

Ignaz Semmelweis
*The Etiology, Concept, and
Prophylaxis of Childbed Fever*

Translated and Edited, with an Introduction,
by
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The University of Wisconsin Press

Contents

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Preface

Because of the drama and pathos of his life and work, many people have some idea of what Ignaz Semmelweis accomplished. Few have bothered to look further. Yet his great book, *Die Aetiologie, der Begriff, und die Prophylaxis des Kindbettfiebers*, is surely among the most moving, persuasive, and revolutionary works in the history of science. Because it concerns birthing practices, it is also of particular current interest. Moreover, it contains, in one volume, the exposition of a scientific theory, an autobiographical account of the origin of the theory, and a good sample of the reaction of the scientific community to the promulgation of that theory. For these reasons, it seemed to me that a translation of the *Aetiologie* should be of general interest and that it would also be an ideal text for a course I regularly teach in the history and philosophy of science. My object in preparing this translation has been to promote awareness of Semmelweis's contribution and significance.

In 1941, Frank J. Murphy published an English translation of the *Aetiologie*. But Murphy himself later identified certain weaknesses in his work, and since the translation appeared in a journal, it was never readily accessible. But for all its weaknesses, Murphy's translation has done much to promote the study of Semmelweis's work, and it was certainly helpful to me in preparing this new translation.

The original edition of the *Aetiologie* is much better written than most scholars seem to believe; nevertheless, it is a book that profits from abridgment. I have abridged the text in three ways. First, I left out about fifty pages of tables. The remaining tables have been renumbered (the pages on which they appeared in the German edition are in brackets), and their contents are identified in the list of tables above. Second, I cut back significantly on Semmelweis's polemical and repetitious responses to his critics, which constitute the sixth chapter. Although I reduced this part of the book to about half its original length, Semmelweis's ar-

gument should not suffer, since almost everything that was left out appears elsewhere in the book. I have usually provided, in brackets, a brief indication of the content of these longer omissions. Third, Semmelweis's sentences often contain redundant phrases; I have eliminated these throughout the entire text. Because I so frequently omit redundancies, I have used ellipses to indicate omissions only when one or more complete sentences have been omitted. Partly because of the abridging process and partly because many of Semmelweis's paragraphs consist of one long sentence, the paragraphs in this edition do not correspond to paragraphs in the original. As an aid to those who wish to refer to the German text, I have inserted, in brackets, page references to the German first edition at the beginning of the translation of the first German sentence from that page; these references indicate when one or more complete pages have been omitted.

Whenever possible I have inserted given names in brackets before surnames on the first occasion that Semmelweis uses the surname unless, of course, Semmelweis himself provides the given names. I have also inserted, in brackets, abbreviated definitions of the technical medical terms that Semmelweis occasionally uses. The definitions are usually from the twenty-fifth edition of *Dorland's Medical Dictionary* and are given at the first occurrence of the technical term. Semmelweis frequently provides some bibliographical information for the books that he cites, but the information is usually included in the text and it is seldom complete. I have completed these references and moved them into footnotes. Since I contributed most of the footnotes, it seemed most practical to identify, with a bracketed comment, those footnotes that are substantially Semmelweis's. Unless there is such a comment, the footnote is my own. The German text has chapter titles for all but the first chapter, and the chapters are unnumbered. As an aid to the reader, I have numbered all the chapters and given a title to the first one.

In examining Semmelweis's figures, I found that some of the percentages do not derive exactly from the figures printed in the tables as their source. In many cases, this seems to have been

because Semmelweis simply dropped final digits rather than rounding them off. Because it is impossible to determine the reason for the errors and because I want to present Semmelweis's figures as his nineteenth-century audience read them, I have left the figures as they appear in the German edition. In a few instances, however, where the divergence seems to be greatest, I have given what would appear to be the correct percentages, in brackets following the figures of the German text.

It remains only to thank several people and institutions who have generously helped me with this project. I would like to thank an understanding department chairman for a slight reduction in teaching load that provided time to finish the project, and Brigham Young University for a generous travel allowance that enabled me to do a lot of last minute checking of sources. I would like to thank the editors of *Medical History* for permission to use my essay on Semmelweis, which appeared in the pages of that periodical, as a basis for the introduction to this edition. In the course of preparing the translation I checked each of Semmelweis's quotations against the original source. In doing so I used the holdings of several libraries, especially the Lane Medical Library at Stanford University, the Eccles Medical Library at the University of Utah, the Library of the College of Physicians in Philadelphia, the Medical Library of Johns Hopkins University, and the National Library of Medicine in Washington. I very much appreciate having enjoyed access to these libraries and the help of their staffs. I would like especially to thank Dorothy Hanks of the History of Medicine section of the National Library of Medicine for help in locating and copying many of Semmelweis's sources. I would like to thank Randy Everett, Travis Tucker, Jan Chambers, Carol Metcalf, Lynn Stosich, and Vanesse Tracy for various kinds of help in preparing different versions of the text. I very much appreciate the help of Jim and Julie Siebach, who typed most of the final version of the translation. I also express appreciation to the readers and to the editorial staff of the University of Wisconsin Press for many helpful suggestions that significantly improved the quality of the final result. I must also thank my two sons, Christopher and Thaddeus, who were

responsible for the wording of certain passages. Finally, I thank my wife, Barbara, for her unwavering confidence and encouragement and for her eagerness to turn every dull conversation to the lively topic of nineteenth-century Hungarian obstetrics.

The Etiology, Concept, and Prophylaxis
of Childbed Fever

Translator's Introduction

"My doctrines exist to rid maternity hospitals of their horror, to preserve the wife for her husband and the mother for her child." In these words, Ignaz Semmelweis summarized his own life's work. His career, however, came at a time when medical theory and practice were changing dramatically, and his work can therefore be viewed from several vantage points.

In the first place, he was among the earliest to adopt certain aseptic medical procedures such as washing with disinfectants and the use of nail brushes.¹ In this respect his work represents an important advance in practical medicine. Most of those who have written on Semmelweis have emphasized this aspect of his work, and there have been numerous discussions of the relative priority of Semmelweis and of Lister and Oliver Wendell Holmes, who adopted similar procedures at about the same time.²

Second, Semmelweis's work came at a time of significant improvement in the care of hospital patients. This was particularly true in the maternity wards with which he was associated. By the beginning of the nineteenth century, large gratis hospitals had been established in most of the major cities of Europe. These hospitals served two functions, both dictated by the humanitarian objectives of the Enlightenment: they provided both free medical care for the indigent and a virtually unlimited supply of disposable bodies, living and dead, on which medical students could learn and practice their crafts. Associated with these hospitals were special maternity clinics. Each year thousands of poor women, usually unmarried, went to the clinics to deliver. In ex-

1. Semmelweis may have been the first to insist on use of the nail brush in cleaning the hands before examinations and surgery. This is briefly discussed in György Gortvay and Imre Zoltán, *Semmelweis: His Life and Work* (Budapest: Akadémiai Kiadó, 1968), pp. 210f.

2. For references, see Frank J. Murphy, "Ignaz Philipp Semmelweis (1818-65): An Annotated Bibliography," *Bulletin of the History of Medicine* 20 (1946), 653-707: 688-707.

change for free medical care and for the services of the associated foundlings homes, these women submitted themselves for use as teaching specimens in the training of obstetricians and midwives. But the maternity clinics were dreaded by the very women they ostensibly served; conditions in the clinics were often horrible. Semmelweis relates that in one major Parisian hospital in 1786, as many as three patients were obliged to sleep in each four-foot-wide bed.³ He also mentions that in Vienna, three hours after delivery, women were required to arise from the delivery bed and walk through a passageway to their own beds,⁴ that women were sometimes obliged to use uncleaned linen that was still stained with the blood and lochial discharge of earlier patients, and that the air of maternity wards sometimes reeked from the emanations of nearby dissection rooms.⁵ But the clinics were especially dreaded because of their frightful mortality rate; often between ten and thirty percent of those who were admitted did not escape with their lives. They died, shortly after delivery, from a disease known as childbed fever or puerperal fever. During most of his career, Semmelweis was associated with maternity clinics in Vienna and Budapest. His autobiographical account contains graphic and moving descriptions of conditions in these clinics. Semmelweis himself was active in the movement to improve the conditions of patients;⁶ he adopted measures to make delivery easier and more comfortable;⁷ and, most important, he drastically reduced the incidence of childbed fever. Because his work was associated with the improvement of the maternity hospitals, he is usually mentioned in social histories of childbirth.⁸

3. See below, p. 153; German edition, p. 204.

4. See below, p. 74; German edition, p. 36.

5. See below, p. 113; German edition, p. 99.

6. He wrote petitions to the government in the effort to secure better facilities for patients. For example, see below pp. 108–10; German edition, pp. 85–98.

7. 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. Gortvay and Zoltán, op. cit., note 1 above, p. 48.

8. For example, see Richard W. Wertz and Dorothy C. Wertz, *Lying-in: A History of Childbirth in America* (New York: Free Press, 1977), pp. 45, 94.

