians.

I regard childbed fever as a non-contagious disease because it cannot be conveyed from every patient with childbed fever to a healthy person, and because a healthy person can contract the disease from persons not suffering from childbed fever. Every victim of smallpox is capable of giving smallpox to healthy people. A healthy person can contract smallpox only from one who has smallpox; no one has ever contracted smallpox from a person suffering from cancer of the uterus. This is not the case with childbed fever. If childbed fever takes a form in which no decaying matter is produced, then it cannot be communicated to a healthy person. However, given a form that produces decaying matter, as for example septic endometritis, the disease is certainly communicable. . . . Moreover, childbed fever may come from diseased states other than childbed fever, for example from gangrenous erysipelas, carcinoma of the uterus, etc. *[194]* . . . A contagious disease is conveyed by the matter which is produced only by that particular disease. . . . Puerperal fever is

22. Arneth, [op. cit., note 3 above], pp. 334–44 [author's note].

conveyed by matter that is the product not only of childbed fever but also of the most heterogeneous diseases. Every corpse, no matter what the cause of death, produces matter that can cause childbed fever. It follows that one must avoid contacting corpses or diseased patients who produce decaying matter, whether or not they are puerperae. A veterinarian who is simultaneously an obstetrician could draw from diseased or dead animals decaying matter that would cause childbed fever in maternity patients.

Thus childbed fever is not a contagious disease, but it is a disease that can be conveyed to a healthy person by means of decaying matter. Childbed fever bears the same relation to erysipelas and its sequelae that it does to every other disease that generates decaying matter. Childbed fever stands in the same relation to erysipelas and its sequelae that it does to every decomposing corpse. In recognizing only erysipelas and its sequelae, beyond puerperal fever itself, as sources of childbed fever, English physicians draw the boundaries much too narrowly. This is shown by the cases previously discussed.

[195] Thus childbed fever is the same disease that occurs among surgeons and anatomists, and following surgical operations, it is the same disease whether decaying matter is brought into the circulation system of males or of females. The decaying matter is not resorbed through the epidermis or through a thick layer of epithelium; in surgeons and anatomists resorption must be preceded by a wound. As a qualified pathological anatomist, Kolletschka had his hands contaminated with decaying matter on countless occasions and he nevertheless remained healthy. Through a prick, resorption was made possible; we know which disease was the consequence. The site of resorption can be any point on the body that is stripped of epidermis and epithelium. The inner uterine surface of maternity patients has neither epidermis nor epithelium; this is the site of resorption of the decaying matter that causes childbed fever. If injuries occur during birth, any part of the genitals, indeed any wounded part of the whole body, can be the site of resorption. In the school year 1857–58 at the obstetrical clinic in Pest, the outer genitals of two maternity patients became gangrenous. One of the students assigned to care for these patients had a small abrasion on one

finger from a needle prick. She contracted lymphangitis with suppuration of the auxiliary glands and was seriously ill for several months.

[196] For patients in the maternity hospitals the genital region is generally the only area suitable for resorption; thus, in order to contract childbed fever, it is necessary that decaying matter be transmitted to the genital area. Since the clothing of the obstetrician does not contact the genitals, the English custom of changing clothes is a harmless but superfluous measure. In 1848 in Vienna, the students and I did not change clothing after activities with objects suitable to induce childbed fever. We only exposed our hands to the operation of chlorine, and in 1848 of 3,556 patients we lost only 45 (1.27 percent) to childbed fever. In cases cited above, an obstetrician visited healthy patients without having changed clothing and his patients died of childbed fever. It was not the clothing but the hands that carried decaying matter. Since hands cannot be changed, they must be disinfected. If clothing became contaminated with decaying matter, the hands were certainly still more contaminated, and it is with the hands that one makes internal examinations.

In order for childbed fever to occur, it is a *conditio sine qua non* that decaying matter is introduced into the genitals. With the exception of internal obstetrical examinations, an individual can carry out every possible medical examination with contaminated hands without the slightest danger. The epidermis prevents the resorption of decaying matter. Obstetricians carry the matter on their hands for hours and even days without harming themselves. *[197]* If this matter is brought into contact with the inner surface of the uterus, even for a moment, it is resorbed and childbed fever results. The hands of the anatomist are often in contact with decaying corpses for hours at a time and he remains healthy. If the epidermis is removed by an injury, however, the disease is generated—this happened to Kolletschka and to the student midwife.