Third, Semmelweis's work came just at a time when medicine was becoming emancipated from certain presuppositions about sexuality. In the early nineteenth century, many medical beliefs and practices were based on discrimination by sex. It is well known that women were systematically excluded from the medical profession, even from the traditional practice of midwifery. "By the mid-nineteenth century this process was so complete and so deeply institutionalized in society that it was necessary to rediscover the fact that women had been engaged in the healing arts in earlier times."⁹ But sexuality was the basis of more subtle forms of discrimination as well. For one thing, the use of such standard therapeutic procedures as bloodletting seems to have been based in part on presuppositions about sexual roles.¹⁰ Moreover, there was a particular range of disorders, such as leukorrhea, hysteria, and puerperal fever, that were specifically identified as women's diseases.¹¹ Through the second half of the century, however, these forms of sexual bias were reduced or eliminated. Women were gradually admitted to the medical profession. There was a revolution in therapeutics that virtually eliminated many of the earlier procedures which had been used to reaffirm and to enforce social and sexual norms.¹² With the adoption of germ theory, such diseases as leukorrhea and puerperal fever could be seen as simple infections that had no essential sexual significance. Germ theory, and the new prophylactic and therapeutic measures that were based on it, rested in part on

9. Noel Parry and José Parry, *The Rise of the Medical Profession* (London: Croom Helm, 1976), p. 164.

10. K. Codell Carter, "On the Decline of Bloodletting in Nineteenth-Century Medicine," *The Journal of Psychoanalytic Anthropology* 5(1982), 219–34.

11. Some of these diseases are discussed by Edward Shorter, "Women's Diseases before 1900," in Mel Albin, ed., *New Directions in Psychohistory* (Lexington, Mass.: Lexington Books, 1980), pp. 183–208. By the middle of the century it had already been recognized that men occasionally contracted hysteria, but it was still regarded as a predominantly female disease. K. Codell Carter, "Germ Theory, Hysteria, and Freud's Early Work in Psychopathology," *Medical History* 24 (1980), 259–74: 265n.

12. Charles E. Rosenberg, "The Therapeutic Revolution," in Morris J. Vogel and Charles E. Rosenberg, eds., *The Therapeutic Revolution: Essays in the Social History of American Medicine* (Philadelphia: University of Pennsylvania Press, 1979), and Carter, op. cit., note 10 above.

a new strategy for characterizing diseases. Semmelweis was among the first to use this strategy.¹³ In this respect it is significant that the first step in Semmelweis's work on puerperal fever, like Freud's first step in his work on hysteria, was recognizing that either sex was vulnerable to a particular disease that had previously been believed to affect only women.¹⁴ Incidentally, Semmelweis's own attitude toward women in the medical profession can be inferred from his comments that all the midwives he trained were more enlightened about childbed fever than Rudolf Virchow, the most famous pathologist of the time, and were better prepared to avoid it than the members of the Obstetrical Society in Berlin.¹⁵ Thus Semmelweis's work reflects the changing sexual orientation of medicine, and it can, therefore, be viewed in relation to the development of modern conceptions of sexuality.

Semmelweis can also be viewed as a contributor to the theoretical basis of medicine. As we will see, Semmelweis seems to have been among the first to adopt a particular strategy for characterizing diseases, a strategy that subsequently became central to medical theory. Yet, while his contributions to the practice of medicine have been generally recognized, this aspect of his work has been almost totally ignored. Moreover, most accounts of Semmelweis's life and work are discursive and superficial narratives that, like many earlier discussions of other figures and topics in medical history, are so "lacking in critical framework as to be of almost no use to succeeding scholars."¹⁶ In reading

13. This strategy and the claim that Semmelweis was among the first to employ it will be justified in the fourth and fifth sections below.

14. Strictly speaking, by the time of Freud, hysteria was generally known to affect men as well as women. At first, Freud seems not to have realized that this was the case. In any event, the disease was still believed to be much commoner among women, and often to be caused by factors associated with women's sexual roles. See Carter, *op. cit.*, note 11 above.

15. See below, pp. 232f.; German edition, p. 477. By contrast, according to Parry and Parry, the common view at this time was that "only a man could combine the necessary scientific and anatomical knowledge with physical strength and precision in the use of obstetric instruments which would allow him safely to practice midwifery." Parry and Parry, *op. cit.*, note 9 above, p. 169.

16. Charles E. Rosenberg, "The Medical Profession, Medical Practice and the History of Medicine," in Edwin Clarke, ed., *Modern Methods in the History of Medicine* (London: Athlone Press, 1971), p. 27.

these accounts one frequently has the impression that the authors relied exclusively on common knowledge or on secondary sources, and that they gave little or no attention to Semmelweis's own writings.¹⁷ Some writers have recognized that in adopting aseptic procedures, Semmelweis may have been more dependent on theoretical considerations than was, say, Holmes.¹⁸ But no one has attempted to explicate those considerations or to determine their relation to the theoretical presuppositions of his contemporaries or successors. For this reason, the endless debates about the relative priority of Semmelweis, Lister, and Holmes, or about the relation of Semmelweis to his teachers in Vienna, simply could not lead to decisive conclusions. Moreover, Semmelweis's theoretical presuppositions provide important clues to understanding the social context of his work. Because physicians treat as well as describe and explain, medical theories are socially immanent in a way that the theories of physics or biology, for instance, are not. Consequently, one must expect that medical theory will intimately reflect and be reflected by the social role of the physician. As suggested above, for example, changes in medical theory often correlate with social changes in the practice of medicine or in the organization of the profession. More than in many sciences, therefore, one would expect medical theory and the social context of the practice of medicine each to provide useful clues about the other. For these reasons it may

17. Murphy pointed this out with respect to one of Semmelweis's early critics, *op. cit.*, note 2 above, pp. 690, 694. Matters haven't improved much since. The most accessible modern edition of Semmelweis's main book, *Die Aetiologie, der Begriff, und die Prophylaxis des Kindbettfiebers*, has been a reprint of the German first edition and was published in 1966 by the Johnson Reprint Corporation in New York and London. This edition is preceded by an introductory essay by Alan Guttmacher, who probably never looked at the text—he claims, for example, that Semmelweis "mentions only one author, Hippocrates, by name" (p. xxvi), whereas in fact Semmelweis mentions nearly one hundred, most of whom are quoted extensively. Still more recently, Sherwin B. Nuland managed to write an "interpretation" of Semmelweis with only one quotation from the *Aetiologie*, and perhaps because "rendering the true sense of Semmelweis's labored German into effective English has proved inordinately difficult," that single quotation was taken from a secondary source. "The Enigma of Semmelweis—an Interpretation," *Journal of the History of Medicine and Allied Sciences* 20 (1979), 255–72: 267.

18. Gortvay and Zoltán, *op. cit.*, note 1 above, pp. 211–13.

be useful to consider the particular theory of disease that Semmelweis adopted and that is presupposed in his writings.

We will begin by considering more carefully the disease on which Semmelweis focused his attention—childbed fever.

II

Childbed fever seems to have been known in antiquity, but the modern name dates from the seventeenth century;¹⁹ by the late eighteenth and early nineteenth centuries, the disease reached horrible proportions. Early in the nineteenth century childbed fever was typically characterized symptomatically. In one essay it was defined as “that disease which is ushered in, from the second to the fourth day of confinement, by shivering, accompanied by acute pain, radiating from the region of the uterus, increased on pressure, and gradually extending all over the abdomen, with suppression of lochia and milk, much accelerated pulse, furred tongue, great heat of skin, and that peculiar pain in the sinciput . . . short breathing, the knees drawn up, and great anxiety of countenance.”²⁰ In harmony with the general program of pathological anatomy, there were numerous attempts to replace such symptomatic characterizations with more precise anatomical ones. The anatomical characterizations usually focused on morbid structural alterations in the uterus. This seemed reasonable, since the disease appeared to be closely associated with the birth process and since autopsies often disclosed morbid alterations in that organ. Such attempts were not particularly successful, however, because the disease left a variety of different and apparently unrelated lesions. According to the basic principles of pathological anatomy, this suggested that puerperal fever was not a single disease but rather a cluster of symptomatically similar diseases, each of which should be associated with a unique set of pathological alterations. Most physicians who wrote on puerperal fever in the early decades of the nineteenth century

19. *Ibid.*, p. 41.

20. C. M. Miller, “On the Treatment of Puerperal Fever,” *Lancet* 2 (1848): 262.

adopted some scheme for classifying different cases of the disease, depending on the specific anatomical lesions that were discovered in autopsy.²¹ However, some physicians found these distinctions to be artificial, and, moreover, there were many cases of the disease in which autopsy disclosed no pathological remains whatsoever.²² As a result there was considerable controversy about the ultimate nature of the disease and about the best means of characterizing it. Through the middle decades of the nineteenth century medical writers often stressed the difficulty of adequately characterizing puerperal fever.²³

There were many different theories about the causes and nature of childbed fever; in particular there were major disputes about whether or not it was contagious. The British and most Americans were impressed by instances in which the disease seemed to have spread from one patient to another, and they

21. This was especially true on the continent. Eduard Martin wrote that the usual terms ‘childbed fever’ and ‘puerperal fever’ were “inappropriate for scientific discourse because they encompass very different pathological manifestations.” “Über eine im Winter 1859–60 beobachtete Epidemie puerperaler Colpitis und Endometritis,” *Monatsschrift für Geburtskunde und Frauenkrankheiten* 16 (1860), 161–76: 161. In place of these terms most physicians used ‘puerperal processes’ and had a scheme for classifying cases that focused on the specific anatomical lesions that were involved. See, for example, Eduard Lumpe, “Die Leistungen der neuesten Zeit in der Gynäkologie,” *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 1 (1845), 341–71: 342; and Carl Braun, “Zur Lehre und Behandlung der Puerperalprocesse und ihrer Beziehungen zu einigen zymotischen Krankheiten,” in Baptist Johann Chiari, Carl Braun, and Joseph Späth, *Klinik der Geburtshilfe und Gynäkologie* (Erlangen: Enke, 1855), p. 423. Both Lumpe and Braun cite several other authors who recommended a similar approach.

22. J. M. Waddy, “On Puerperal Fever,” *Lancet* 2 (1845), 671f.: 671. James Young Simpson, “Some Notes on the Analogy between Puerperal Fever and Surgical Fever,” *Monthly Journal of Medical Science* 11 (1850), 414–29: 419.

23. “There is almost no disease which varies more than puerperal fever does in different cases, in the intensity of its symptoms, and in the forms which they assume. . . . There is no disease to which it is so difficult to assign a set of pathognomonic phenomena.” *Ibid.*, pp. 425f. “There are major difficulties in giving a satisfactory definition of puerperal fever because in most special cases it is impossible to identify a common characteristic criterion for this disease.” Franz Kiwisch von Rotterau, *Klinische Vorträge über specielle Pathologie und Therapie der Krankheiten des weiblichen Geschlechtes*, 4th ed. (Prague: J. G. Calve, 1854), vol. 1, p. 600.

concluded that it was often the result of contagion. Continental physicians, on the other hand, although sometimes admitting that the disease could occasionally be contagious, reported instances in which the disease did not spread as one would have expected of a contagious disease, and they generally emphasized other kinds of causation. We will consider each of these opinions in somewhat greater detail.

In 1843 Oliver Wendell Holmes published an essay entitled "The Contagiousness of Puerperal Fever"; in 1855 the essay was reprinted with an introductory note and with an appendix containing additional references and cases but with no change in the body of the text.²⁴ Holmes's main object was to show that "the disease known as puerperal fever is so far contagious as to be frequently carried from patient to patient by physicians and nurses."²⁵ Holmes's conclusion and most of the specific case histories on which the conclusion is based are from earlier British literature. Holmes himself admitted, both in the essay and again in the later introductory note, that the position he espoused was a majority view. "A few writers of authority can be found to profess a disbelief in contagion—and they are very few compared with those who think differently."²⁶ But Holmes felt that the existence of the minority justified the essay.

Holmes cited nearly twenty cases in which physicians examined or otherwise treated patients with puerperal fever, or in which they performed autopsies on persons who had died from puerperal fever and in which other patients subsequently contracted the disease. This suggested that the disease was spread from patient to patient and that the attending physician acted as the carrier. Hence, Holmes concluded that there must be some conta-

24. The essay appeared originally in the *New England Quarterly Journal of Medicine and Surgery* in 1843; it was reprinted in Holmes's collected *Medical Essays* (New York: Houghton, Mifflin, and Co., 1883), pp. 103–72.

25. *Ibid.*, p. 131.

26. *Ibid.*, p. 129. A year before the first publication of Holmes's essay, the editors of *Lancet* reported that in a discussion of puerperal fever at a meeting of the London Medical Society "the chief apparent circumstance is the diversity of opinion . . . as to the nature, . . . the symptoms and the treatment of the affection. . . . One fact only respecting the disease was generally admitted, namely, its unquestionable contagiousness." *Lancet* 1 (1842): 879.

gion that was generated by puerperal fever victims and that could be carried to other patients. At the end of this essay he warns obstetricians against taking "any active part in the postmortem examination of cases of puerperal fever" and that "on the occurrence of a single case of puerperal fever in his practice, the physician is bound to consider the next female he attends in labor . . . as in danger of being infected by him."²⁷ Holmes considers cases suggesting that puerperal fever can be produced "by an infection originating in the matter or effluvia of erysipelas."²⁸ But he finds the relation of puerperal fever to other continued fevers to be "remote and rarely obvious." Thus while he mentions very briefly reports that "puerperal fever has appeared to originate from a continued proximity to patients suffering with typhus," he adds that these cases are so relatively rare that they "hardly attract our notice in the midst of the gloomy facts by which they are surrounded."²⁹ Holmes never suggests that patients suffering from other diseases or corpses of persons who died from other diseases present any special danger to delivering women. Indeed, he observes that "the number of cases of serious consequences ensuing from the dissection of the bodies of those who had perished of puerperal fever is so vastly disproportioned to the relatively small number of autopsies made in this complaint as compared with typhus or pneumonia (from which last disease not one case of poisoning happened), and still more from all diseases put together, that the conclusion is irresistible that a most fearful morbid poison is often generated in the course of this disease."³⁰

Holmes did not believe that exposure to the contagion was necessary in order for one to contract the disease—it could also come about in other ways. Each time Holmes states his main point precisely he says that the disease is sometimes (or frequently) carried from one patient to another.³¹ "It is not pretended that the disease is always, or even, it may be, in the ma-

27. Holmes, *op. cit.*, note 24 above, p. 168.

28. *Ibid.*, p. 164.

29. *Ibid.*, p. 165.

30. *Ibid.*, p. 162.

31. *Ibid.*, pp. 112, 129, 131.

jority of cases, carried about by attendants; only that it is so carried in certain cases."³² Following the British, Holmes distinguished cases arising from infection from other cases that were epidemic or sporadic. "It is granted that the disease may be produced and variously modified by many causes besides contagion, and more especially by epidemic and endemic influences."³³ In the chronologically later introductory note, he writes that his theory "makes full allowance for other causes besides personal transmission, especially for epidemic influences."³⁴ In the literature of the times, epidemic influences were generally identified with atmospheric or terrestrial factors that could not be specified with any precision.³⁵ Since these factors could not be measured, the only criterion for deciding whether a given case of puerperal fever was epidemic was the frequency of similar cases in the surrounding area.³⁶ Sporadic cases were neither epidemic nor infectious; one discussion ascribed sporadic cases "to difficult labor; to inflammation of the uterus; to accumulation of noxious humours, set in motion by labour; to violent mental emotion, stimulants, and obstructed perspiration; to miasmata, admission of cold air to the body, and into the uterus; to hurried circulation; to suppression of lacteal secretion; diarrhea; liability to putrid contagion from changes in the humours

32. *Ibid.*, p. 123.

33. *Ibid.*, p. 133.

34. *Ibid.*, p. 107.

35. For example, in Robley Dunglison, ed., *The Cyclopaedia of Practical Medicine* (Philadelphia: Lea and Blanchard, 1849), one finds the following comments: "There are presumable properties in the air, yet unknown save in their destructive effects" (1:674); febrile poisons may originate "in a peculiar unknown pestilential condition of the atmosphere . . . invisible and without taste or smell [known only by its] noxious effects on the animal body" (2:177); variations in febrile diseases "have been ascribed to the influence of some atmospheric or terrestrial agency, of which little or nothing is known except the effects it produces in the propagation and malignity of diseases" (2:181). Lumpe says similar things, *op. cit.*, note 21 above, pp. 345–47.

36. Holmes, *op. cit.*, note 24 above, p. 113. See also W. Tyler Smith, "Puerperal Fever," *Lancet* 2 (1856), 503–35: 503. Semmelweis discusses the confusion in the concept of epidemics and observes that this confusion was partially responsible for the failure to discover the cause of puerperal fever. See below, pp. 85f., 121; German edition, pp. 50f., 118.

during pregnancy; hasty separation of the placenta; binding the abdomen too tight; sedentary employment; stimulating or spare diet; [or to] fashionable dissipation."³⁷ In addition to contagion, epidemic influences, and the multitude of factors recognized as responsible for sporadic cases, Holmes also believed, as did most physicians who wrote on the disease, that puerperal fever could arise spontaneously.³⁸ Some physicians also ascribed cases of the disease to providence.³⁹

In 1845, two years after the first publication of Holmes's essay, Eduard Lumpe, assistant at the first obstetrical clinic in Vienna, published an informed and carefully documented summary of current continental opinions about puerperal fever.⁴⁰ Lumpe discussed the difficulty of identifying consistent anatomical remains in terms of which the disease could be characterized. He confidently asserted that the disease was predominantly epidemic—"this is adequately proved by occasional increases and decreases in the number of cases without any change in those factors most commonly recognized as causes, by the simultaneous occurrence of cases, and by the similarity of the course of those cases that are simultaneous."⁴¹ Lumpe felt that the disease usually attacked the uterus and that this could be the point of attack even if autopsy disclosed only minor changes in the uterus itself. According to Lumpe, maternity patients were particularly disposed to the disease because "lochial secretions, the purpose of which is

37. Waddy, *op. cit.*, note 22 above, p. 671.

38. Holmes, *op. cit.*, note 24 above, pp. 139f., cites with approval a long passage in which a Dr. Blundell observed that "this fever may occur spontaneously." To say officially that a disease occurred spontaneously did not mean that it occurred without causes but only that the causes eluded observation. One can find passages in which this distinction was not carefully maintained, however.

39. Holmes severely criticized Charles D. Meigs for ascribing certain cases of puerperal fever to providence. Holmes, *ibid.*, pp. 103, 125. This view was certainly connected with the fact that most of those who delivered in maternity clinics were unmarried.