Because of the arrangement of the rooms in the first clinic, general rounds were made twice daily in the following order: first was the visit to the labor room, where half the healthy patients were examined; then the diseased patients were examined;

and finally the remaining half of the healthy patients were visited. Suppose in visiting the diseased patients we contaminated our hands. If, without having washed in chlorine, we had then taken the pulses of healthy patients, if we had felt their abdomens externally, in a word, if we had made all the necessary medical examinations with the exception of internal obstetrical examinations, we would not have propagated childbed fever. Childbed fever cannot be induced through external unwounded surfaces of the body. Therefore, it is not spread as is smallpox when the outer surface of a healthy individual comes within breathing distance of a diseased person. Of course, if the exhalations of a diseased individual enter the uterus, then the disease is certainly spread.

[198] We noted that changing clothing after visiting a diseased patient is harmless but superfluous. Clothing can cause childbed fever only when its exhalations enter the uterus with the air, and clothing is not easily contaminated to this degree. Clothing could also cause childbed fever if, for example, the cuff of one's jacket is contaminated with decaying matter and contacts the genitals during the birth. This, however, is not a common happening. In this sense clothing certainly can be harmful, but not in the way the English believe. They hold that puerperal contagium, like smallpox contagium, can be conveyed on clothing to a healthy patient and that it is then absorbed through the outer surfaces of the body, thereby causing childbed fever. . . . *[199]* . . . I have clearly indicated how my opinion concerning the nature and spread of childbed fever differs from the opinion of English physicians. . . .

It was once a mystery how an epidemic disease could also be brought about through trauma. Knowing now that puerperal fever comes about through resorption of decaying matter, this is no longer a mystery. After a difficult operation with forceps, certain parts of the genitals may be crushed and become necrotic. If these necrotic parts are resorbed, childbed fever is caused by self-infection.

The geographical distribution of childbed fever proves that it is not caused by atmospheric influences but rather by contact with external decaying matter. [Conrad Theodor Carl] Litzmann writes,

> *[200]* Most epidemics known to us are limited to central Europe. Notes about epidemics outside Europe are incomplete; they include observations on childbed fever from [Hugh Lennox] Hodge in Philadelphia and from Scholz in Jerusalem. Generally cold and moist lands appear especially afflicted; for example, England is more seriously afflicted than France. The same holds for cities on the banks of large rivers, for example, Vienna. On the other hand, according to Brydone's report, women in Sicily seldom become ill after delivery. Savary reports in his letters from Egypt that nursing diseases are entirely unknown there, and Dr. Salles reports that during his three-year visit to South America he saw no cases of childbed fever. Nevertheless, these reports are too incomplete to warrant conclusions. Probably childbed fever is spread over the entire world, and its frequency depends less on climate than on the presence or absence of large cities, in particular of large maternity institutions.[23]

Being convinced that childbed fever originates by the resorption of decaying matter, I interpret Litzmann's remarks as follows. Certainly in isolated cases of self-infection, childbed fever occurs throughout the world. Also, throughout the world there are diseased persons whose diseases generate decaying matter and who are treated by the same medical personnel who assist deliveries. Consequently, childbed fever occurs occasionally throughout the world because of infection. *[201]* Certainly childbed fever would be more frequent if individuals were more frequently infected with decaying organic matter. However, this happens only in central Europe where there is occasion for handling decaying matter and where there is opportunity for many persons in maternity hospitals to be infected. Childbed fever is particularly associated with large cities because large maternity hospitals are found there. The cities do not cause childbed fever, since childbed fever can be suppressed by closing the hospitals and by allowing women to be delivered in the cities themselves.

The puerperal epidemic in Vienna was not caused by Vienna's location on the banks of a large river, since for twenty-five years less than 1 percent of Viennese patients died of the disease. Moreover, the use of chlorine washings did not make the Dan-

23. C. T. Carl Litzmann, *Das Kindbettfieber in nosologischer, geschichlicher und therapeutischer Beziehung* (Halle: [Eduard Anton], 1844), p. 129 [author's note].

ube dry, but the epidemic ceased. If the Danube had caused the epidemic in Vienna, why would the epidemic appear in Vienna but not in all the other places along its banks? If childbed fever does not occur in Sicily, Egypt, and South America, it is certainly not because they lack water. Rather it is because in these areas pathological anatomy, the pride of the Viennese school and the scourge of the Viennese maternity hospital, is not yet dominant.

[202] Published reports of the English maternity hospitals indicate an average mortality of 1 percent; the French lose 4 percent. Thus Litzmann is simply wrong in claiming that England is more subject to childbed fever than France.