40. Lumpe, *op. cit.*, note 21 above.

41. *Ibid.*, pp. 342f. Eduard Caspar Jakob von Siebold used the same argument, on p. 343 of "Betrachtungen über das Kindbettfieber," *Monatsschrift für Geburtskunde und Frauenkrankheiten* 17 (1861), 335–57, 401–17; and 18 (1862), 19–39.

to remove waste matter, can be retained or absorbed. This induces decay of the blood. The absorption of harmful matter is much more likely following delivery, as the epithelium of the womb is discharged and regenerated and lacerated veins are present."⁴² In addition to epidemic influences Lumpe identified the usual range of incidental causal factors. These included general deprivation, worry, shame, attempted abortion, fear of death, dietary disorders, exposure to cold, local miasmas, difficult delivery and especially damage to tissue because of the use of mechanical devices in delivery, and the retention and subsequent decomposition of the placenta.⁴³ Lumpe also mentioned the British view that the disease could be contagious, but he clearly regarded contagion as a minor consideration.⁴⁴ Other writers observed that on the continent, obstetricians simply had no experiences that confirmed the British belief in contagion.⁴⁵

Because puerperal fever was ascribed to such a wide range of different causes, there was nothing resembling coherent unified strategies for preventing or for treating the disease. The English, of course, disinfected their hands and changed their clothing after contacting persons with the disease,⁴⁶ but they insisted that the disease did not always originate by contagion, and no one imagined that these prophylactic steps would prevent all cases of the disease. In the early nineteenth century, so-called antiphlogistic treatments constituted an important part of therapy. These measures included bloodletting, dietary restrictions, purgatives, lotions to cool the patient, etc. Puerperal fever, like most fevers, indicated an antiphlogistic regimen and most of those who wrote

42. Lumpe, op. cit., note 21 above, p. 345.

43. Ibid., pp. 345–49. Braun, op. cit., note 21 above, pp. 485–87, lists thirty different causes for the disease; Braun's list is discussed by Semmelweis, see below, pp. 246–49; German edition, pp. 530–35.

44. Lumpe, op. cit., note 21 above, p. 348.

45. Kiwisch says this in *Canstattische Jahresbericht über die Fortschritte in der Heilkunde im Jahre 1845*, vol. 3, *Specielle Pathologie und Therapie* (Erlangen: Ferdinand Enke, 1846), pp. 430f. Semmelweis quotes this; see below, pp. 219f.; German edition, pp. 430f.

46. Edward Blackmore also recommended that physicians not neglect to wash their hair and teeth. "Observations on the Nature, Origin, and Treatment of Puerperal Fever," *Medical Examiner* 8 (1845), 292–304: 297.

on the disease endorsed this strategy.⁴⁷ On the other hand, it was usually the privileged classes—those who ate too much and too richly, who worked too little and thought too much—who became plethoric and who required antiphlogistic treatment. The poor usually required supportive treatment, the very opposite to the antiphlogistic regimen. Some physicians felt that since the women who suffered puerperal fever were usually poor, supportive treatment was necessary. Thus contradictory therapies were recommended for puerperal fever, and the therapy that was selected in a particular case was usually justified by a consideration of factors unique to the individual patient in question.⁴⁸ Moreover, since supportive and antiphlogistic treatments were intended to compensate for opposite conditions, in theory it could happen that the disease was caused by the very measures used to treat it. Needless to say, there was considerable confusion and skepticism in discussions of therapies for puerperal fever; contemporary physicians frequently complained that there simply was no known therapy that was effective against the disease.⁴⁹

Explanations were similarly defective. Lumpe called attention to various facts about the disease—he noted, for example, that it was particularly frequent in the winter, that (even though this

47. In 1848 C. M. Miller wrote that when he observed symptoms of puerperal fever he immediately ordered "eight or a dozen leeches to be scattered over the abdomen." Op. cit., note 20 above, p. 262. See also Edward William Murphy, "On Puerperal Fever," *Dublin Quarterly Journal of Medical Science* 24 (1857), 1–30: 5f.

48. See, for example, Robert Lee, "Fever, Puerperal," in Dunglison, op. cit., note 35 above, vol. 2, pp. 231–47, p. 231. Smith, op. cit., note 36 above, pp. 533f says the same thing. On the continent Kiwisch, op. cit., note 23 above, pp. 660–65, as well as Braun and others, made similar remarks.

49. "After much discussion everyone comes finally to the same depressing conclusion that an effective procedure for puerperal fever has yet to be discovered." Ferdinand Adolph Kehler, "Zur Behandlung des Kindbettfiebers," *Monatsschrift für Geburtskunde und Frauenkrankheiten* 18 (1861), 209–23: 210. Siebold, following an earlier writer, remarks that "there is perhaps no disease process against which so many different, often totally contradictory, methods of treatment have been recommended as has been the case for childbed fever"; after trying every procedure that theory or experience could suggest, one ultimately concludes that "they must all be rejected as useless and ineffective." Siebold, op. cit., note 41 above, p. 19.

contradicted the literal meaning of the name of the disease) it could occur before birth, and that it was almost always associated with the maternity clinics. He also pointed out that the Viennese maternity hospital included two adjacent clinics that were alike in every respect, and that one of these clinics had a much higher mortality rate than the other.⁵⁰ The only explanation that he could give for these facts was that they reflected differences in atmospheric and miasmatic influences. On the other hand, he also admitted that nothing was known about these influences except the very facts they purportedly explained. There was no attempt to explain how fear, shame, worry, or the trauma of a difficult delivery could all account for the same disease that was otherwise ascribed to atmospheric influences. Thus there simply were no adequate explanations for many observed facts.

This was the situation when, in 1847, Ignaz Philipp Semmelweis was appointed assistant in the first clinic of the Viennese maternity hospital—the same clinic in which Lumpe had worked just a few months earlier.

III

Semmelweis was born in Taban, the oldest part of what is now Budapest, on 1 July 1818. He was the fifth of ten children of József and Terézia Müller Semmelweis; József was a successful grocer. The family spoke a dialect of German, but Ignaz also became fluent in Hungarian. Semmelweis began studying law at the University of Vienna in the autumn of 1837, but by the following year, for reasons that are no longer known, he had changed to medicine. He was awarded his doctorate degree in medicine in 1844. After failing to obtain an appointment in a clinic for internal medicine, Semmelweis decided to specialize in obstetrics. On 1 July 1846, his twenty-eighth birthday, Semmelweis became an assistant physician in the Viennese maternity hospi-

50. Lumpe says, "Whoever doubts the power of local miasmatic influences should try to find some other explanation for the fact that through several years . . . for every ten or twelve patients lost in the second clinic, the first clinic loses four to five times as many." *Op. cit.*, note 21 above, p. 347.

tal. There he was immediately confronted by the horrible reality of childbed fever.

In the early decades of the nineteenth century the Viennese maternity hospital consisted of a private ward for women sufficiently affluent to pay their own medical costs, and two gratis clinics. Obstetricians were trained in the first clinic, midwives were trained in the second. For several years the incidence of puerperal fever in the first clinic averaged between three and five times that in the second. For this reason, women particularly dreaded the first clinic and tried desperately to avoid being admitted there. Various commissions investigated the high mortality in the first clinic and measures were proposed to reduce it; all the measures proved ineffective. The prevalence of the disease, together with the ineffectiveness of prophylactic measures, suggested that the disease was epidemic. Everyone agreed that there could be no defense against the harmful influences of the atmosphere.⁵¹

Shortly after being appointed as assistant in the first clinic Semmelweis seems to have begun looking for the cause or causes that could explain the difference in mortality rates. He recognized that the two clinics—located side by side and even sharing certain rooms—were necessarily subject to the same atmospheric influences. This fact convinced Semmelweis that the difference in mortality could not be due to atmospheric influences—in other words, that the disease was not epidemic. He then began a tenacious quest for endemic factors that could explain the difference in death rates. He decided that overcrowding, rough handling, specific medical procedures, inadequate ventilation, dietary irregularities, as well as particular physiological or psychological conditions of the patients, could not explain the incidence of puerperal fever, since all of these factors were either equally operative in both clinics or worse in the second clinic. Semmelweis gave particular attention to factors that

51. As Semmelweis himself observed, "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." See below, p. 121; German edition, p. 117.

were different in the two clinics. For example, he tells us that he discontinued supine deliveries in favor of deliveries from the lateral position simply because that was the practice 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."⁵² Of course, these measures were without effect. At this time Karl Rokitsansky, who was director of the Institute for Pathological Anatomy and who ultimately became the most prominent pathological anatomist in Europe, assisted Semmelweis by allowing him to dissect all the female corpses from the hospital. Semmelweis was able only to confirm the confused findings of other anatomists, however; he found no clear indication of the causes of the disease.

As Semmelweis later told the story, the crucial event in his quest for the endemic cause of puerperal fever occurred in March 1847. Professor Jakob Kolletschka, who had been his friend and teacher, died from a minor injury incurred while dissecting a corpse. When Kolletschka's body was dissected, pathological remains were found that were similar to those obtained in dissections of women who died from puerperal fever.⁵³ Semmelweis had already concluded that the puerperal state was not a necessary condition for inception of the disease—he noted that women could contract the disease and even die from it during delivery or even during pregnancy.⁵⁴ He observed also that when women died of puerperal fever their own newborn infants, both male and female, sometimes died of a fever that left similar pathological remains. From this he concluded that the infants also died of puerperal fever. "To recognize these findings as the consequence of puerperal fever in the maternity patients but to deny that identical findings in the corpses of the newborn are the conse-

52. See below, p. 87; German edition, p. 52.

53. See below, pp. 87f.; German edition, p. 53.

54. See below, pp. 80, 117; German edition, pp. 43, 106. Lumpe made the same observation, op. cit., note 21 above, p. 343.

quence of the same disease is to reject pathological anatomy."⁵⁵ Similar reasoning forced Semmelweis to conclude that Kolletschka also died from the same disease. "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."⁵⁶ Semmelweis now took a remarkable and decisive step: "I was forced to admit that if [Kolletschka's] 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."⁵⁷ It is easy to overlook the profound originality of this argument. Even if they had admitted that Kolletschka and the maternity patients had died from the same disease, Semmelweis's contemporaries would not have admitted that the causes would have been the same. As we have

55. See below, p. 77; German edition, p. 40, cp. p. 43. In this passage Semmelweis notes that this conclusion forced him to recognize that the whole concept of childbed fever was wrong.

56. See below, p. 88; German edition, p. 53. Erna Lesky notes that Semmelweis's first account of the discovery was published eleven years after the event, "whereas Hebra's and Skoda's versions given in the very year of the discovery—1847—and subsequently in 1848 and 1849, make no mention of this outstanding heuristic importance of the Kolletschka case. Hence it is more than likely that in reviewing the events that led to his discovery, Semmelweis in 1858 exaggerated the significance of this case." *The Vienna Medical School of the Nineteenth Century* (Baltimore: Johns Hopkins University Press, 1976), p. 185. But Hebra and Skoda did not purport to give historical accounts; their only object was to present and to justify a scientific discovery. Semmelweis says explicitly that it was his intention to present historically the events leading to his discovery. See below, p. 61; German edition, p. iv. Moreover, the account of Semmelweis's lecture before the Viennese Society of Physicians on 15 May 1850 clearly reports Semmelweis as having said that he was led to his theory by the difference in mortality between the clinics *together with* the pathological similarities between childbed fever and pyemia in surgeons and anatomists. *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 6 (1850) 2:cxxxvii–cxl: cxxxvii f. This was certainly an allusion to Kolletschka that no one in Vienna could have missed. I see no reason to think that Kolletschka's death was any less important to the development of Semmelweis's thought than Semmelweis himself tells us it was. This is the view taken by József Antall; see, for example, *Pictures from the Past of the Healing Arts* (Budapest: Semmelweis Orvostörténeti Múzeum, Könyvtár és Levéltár, 1972), p. 78.

57. See below, p. 88; German edition, p. 54, cp. pp. 40, 273, 342.

seen, they accepted the possibility of a whole range of causes. By reasoning in this way, Semmelweis shows that he was assuming a new characterization of puerperal fever—one according to which it had only one possible cause. This will be examined more fully in the next section.

The cause of Kolletschka's disease was known; it was the introduction of decaying matter into his blood from the contaminated autopsy knife. Semmelweis realized that students and other persons associated with the clinic—particularly he himself—were conducting autopsies and then, after washing with ordinary soap and water or without washing at all, were examining the maternity patients. The smell of the examiners' hands convinced Semmelweis that washing in the ordinary fashion did not remove the decaying organic matter with which they had become contaminated in the dissections. The lacerations associated with the birth process provided access through which the decaying organic matter was introduced into the patients' blood systems. The result was the same as it had been in Kolletschka. Thus, Semmelweis quickly identified the contaminated hands of the examining physicians as the source of the decaying matter that spread the same disease among the patients. In the latter half of May 1847, Semmelweis began to require everyone associated with the clinic to wash in a chlorine solution before having physical contact with the patients. The mortality rate in the first clinic immediately fell to about the same level as in the second clinic.

It is possible that Semmelweis first concluded simply that the increased mortality in the first clinic was due to a cadaverous poison. In the next few weeks, however, this hypothesis proved to be too restricted.⁵⁸ Within a short time of adopting the chlorine washings, Semmelweis noted that there were still occasional outbreaks of childbed fever. Some of these were due to students who were inadequately conscientious in the use of chlorine, but

58. Semmelweis suggests that he started with the hypothesis that the disease was due to cadaverous poison (see below, pp. 92f.; German edition, pp. 59f.), and Franz Hektor Arneth, an associate of Semmelweis's, said so quite explicitly. "Note sur le moyen proposé et employé par M. Semmelweis pour empêcher le développement des épidémies puerpérales dans l'hospice de la maternité de Vienne," *Annales d'Hygiène Publique et de Médecine Légale* 45 (1851), 281–90: 287.

he traced other outbreaks to infection from other sources—for example, to a reeking ulcer and to a discharging lesion. In this way Semmelweis broadened what may have been his original hypothesis—childbed fever was due not only to cadaverous poison but also to infection by decaying matter from any source.

A few physicians immediately recognized the practical significance of these results and notified their colleagues throughout Europe. Ferdinand Ritter von Hebra, editor of a major medical periodical and a leading dermatologist, announced the discovery in editorials in December 1847 and in April 1848.⁵⁹ Hebra claimed that the practical significance of Semmelweis's work was comparable to the significance of Jenner's use of cowpox inoculations. Over the next few months Semmelweis and his friends and colleagues sent letters to various obstetricians and to directors of obstetrical clinics; they announced Semmelweis's achievement and requested responses. C. H. G. Routh, who had been a student in the first clinic and who had returned to his home in England, wrote a lecture on Semmelweis's work. The lecture was delivered in November 1848; it was printed in a prominent medical journal and reviewed in several others.⁶⁰ Friedrich Wiegner, another of Semmelweis's students, published a similar essay in Strasbourg.⁶¹

In January 1849 Josef Skoda proposed that the medical faculty

59. Ferdinand Hebra, "Höchst wichtige Erfahrungen über die Aetiologie der an Gebäranstalten epidemischen Puerperalfieber," *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 4 (1847), 1:242–44; and "Fortsetzung der Erfahrungen über die Aetiologie der in Gebäranstalten epidemischen Puerperalfieber," *ibid.*, 5 (1848), 64f. Both editorials, as well as other early documents related to Semmelweis's work, are reprinted in Tiberius von Györy, *Semmelweis' gesammelte Werke* (Jena: Gustav Fischer, 1905).

60. Charles Henry Felix Routh, "On the Causes of the Endemic Puerperal Fever of Vienna," *Medico-surgical Transactions* 32 (1849), 27–40. The lecture was delivered by Edward William Murphy, since Routh was not a fellow of the Royal Medical and Surgical Society, the society to which the lecture was delivered. For a list of the reviews, as well as for bibliographic information about many of the early papers relating to Semmelweis, see Murphy, *op. cit.*, note 2 above, p. 654f.

61. Friedrich Wiegner, "Des moyens prophylactiques mis en usage au grand hôpital de Vienne contre l'apparition de la fièvre puerpérale," *Gazette médicale de Strasbourg* 9 (1849): 99–105.

of Vienna appoint a commission to investigate the practical applicability and the statistical foundations of Semmelweis's work.⁶² Skoda was a brilliant professor of medicine who ultimately became one of the leading members of the Vienna medical school. At this time, however, he was among the younger members of the faculty and was struggling against the unsympathetic conservatism of the firmly entrenched senior members. Skoda's proposal was initially accepted unanimously by the medical faculty. When the commission was named, however, it did not include Johann Klein, head professor of obstetrics, chief of the obstetrical clinic, and Semmelweis's immediate superior. Klein withdrew his support for the proposal. To some extent Klein may have felt threatened by Semmelweis's work. Investigations by the prominent Austrian medical historian, Erna Lesky, however, have shown that broader issues were involved.⁶³ Klein's hostility seems not to have been directed against Semmelweis personally so much as against the younger faculty members in general who were engaged in a power struggle with Klein and his associates. Semmelweis became one focal point in this struggle. Klein interceded with higher administrative authorities, and Skoda's proposal was ultimately overturned. Two months later, in March 1849, Semmelweis was discharged from the first clinic. His attempts to secure an extension of his two-year period of service, supported by Skoda and Rokitansky but opposed by Klein, came to nothing.⁶⁴ During 1849 Semmelweis was denied other appointments that would have enabled him to remain in Vienna and to continue his work.

These months were not without hopeful signs, however. Carl Haller, provisional adjunct director of the Viennese General Hospital, published statistics that supported Semmelweis.⁶⁵ At

62. See below, p. 82; German edition, p. 46.

63. Erna Lesky, *Ignaz Philipp Semmelweis und die Wiener medizinische Schule* (Vienna: Hermann Böhlau, 1964), pp. 11–54.

64. *Ibid.*, pp. 35–51.