The history of childbed fever proves that it is not caused by atmospheric influences but rather by the spread of decaying matter. In his history of childbed fever, in which all the epidemics before 1841 are reported, Litzmann writes:

> Insofar as existing historical documents permit one to judge, childbed fever is a modern disease. The cases reported by Hippocrates that are generally identified as such do not belong to this classification. There are only examples of bilious fever, then common, which among maternity patients was no different from its appearance among non-maternity patients or men; Hippocrates himself never identified it as a separate and distinguishable disease. Pain in the right hypochondrium, bilious diarrhea and vomiting, headache with delirium or fainting, fever with more or less frequent irregular chilling constitute the symptoms common to all these cases. It is not noted, as a basis for differentiation, that among maternity patients there is a disruption in puerperal secretions. In only three cases do we find a suppression of lochia, and in two of these, pain in the area of the uterus is mentioned. In recent times [Theodor] Helm has shown that recurring chilling, from which several of the ill suffered, in connection with the disease in question, indicates metrophlebitis [inflammation of the veins of the uterus].
>
> *[203]* . . . We encounter the first, as yet unclear indication of childbed fever in the second half of the seventeenth century at the Hôtel-Dieu in Paris. [Phillipe] Peu relates that mortality among the newly delivered was very great and greater in certain seasons than others. The year 1664 was particularly devastating. This striking mortality was attributed to the fact that the maternity rooms were directly over the hall where the wounded were situated. The mortality of the maternity patients was in direct proportion to the number of wounded. In humid weather, warm or cold, the rate was higher; in dry weather it was variable. By changing the location of the maternity patients to the lower floor the disease was eliminated. The description is highly deficient and tells only that the patients were subject to hemorrhages and that their corpses were full of abscesses.[24]

Osiander gives us more precise disclosures regarding the Hôtel-Dieu and the prevalence of childbed fever there.

> In the noteworthy report of the hospitals of Paris which [Jacobus-Rene] Tenon prepared for the government in 1788, we read that the lower abdominal infection, *la fièvre puerpérale*, had raged every winter since 1774 among the maternity patients of the Hôtel-Dieu, and that often as many as 7 of every 12 patients suffered from it. *[204]* This is less surprising if one knows the deficient conditions then endured by patients in the Hôtel-Dieu. They were enclosed in the upper story in low-ceilinged, small halls overcrowded with beds. Three patients would often lie next to one another in a bed four feet wide. In 1786, 175 pregnant and newly delivered women and 16 attendants slept in sixty-seven beds of ordinary width. Moreover, the hall for maternity patients was directly over other sick rooms. Thus while the wounded were not, as formerly, directly under the maternity patients, one can certainly assume that the proximity of large halls for the diseased contributed to the corruption of the air and to the creation of a dangerous miasma in the halls for maternity patients.[25]

Thus, the first recognized epidemic of childbed fever was not caused by atmospheric influences but rather in the way that I have taught. Perhaps no historians can be found to reveal the mystery of the countless remaining puerperal epidemics. In any case, historians do record autopsy results from each respective epidemic; this reveals the source from which the epidemic drew its existence. *[205]* I have described how epidemics began at the

24. Ibid., pp. 130f.
25. Osiander, [op. cit., note 4 above,] p. 243 [author's note].

Viennese maternity hospital, at the maternity ward at St. Rochus, and at the obstetrical clinic at Pest. By using these descriptions as a guide, perhaps one can dispel the mystery surrounding other epidemics in maternity hospitals.

The history of epidemics at the Viennese maternity hospital proves that the frequency and virulence of epidemics are directly related to the development of the anatomical orientation in medicine. In 1789 Boër entered his teaching post and, in 1822, was succeeded by Professor Klein. Boër, the reformer of obstetrics, the author of [a textbook on] natural obstetrics, was discouraged because of what was then regarded as an enormous mortality rate, and he retired prematurely from his post. Yet in twenty-one years Boër lost less than 1 percent of his patients, in six years he lost 1 percent, in four years 2 percent, in one year 3 percent, and in one year 4 percent. How horribly has the anatomical orientation increased the mortality rate! Between 1822 and 1858, even including the years when chlorine washing was employed, the mortality rate was the following: one year 0 percent, three years 1 percent, six years 2 percent, four years 3 percent, six years 4 percent, four years 5 percent, three years 6 percent, four years 7 percent, five years 8 percent, and one year 12 percent. . . .