65. Haller's paper, which was originally delivered 23 February 1849, appeared as *Ärztlicher Bericht über das k. k. allgemeine Krankenhaus in Wien und die damit vereinigten Anstalten*, *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 5 (1849), 2:535–46: 536–38. The relevant passages are reprinted in Györy, op. cit.,

Skoda's suggestion, Semmelweis undertook animal experiments that provided some confirmation of his views.⁶⁶ In October 1849 Skoda delivered a lecture that was very supportive of the practical significance of Semmelweis's work.⁶⁷ In the following May Semmelweis himself gave a lecture on his discovery that was generally well received.⁶⁸ In the next year a former colleague from the second clinic, Franz Hektor Arneth, gave lectures in Paris and in London.⁶⁹ But by the fall of 1850 Semmelweis seems to have become discouraged at his prospects in Vienna. After the continued hostility of the older faculty members and further frustrations in attempting to secure an appointment, in October 1850 Semmelweis suddenly left Vienna and returned to Budapest. There have been many attempts to explain why Semmelweis abandoned his struggle in this way. The most plausible explanation, the one given by Semmelweis himself and by his friend and colleague Lajos Markusovszky, was that he was simply unable to endure further frustrations in Vienna.⁷⁰ In any case the move was associated with a dramatic change in his relations to

note 59 above, pp. 34f. Semmelweis discusses this report, see below, pp. 72f.; German edition, pp. 280–82.

66. See below, p. 105; German edition, pp. 76–81.

67. Josef Skoda, "Über die von Dr. Semmelweis entdeckte wahre Ursache der in der Wiener Gebäranstalt ungewöhnlich häufig vorkommenden Erkrankungen der Wöchnerinnen und des Mittels zur Verminderung dieser Erkrankungen bis auf die gewöhnliche Zahl," *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 6 (1850), 1:107–17. The lecture was reprinted in Györy, op. cit., note 59 above, pp. 36–45. For bibliographic information regarding some of the reviews and reactions to Skoda's lecture, see Murphy, op. cit., note 2 above, p. 656.

68. The lecture itself was not published, but there were minutes from the lecture and from the following discussions. *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 6 (1850), II:cxxxvii–cxl, clxvi–clxix, and 7 (1851), liiii–x. These are reprinted in Györy, op. cit., note 59 above, pp. 49–58. Eduard Lumpe also wrote a response. "Zur Theorie der Puerperalfieber," *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 6 (1850), 2:392–98.

69. Arneth, op. cit., note 58 above; and "Evidence of Puerperal Fever depending upon the Contagious Inoculation of Morbid Matter," *Monthly Journal of Medical Science* 12 (1851), 505–11.

70. See below, pp. 105f.; German edition, p. 81; and Gortvay and Zoltán, op. cit., note 1 above, pp. 70–73. Erna Lesky gives a different interpretation, op. cit., note 63 above, pp. 62–78.

his former advocates and friends. From that time on Skoda and Rokitansky withdrew much of their support for Semmelweis; apparently neither Skoda nor Rokitansky ever so much as mentioned Semmelweis in their lectures.⁷¹

Over the next several years Semmelweis remained silent on the matter of puerperal fever—as he himself tells us, he expected that the truth and the importance of his work would lead to its ultimate acceptance without further effort on his part.⁷² This did not prove to be the case. Most of the responses to his work were unfavorable; his ideas were consistently misunderstood and misrepresented; and he seems not to have been taken seriously in Vienna or even in Budapest.⁷³ Finally, in 1858, Semmelweis ended a decade of silence with a flurry of publications. In 1858 he delivered a series of lectures before the Medical Society of Pest; these were published later in the same year in an Hungarian medical periodical.⁷⁴ In 1860 he published an essay explaining the difference between his views and those of the British.⁷⁵ In

71. Gortvay and Zoltán, op. cit., note 1 above, p. 72; Georg Silló-Seidl, "Unveröffentlichte und Neuentdeckte Semmelweis-Dokumente," *Orvostörténeti Közlemények* 26 (1978), 187–210; 209. Although, perhaps on the basis of the practical significance of Semmelweis's work, both Skoda and Rokitansky recommended Semmelweis when the time came to choose a replacement for Klein. Lesky, op. cit., note 63 above, pp. 83–93.

72. See below, p. 62; German edition, p. v.

73. Ede Flórián Birly, Professor of Obstetrics at the University of Pest, never accepted Semmelweis's teachings; he continued to believe that puerperal fever was due to uncleanness of the bowel. Gortvay and Zoltán, op. cit., note 1 above, pp. 81f., 122. See below, p. 109; German edition, pp. 136f. In 1856 József Fleischer sent a notice to the *Wiener medizinische Wochenschrift* announcing Semmelweis's success at the clinic of the University of Pest; Leopold Wittelshöfer, who was the editor, remarked sarcastically that it was time people stopped being misled about the theory of chlorine washings. *Wiener medizinische Wochenschrift* 6 (1856), 536.

74. Ignaz Philipp Semmelweis, "A gyermekágyi láz kóroktana," *Orvosi hetilap* 2 (1858), 1–5, 17–21, 65–69, 81–84, 321–26, 337–42, 353–59. Translations of the lectures are reprinted in Györy, op. cit., note 59 above, pp. 61–83.

75. Ignaz Philipp Semmelweis, "A gyermekágyi láz fölötti véleménykülönbség köztem s az angol orvosok közt," *Orvosi hetilap* 4 (1860), 849–51, 873–76, 889–93, 913–15. A translation is reprinted in Györy, op. cit., note 59 above, pp. 83–94.

the same year his main work appeared, *Die Aetiologie, der Begriff, und die Prophylaxis des Kindbettfiebers*.⁷⁶ Unfortunately, everyone seems to have felt that they already knew as much about his opinions as they cared to know; apparently almost no one bothered to read his book.⁷⁷ Few have bothered to read his book since. What one finds there is a crucial conceptual innovation. Semmelweis's argument in the *Aetiologie* repudiates the notion that pathological anatomy is the ultimate foundation of medicine; in its place he employs a new strategy that was destined to become one of the defining characteristics of scientific medicine.

IV

Perhaps the easiest and most natural way to characterize any particular disease is in terms of specific changes in the human body. Thus, at the beginning of the nineteenth century, diseases were generally characterized in terms of abnormal signs and symptoms that were observable under ordinary clinical conditions. For example, one influential characterization of hydrophobia was a "complete horror of fluids reaching to such a degree, that their deglutition becomes almost impossible."⁷⁸ However, these characterizations were vague and it was sometimes impossible to identify reliably the disease from which a patient suffered or had died; for example, it was often impossible to distinguish between hydrophobia and tetanus. Through the

76. The *Aetiologie* was first published by C. A. Hartleben's Verlag in Pest, Vienna, and Leipzig, and bears the date of 1861. Gortvay and Zoltán indicate that the book actually appeared in October in 1860. Op. cit., note 1 above, p. 132. The only English edition to date was a literal translation by Frank P. Murphy that appeared in the periodical *Medical Classics* 5–8 (1941), 339–773. Murphy himself pointed out some of the defects in his translation. Op. cit., note 2 above, p. 706.

77. For example, when the book first appeared, the prominent Viennese obstetrician Joseph Späth dismissed it with the remark that Semmelweis's views had been known for fourteen years. Gortvay and Zoltán, op. cit., note 1 above, p. 141.

78. From a lecture in Paris by Gabriel Andral. The lecture was reprinted under the title "Perversions of Sensibility: Hydrophobia," in *Lancet* 1 (1832), 805–09; 806.

early decades of the nineteenth century there were intense efforts to provide more precise characterizations in terms of internal structural lesions. If each disease could be characterized in this way, then at least in autopsy it would be possible, in principle, to determine precisely the specific disease from which a person had died. The study of these internal changes—pathological anatomy—rose to prominence in France just at the beginning of the century, and through the next decades it dominated medical thought and research throughout Europe.⁷⁹ The rise of pathological anatomy brought important changes; by emphasizing observation and the accumulation of facts, it provided the basis for a positivistic attack on the unrestrained speculative theories of earlier decades. For this reason it is customary to identify the rise of pathological anatomy as the beginning of scientific medicine.⁷⁹ However, pathological anatomy left much of medical theory and practice intact. In particular, pathological anatomy did not alter the basic strategy of characterizing diseases in terms of physical changes in the human body.

As long as diseases are characterized in terms of some physical state—whether in terms of clinical signs and symptoms or in terms of anatomical lesions—it is always possible, in principle, for different cases of one disease to have different causes. Nineteenth-century physicians found this situation entirely acceptable. In standard texts, virtually all diseases were ascribed to various different causes.⁸⁰ For example, physicians recognized that hydrophobia—a horror of fluids—could be caused by certain fevers, by physical disorders in the throat, by emotional or psychological factors, as well as by the bites of rabid dogs.⁸⁰ Characterizations of diseases derived from pathological anatomy, while more precise than those based on the study of symptoms, still allowed for the possibility of a variety of different causes. Thus even late into the nineteenth century, after numerous attempts

79. To take radically different examples, Richard Harrison Shryock, *The Development of Modern Medicine*, reprinted from the 1936 edition (Madison: University of Wisconsin Press, 1979), pp. 151–55, and Michel Foucault, *The Birth of the Clinic* (New York: Pantheon Books, 1973), pp. 146, 197, both identify Xavier Bichat, the French pathological anatomist, as the founder of scientific medicine.

80. Andral, op. cit., note 78 above, p. 806.

to characterize hydrophobia in terms of morbid alterations, fear was still regularly regarded as a possible cause of the disease.⁸¹

Characterizing diseases so that each disease can have various unrelated causes results in serious practical and theoretical limitations, however. First, given such a characterization, it is practically impossible to generate effective techniques for controlling the disease. If a disease such as hydrophobia can be caused in different and essentially unrelated ways, then no single set of prophylactic or therapeutic measures can be relied on to be consistently effective. It is possible that the prevention or treatment of one class of cases of a disease may require procedures exactly opposite to those required in other cases of the same disease. Indeed, the very steps necessary to control one cause of a given disease could themselves constitute a different cause of that same disease.⁸² This makes prevention and treatment so complex and confusing that effective practical medicine is all but impossible. Second, with these characterizations it is very difficult to generate simple and correct causal explanations for the observed facts. Through experience, one may accumulate a wide range of facts about any disease—for example, one may observe certain symptoms and find that these are regularly associated with specific anatomical lesions, that they have a certain course of development, that they spread to other persons in certain patterns, that

81. For example, in 1877 the editors of *Lancet* warned against diagnosing psychogenic or “mental” hydrophobia as true hydrophobia because “just as prophecy has often no small influence on its fulfillment, the diagnosis of hydrophobia conduces to its apparent verification.” Thus, although the editors “doubt whether there are any authenticated instances in which a disease unquestionably emotional in its origin ran a rapid course to a speedy death,” mistaken diagnoses may be self-fulfilling. To help prevent mistakes, the editors point out that whereas in true hydrophobia the convulsive spasms involve “the larynx as well as the pharynx, . . . in mental hydrophobia the spasm is pharyngeal only.” The editors also express the expectation that “microscopical investigation may become of great importance as a test of the accuracy of the diagnosis in doubtful cases.” *Lancet* 2 (1877), 399f.

82. For example, physicians frequently identified both excessive and inadequate diet as causes of the same disorder. It would be possible that the measures adopted to correct one of these factors could cause the disorder by bringing about the other factor.

they occur at certain times and places, and that certain people succumb while others do not. As long as each disease is characterized so that it can have various causes, it is very difficult to construct satisfactory causal explanations for most of the observed facts. For example, assuming that hydrophobia can be caused by fevers, contusions, fear, or dog bites, and that each of these factors can also cause other diseases, how can one possibly explain the relatively fixed course of development of the disease or patterns in its dissemination? Thus, admitting the possibility of a variety of different causes obstructs both the discovery of effective techniques for controlling disease and the development of explanations for the observed facts.

Typically these difficulties were overcome as follows. Beginning with a disease defined symptomatically or in terms of pathological modifications, specific conditions were identified that were causally necessary for many or most of the recognized cases of the disease. The disease was then given a new characterization in terms of that necessary condition. This, of course, entailed reclassifying many particular cases, since the new definition would include some cases with different symptoms (but the same cause), and it would exclude other cases with the same symptoms (but a different cause). These etiological characterizations had both practical and theoretical advantages. Since every case of the newly characterized disease had the same cause, it was possible to develop consistent and reliable techniques for preventing and treating the disease. Moreover, since every case of the disease now had a common necessary cause, it was possible to explain many of the observed facts in terms of that necessary cause. This new strategy first became prominent in work on the infectious diseases, where its application is obvious. But many specific developments in late-nineteenth-century medicine can be understood as applications of this basic strategy in various areas of medicine.⁸³

Semmelweis probably began his work looking for the cause that could explain the difference in mortality between the two clinics. He quickly found it to be decaying cadaverous matter

83. Carter, *op. cit.*, note 11 above.

that was conveyed to patients in various ways. He had shown that preventing the spread of this matter reduced the mortality level to that of the second clinic. He also knew that fifty years earlier, long before physicians in Vienna began to contaminate their hands by performing autopsies, about one percent of the patients died from puerperal fever. His chlorine washings had, therefore, achieved about the best results that he could reasonably expect; the first clinic was averaging about the same mortality rate as the second clinic (where students did not perform autopsies), and about the same rate as both clinics before the adoption of pathological anatomy. Semmelweis could simply have concluded that he had found the cause of the excess mortality in the first clinic and that the residual one percent of cases were due to one or more of the other recognized causes of the disease. This would have been completely compatible with the possibility that the so-called sporadic cases that continued to occur in both clinics were due to various other epidemic or endemic factors. It would, in other words, have been completely compatible with the recognized etiology of childbed fever. Semmelweis did not stop here, however; instead, he took a radical new step—one that he could not justify by any evidence, one that was rejected by almost every person (friend or foe) who responded to his work. Semmelweis insisted that, without exception, every case of childbed fever was due to the resorption of decaying organic matter through the damaged body surfaces. Semmelweis judged that the decaying matter was usually conveyed to patients from outside their bodies. He hypothesized that in about one percent of the patients, the decaying matter was not introduced from external sources but generated internally.⁸⁴

It is not possible to be certain exactly when Semmelweis took this crucial step. He himself did not publicly discuss his results until 1850; with the possible exception of Friedrich Wiegner's,⁸⁵

84. See below, p. 116; German edition, p. 106.

85. Wiegner was an eyewitness to Semmelweis's discovery. In his early paper he discussed decaying organic matter and discounted epidemic atmospheric influences. On the other hand, he did not assert that the disease is always due to decaying organic matter or that other factors are never causes. *Op. cit.*, note 61 above. Moreover, it would be hard to reconcile some later comments by Wiegner

none of the earlier discussions of Semmelweis's work suggest that he claimed to have identified a universal necessary cause for all cases of the disease. Hebra's announcements claimed only that most cases of puerperal fever in the Viennese hospital were due to cadaverous infection.⁸⁶ In his lecture in 1848, Routh said that his remarks applied "especially to Vienna, although it is believed that similar causes are brought into operation in other countries, and might as effectively be combated."⁸⁷ This was a far weaker claim than anything Semmelweis ever published on the subject. Semmelweis and his associates also sent letters to many leading obstetricians announcing his results. One of these letters, written by Heinrich Hermann Schwartz, was ultimately published in a Danish medical periodical; in this letter Schwartz did not imply that all cases of the disease are due to one cause.⁸⁸ Moreover, the few persons who responded to these letters seem not to have understood Semmelweis to be claiming that puerperal fever had a necessary cause. James Young Simpson observed that he saw no difference between Semmelweis and the British on this matter,⁸⁹ and Christian Bernard Tilanus, while claiming to agree with Semmelweis, continued to believe that many cases of puerperal fever were due to epidemic influences.⁹⁰ Skoda's lec-

and by his associates with the idea that every case of the disease is due to decaying matter. See below, pp. 126–28; German edition, pp. 132–34.

86. Hebra, op. cit., note 59 above.

87. Routh, op. cit., note 60 above, p. 27. Routh also says that puerperal fever, "like all other fevers, may be very much modified by epidemic influences" (p. 38). Semmelweis would not have agreed with this.

88. The letter was originally sent to Gustav Adolph Michaelis in Kiel. Michaelis forwarded the letter to Karl Édouard Marius Levy in Copenhagen. Levy published this letter together with a critical response. "De nyeste Forsög i Födselsstiftelsen i Wien til Oplysning om Barselberens Actiologie," *Hospitals-Meddelelser* 1(1848), 199–211.

89. See below, pp. 74f.; German edition, pp. 282–84. As Semmelweis noted, Simpson gradually understood more perfectly what was at issue between himself and Semmelweis; in an essay published in 1850 Simpson refers to Semmelweis positively. Simpson, op. cit., note 22 above, p. 429. However, Simpson continued to believe that some cases of puerperal fever were epidemic and not due to the resorption of decaying animal matter. *Ibid.*, p. 427, and "Medical News," *Monthly Journal of Medical Science* 13(1851), 71–81: 72, 78.

90. See below, pp. 188–90; German edition, pp. 310–13.

ture in October 1849 does not once suggest that Semmelweis purported to have found a necessary cause for all cases of the disease—Skoda describes Semmelweis's work as an attempt to reduce mortality in the first clinic to the normal levels.⁹¹ Indeed, one of the respondents to Semmelweis's subsequent lecture noted quite explicitly that nothing Skoda said precluded the possibility that many cases of the disease were due to the usual epidemic and endemic causes.⁹² Franz Kiwisch von Rotterau of Würzburg and Wilhelm Friedrich Scanzoni and Bernhard Seyfert of the Prague maternity hospital responded to Skoda's lecture mainly by objecting that Skoda had said nothing new. Kiwisch pointed out that the British had long expressed similar views;⁹³ Scanzoni and Seyfert objected only to derogatory insinuations about the Prague hospital and to the suggestion that the excessive mortality in Prague was also due to cadaverous matter.⁹⁴ Because Semmelweis himself published nothing during these months, it is possible either that Semmelweis had not yet concluded that puerperal fever was always due to decaying matter or that, for whatever reason, his associates chose to state and to defend only

91. Skoda, op. cit., note 67 above.

92. Theodor Helm said, in fact, that neither Semmelweis himself nor Skoda in his earlier lecture had excluded the possibility of such other causes as difficult delivery and emotional disturbance, and he suggested that such causes might be responsible for the normal number of deaths while infection from cadaverous poison might cause the extraordinary number of deaths in maternity clinics. This is recorded in the minutes of the discussion of Semmelweis's lecture, op. cit., note 68 above, p. viii. As we will see, Semmelweis had not excluded these possibilities in the sense of having proved that they did not exist; but he had at least denied that there were such causes. Skoda did not even go that far.