[206]. . .It is unimportant whether the cases cited by Hippocrates were childbed fever. Only a few cases were involved, and these could have occurred by self-infection. Alternatively, they may have been cases of infection from external sources; certainly even in Hippocrates' time there were sick people whose diseases generated decaying matter, and there were also male and female medical personnel who treated these diseased persons along with women who were pregnant and in labor. In this way external infection was possible. Boër says this of Hippocrates:

> One is filled with reverence and astonishment, when treating puerperal fever and opening corpses of those who died from it, when contemplating the course and consequences of the disease, which Hippocrates reported so truly and appropriately more than 2,000 years ago. If every century could produce one physician so observant, rather than so many who are educated in theoretical systems, how much more would have been achieved for humanity and for animal life generally. *[207] The Book of Women's Diseases* contains from paragraph sixty nearly to paragraph ninety an historical description of all those forms under which puerperal fever sporadically manifests itself. *The Book of Epidemics* contains observations regarding its epidemic manifestations that are so accurately and masterfully depicted that it could not have been done more correctly had it been recorded yesterday at bedside or in the dissection room.[26]

From the success of the chlorine washings I am convinced that atmospheric influences have not brought about the endless series of puerperal epidemics. The cases reported in the medical literature are all avoidable cases of infection from without. Readings in the history of childbed fever confirm this perfectly.

I will now recapitulate the facts that have convinced me that there are no atmospheric-cosmic-terrestrial influences capable of causing childbed fever, and that there never have been such influences. . . . The most important reason is that in three different institutions I greatly reduced mortality by destroying decaying matter. This could not have been done if atmospheric influences caused childbed fever. *[208]* The disease appears in large numbers in every season; this is not compatible with the atmospheric concept of childbed fever, but the reason is clear as soon as one knows that childbed fever arises from external infection. . . . Atmospheric influences cannot explain why hospitals are spared for many years while later, for many more years, they are afflicted annually with so-called epidemics of childbed fever. . . . *[209]* Atmospheric influences cannot explain why different divisions in one and the same institution have different conditions of health, or how it happens that maternity patients in the city at large are healthy while patients in hospitals suffer from epidemic childbed fever, or why to save these patients it is necessary to close the maternity hospitals and to force these women to be delivered in the city. Atmospheric influences cannot explain why, at a given time, different maternity hospitals in the same city have different conditions of health. But all this is

26. [Rogers] Lucas Johann Boër, *Abhandlungen und Versuche zur Begrundung einer neuen, einfachen und naturgemässen Geburtshülfe* (Vienna: [von Mösk], 1810), [vol. 2, pp. 3f.; author's note].

clear once we know that childbed fever originates by the spread of decaying matter. . . .

At the first clinic during a six-year period the mortality rate was three times as great as in the second clinic, although the clinics were separated only by a common anteroom. In Strasbourg two wards were separated only by a room containing the beds of student midwives, but the wards had significantly different mortality rates. *[210]* In the Maternité childbed fever raged as early as the end of the eighteenth century; in Vienna it began in 1823. In Dublin during ninety-eight years mortality reached 3 percent in only two years. In seven maternity hospitals in England, Ireland, and Scotland the mortality rate averaged only 1 percent. According to the epidemic theory of childbed fever, atmospheric influences which cause the disease are spread over whole continents. . . . How can such a theory be brought into harmony with the observed distribution of the disease? Why were atmospheric influences that already afflicted the Maternité at the end of the eighteenth century so slow in being extended to Vienna? How did these influences finally come to operate more furiously in Vienna than in Paris? How is it that these influences were so weakened in England, Scotland, and Ireland? Given that the Viennese clinics shared a common anteroom, what barrier protected the second clinic so successfully from influences that were otherwise spread over the whole continent? . . . *[211]* . . . As soon as one knows that childbed fever arises from decaying matter which is conveyed from external sources, explanations are easy. . . .

Out of respect for obstetricians I prefer to believe that until now no one has attempted to reconcile the epidemic theory with these universally known facts. I cannot believe that anyone seriously interested in the truth can continue to believe in the epidemic theory after its disharmony with these facts becomes apparent. *[212]* Anyone who has been confronted by these facts and who, nevertheless, continues to believe in epidemic childbed fever does not have the courage to acknowledge the truth. Perhaps such persons fear that upon recognizing the truth a great guilt is imputed. However, the facts cannot be changed, and denying the truth only increases guilt. Those who continue to believe in epidemic childbed fever can have no conviction or comprehension, they carry in their minds only memorized words.

The doctrine of epidemic childbed fever explains the unknown with the unknown. There are many deaths; we know not why. One seeks to explain these by unknown atmospheric influences, but one cannot specify any particular influences, since childbed fever occurs in every season and in every climate.

These are the reasons for my conviction. For the sake of humanity, I wish that everyone who reads this may derive the same conviction. To spare further review, those considerations that are advanced in support of atmospheric influences and that have not yet been refuted will be examined in the course of criticizing my opponents.

Chapter 4

Endemic Causes of Childbed Fever

[213] Overcrowding in maternity hospitals is an endemic cause of childbed fever only because in a crowded hospital it is more difficult to maintain the necessary degree of cleanliness and to isolate dangerous individuals. Overcrowding can foster the creation and spread of decaying matter in these ways. However, when the necessary degree of cleanliness is observed, so that no decaying matter is created, or when dangerous individuals can be isolated, or when no dangerous persons happen to be present, it does not matter whether a maternity hospital is crowded. [Semmelweis provides forty-four pages of tables to prove that the mortality rate in the Viennese hospital was not related to the degree of overcrowding. This largely repeats Table 3 above.]

[214–58] We have not yet considered puerperal miasma, because in the first clinic this was never used to explain the mortality rate. [For the sake of completeness] we will now do so. If several healthy patients and their infants are in one room, the air becomes saturated with skin odors, milk secretion, lochial discharge, etc. If these exhalations are not promptly removed through ventilation, they begin to decompose. If the decomposed exhalations penetrate the genitals of the patients, childbed fever can result. If diseased patients are in a room and if decaying matter is exuded by them, it can become mixed with the air in the room and penetrate the genitals of the patients and can cause childbed fever. *[259]* If this is what one understands by puerperal miasma, then I do not object; anything else that may be understood by puerperal miasma is non-existent. To protect against the decomposition of physiological exhalations it is sufficient to ventilate by opening windows. To protect healthy patients from the ex-

halations of decaying matter from diseased patients, the diseased patients must be isolated.

Since the creation of puerperal miasma supposedly depends on the number of patients present, the preceding tables showing that mortality is not dependent on overcrowding also prove there is no creation of puerperal miasma. This also follows from the fact that I significantly reduced mortality in the first clinic without taking any precautions to destroy puerperal miasma. The only prophylaxis for childbed fever was washing the hands with chlorine.

Puerperal fever does not generate a contagium, it produces a miasma only in the sense previously mentioned, and it cannot be contracted through uninjured outer body surfaces. It follows that puerperal fever does not infect a hospital so that healthy patients become infected merely by being there. *[260]* There can be few places where more maternity patients have died than in the sick room of the first clinic. After I initiated chlorine washings, the sick room was only occasionally required, so it was used as a maternity room. But without tearing out the floor or scraping down the walls, and after only changing the beds, the patients cared for in this room remained healthy. A location can bring forth childbed fever only if it becomes so polluted with decaying matter that the exhalations of the decaying matter saturate the air and penetrate the genitals of the individuals therein. A dissection room could become contaminated to this degree, but rooms in a maternity hospital could not.

Fear is not a causal factor in childbed fever; fear can neither transmit decaying matter to an individual nor cause it to be generated internally. As I mentioned, fear cannot explain the origin of the high mortality rate in the first clinic, because fear comes only as a consequence of such a mortality rate. Similarly, I was totally unable to prevent patients from being afraid, but nevertheless they became healthier. They were admitted with the same fear but childbed fever declined. If fear were an etiological factor in childbed fever, childbed fever would appear outside the hospitals as frequently as inside; fear is not limited to those who deliver inside the hospital. As every active obstetrician knows,

women fear throughout pregnancy that they will not survive delivery; even after many pregnancies they fear that this time they will pay for delivery with their lives. *[261]* In nearly all texts on obstetrics, one reads that particularly toward the end of pregnancy the fear of death embitters the lives of pregnant women. Many fear death ten or twelve times in this way without contracting puerperal fever.

Most of those who deliver in maternity hospitals are single women from the comfortless classes. Throughout pregnancy they have earned their bread by hard work, they have experienced need and suffering, they have been deprived of emotional support, and they have led generally unhappy and dissolute lives.[1] But these circumstances do not infect them with decaying matter, nor do they foster the internal generation of decaying matter. Therefore these factors do not cause childbed fever. Certainly not everyone who delivers in a maternity hospital lives in this way. [Moreover,] if these factors caused the disease, the mortality rate outside the hospital would be larger—not all those who deliver outside the hospitals are happy, virtuous women who spend their days in pleasant living.

It has been proposed that women are embarrassed to deliver in the presence of men and that offended modesty is a causal factor in childbed fever. But offended modesty does not convey decaying matter from without and it does not create it internally. The thoughtlessness of discussions of the etiology of childbed fever is evident here: victims are depicted as dissipated and yet assumed to possess such tender modesty as never appears in the higher classes. *[262]* Women in the highest circles also give birth in the presence of physicians; their offended modesty does not

1. Most of those who delivered in maternity hospitals were unmarried. Physicians often regarded this as of etiological significance. One British physician noted, "In hospitals seduced women are always an easy sacrifice; but, even among the affluent, powerful secret causes of mental depression may act with as much force, and expose them to its influence. Such causes are generally unknown to the physician." Edward William Murphy, "On Puerperal Fever," *Dublin Quarterly Journal of Medical Science* 24(1857), 1–30: 19. These views certainly were related to the idea that puerperal fever was sometimes due to providence. Oliver Wendell Holmes, "The Contagiousness of Puerperal Fever," in *Medical Essays* (New York: Houghton, Mifflin, and Co., 1883), pp. 103, 125.

cause them to die of childbed fever. Because of our system for educating midwives, they are sufficiently trained to assist with most births. Fortunately for both mother and child, only rare cases require the help of an obstetrician. In many lands obstetricians are called only in these rare cases. However, the help that only the obstetrician can provide must often be provided quickly if it is to be of any use. If the obstetrician is not called until danger arises, he often arrives too late to help. Because of such experiences, an obstetrician should attend every birth. If offended modesty were a causal factor in childbed fever, in order to protect a few persons from this sort of danger, every patient would need to be exposed to the threat of childbed fever. If offended modesty were the etiological factor in childbed fever, male obstetricians would have to be forbidden.

Conception, pregnancy, hyperinosis, hydremia, plethora, individuality, dietary mistakes, and chilling are not etiological factors in childbed fever because these factors neither convey decaying matter from without nor foster its generation internally. *[263]* If these were etiological factors, childbed fever would not be limited to central Europe or to modern times.

Conveyers of decaying matter include chief physicians and assistants who, either for their own instruction or in teaching their students, contaminate their hands; the head of a surgical ward who simultaneously directs an obstetrical ward; directors of united gynecological and obstetrical wards; students of practical obstetrics who attend pathological or forensic autopsies or autopsies of persons who die in maternity wards, or who visit surgical wards; students of obstetrics who enroll in courses involving operations on corpses, or who attend courses in the microscopic study of organic tissues; functioning assistants who assign students to perform obstetrical operations on cadavers; assistants and students who perform autopsies; directors of maternity clinics and their assistants who treat diseases in which decaying matter is generated; sick patients who are delivered or treated in rooms in which healthy patients are also delivered or treated; hospital personnel who administer douches to the ill and then examine large numbers of healthy people; the many items, for example sponges, instruments, washing bowls, etc., that are used

both for the ill and for the healthy; linen and bed equipment that is not always kept sufficiently clean; air in maternity hospitals that is saturated with decaying matter, either because the exhalations of the patients are not removed by ventilation, or because decaying matter is conveyed to the hospital as, for example, from a nearby morgue or from an open sewage canal. Outside the hospital, the same etiological factors induce childbed fever; for example, medical personnel, male or female, who examine with contaminated hands, or who use contaminated equipment. *[264]* All these are causal factors; many more could be added if it were not superfluous to do so. Everything that brings decaying matter to the individual from external sources belongs in this category. These are the etiological factors that devastate maternity patients—devastations falsely attributed to atmospheric influences.

Etiological factors that can cause an individual to generate decaying matter internally include retention and decomposition of normal lochial discharge; retention of the placenta, or of the remnants of the placenta or its membrane; retention of blood clots in the uterus; and crushing of the genitals during prolonged labor, or in consequence of operations that produce necrosing lacerations in the pelvic tissues. *[265]* Only extended observation can reveal whether there are additional causes of self-infection. Until now my observations have been made in institutions where it was not possible to prevent all infection from without. However, cases of self-infection may be few, since in Vienna of 2,012 patients in 1797 and 2,046 patients in 1798 only 5 patients died each year; 1 in 400.

Index