93. Franz Kiwisch von Rotterau, "Einige Worte über die vom Prof. Skoda veröffentlichte Entdeckung des Dr. Semmelweis die Entstehung des Puerperalfiebers betreffend," *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 6 (1850), 2: 300–306: 301f.

94. Wilhelm Friedrich Scanzoni, "Über die von Dr. Semmelweis entdeckte, wahre Ursache der in der Wiener Gebäranstalt ungewöhnlich häufig vorkommenden Erkrankungen der Wöchnerinnen und das Mittel zur Verminderung dieser Erkrankungen bis auf die gewöhnliche Zahl," and Bernhard Seyfert, "Ergänzende Bemerkungen zu dem vorstehenden Aufsätze." Both appear in *Vierteljahrsschrift für die praktische Heilkunde* 26 Literarischer Anzeiger (1850), 2:25–33 and 34–36 respectively.

these weaker claims. There is no documentary evidence from the years before 1850 that Semmelweis or his associates believed that he had identified a universal necessary cause.

Semmelweis's May 1850 lecture was not published in full; we know it only from the secretary's minutes, and from the recorded responses of those who subsequently discussed the lecture.⁹⁵ These sources make it quite clear, however, that by May 1850 Semmelweis conceived of his results as applying to every case of puerperal fever.⁹⁶ Moreover, it is very clear from the writings of those who responded to Semmelweis after the 1850 lecture that he, in contrast to Skoda and Routh, was generally understood to have advanced this claim.

Eduard Lumpe, who had preceded Semmelweis as assistant in the first clinic and who knew essentially everything that Semmelweis used to support his claim, responded to Semmelweis's 1850 lecture as follows: "When one thinks how, since the first occurrence of puerperal fever epidemics, observers of all times have sought in vain for its causes and the means of preventing it, Semmelweis's theory takes on the appearance of the egg of Columbus. I was myself originally overjoyed as I heard the fortunate results of the chlorine washing; like everyone else, I too have had the misfortune to witness many blossoming young women fall before this devastating plague. However, during my two years as assistant in the first clinic, I observed incredible variations in the incidence of sicknesses and death. Because of this . . . any other possibility is more plausible than one common and constant cause."⁹⁷ After numerous criticisms of Semmelweis, Lumpe concludes: "If adoption of the washings makes it possible to avoid even the least significant of the many concur-

95. Op. cit., note 68 above.

96. Ibid., pp. cxxxvii–cxl; and cp. also the remarks of Heinrich Herzfelder, the head secretary who recorded the minutes: "Bericht über die Leistungen der k. k. Gesellschaft der Ärzte in Wien während des Jahres 1850," *Zeitschrift der k. k. Gesellschaft der Ärzte zu Wien* 8 (1851), vii, also quoted by Semmelweis, see below, p. 210; German edition, p. 398.

97. Lumpe, op. cit., note 68 above, pp. 392f.; also quoted by Semmelweis, see below, pp. 223f.; German edition, pp. 443, 445.

ring factors that cause puerperal fever, then their initial adoption was a sufficiently large service. However, whether this is in fact the case, only the future will be able to decide. In the meantime, I believe we should wait and wash."⁹⁸ After the May 1850 lecture, Scanzoni no longer objected that Semmelweis had said nothing new; instead he rejected Semmelweis's theory as false. Scanzoni clearly admitted the possibility that puerperal fever could originate in the way that Semmelweis discussed. Yet Scanzoni rejected Semmelweis's views on the grounds that the disease was primarily due to atmospheric or miasmatic influences and that it could sometimes be caused by such other factors as emotional trauma.⁹⁹ Hermann Lebert, Professor at Breslau, wrote: "It is questionable whether those who have died of this disease can have been directly inoculated by poison from corpses. Semmelweis has elevated this possibility into a system. In any case this would be only one of many possibilities of conveyance."¹⁰⁰ Anton Hayne, a veterinarian who claimed priority for Semmelweis's discovery while at the same time rejecting it as false, noted that animals frequently contract a disease corresponding to childbed fever and that this is a consequence of dietary errors, injuries, exposure to cold, and so forth. He wrote that in cases where none of these factors can be identified "the disease can be attributed only to a miasma or to a contagium."¹⁰¹ Paul-Antoine Dubois, whom Semmelweis identified as the foremost French obstetrician, held that while one could not dispense with precautionary measures to guard against contagion, the contagious element is neither as effective nor as pervasive as Semmelweis

98. Lumpe, op. cit., note 68 above, p. 398; also quoted by Semmelweis, see below, p. 225; German edition, p. 454.

99. Wilhelm Friedrich Scanzoni, *Lehrbuch der Geburtshilfe*, 3rd ed. Auflage (Vienna: L. W. Seidel, 1855), vol. 3, p. 1,010; quoted by Semmelweis, see below, p. 209; German edition, p. 396.

100. Hermann Lebert, *Handbuch der praktischen Medicin* (Tübingen: H. Laupp, 1859), vol. 2, pp. 759f.; quoted by Semmelweis, see below, p. 221; German edition, p. 436.

101. Hayne's claim to priority is recorded in the minutes of the discussion of Semmelweis's lecture, op. cit., note 68 above, p. v. His other remarks are quoted by Semmelweis, op. cit., note 76 above, p. 442.

claimed, and that even before delivery, other factors predispose women to the disease.¹⁰² Joseph Hermann Schmidt, Professor of Obstetrics in Berlin, approved of obstetrical students having ready access to morgues in which they could spend time while waiting for the labor process. He asked how Semmelweis's hypothesis could be reconciled with the observation that the disease occurs in relatively few normal deliveries. He then admitted that while the resorption of decaying matter may be "one path that leads to childbed fever, it is certainly not the only one."¹⁰³ D. Everkin of the Paderborn maternity clinic wrote: "I could not imagine that this circumstance is the universal cause, but I was led [by your communication] to avoid undertaking any procedures on maternity patients after examining corpses." He then warned Semmelweis that nowhere is one more frequently tempted with the *post hoc ergo propter hoc* fallacy than in medicine.¹⁰⁴ Carl Braun, Semmelweis's successor as assistant in the first clinic, identified thirty causes of childbed fever; the twenty-eighth of these was cadaverous infection. Others included conception and pregnancy, uremia, pressure exerted on adjacent organs by the shrinking uterus, emotional traumata, mistakes in diet, chilling, and epidemic influences. He too rejected Semmelweis for refusing to admit that these other factors were possible causes.¹⁰⁵ Everyone who responded critically to Semmelweis's 1850 lecture objected to his claim to have found the one necessary cause for all cases of the disease.

We have seen that neither Hebra, Skoda, Routh, nor Tilanus

102. Dubois's remarks are quoted by Semmelweis, *ibid.*, p. 458.

103. Joseph Hermann Schmidt, "Die geburtshülfliche-klinischen Institute der königlichen Charité," *Annalen des charité-Krankenhauses zu Berlin* 1 (1850), 485-523: 501; also quoted by Semmelweis, see below, p. 227; German edition, p. 463.

104. These remarks are from a personal letter Semmelweis received from Everkin; the letter is quoted by Semmelweis, see below, p. 228; German edition, p. 467.

105. Braun's thirty causes appear in *op. cit.*, note 21 above, p. 451, and in his *Lehrbuch der Geburtshilfe* (Vienna: Braumüller, 1857), p. 914. In the first of these, published in 1855, he mentions Semmelweis in connection with his discussion of cause number twenty-eight, cadaverous poisoning. In the later version, however, although he discusses the same cause in the same terms, all references to Semmelweis have been dropped.

seems to have recognized that Semmelweis was making this claim. Remarkably, one finds exactly the same circumstance in the writings of those who endorsed Semmelweis after the 1850 lecture. Justus Liebig included a favorable reference to Semmelweis in his *Chemical Letters*, and Semmelweis spoke of him as a supporter, but Liebig's passage concludes, "certainly other causes of childbed fever will be identified. However, no unprejudiced person can doubt that the one identified so insightfully by Dr. Semmelweis at the maternity hospital in Vienna is among the causes."¹⁰⁶ After 1850 Semmelweis was frequently mentioned favorably by British physicians, but none of them seem to have recognized that Semmelweis was claiming to have found one single cause for every case of the disease.¹⁰⁷ Semmelweis mentions especially Edward William Murphy as sympathetic to his views.¹⁰⁸ Murphy, who delivered Routh's first lecture on Semmelweis, did mention Semmelweis favorably, but he explicitly acknowledged a whole range of causal factors. Two pages before a reference to "the valuable observations of Dr. Semmelweis [sic] on this disease," Murphy notes that "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 influences."¹⁰⁹ Even Franz Hektor Arneth, who spoke on Semmelweis's behalf in the discussions of the May lecture and who did much to promulgate Semmelweis's view in France and in England, wrote nothing to imply that every case of childbed fever was due to one cause.¹¹⁰ In 1861 Ferdinand Adolph Kehrer published a long discussion of childbed fever; he endorsed Semmelweis's prophylactic procedures and

106. Justus Liebig, *Chemische Briefe* (Heidelberg: C. F. Winke, 1851), p. 714; also quoted by Semmelweis, see below, p. 217; German edition, p. 422.

107. For example, Simpson, *op. cit.*, note 22 above, p. 429; Holmes, *op. cit.*, note 24 above, p. 170; Smith, *op. cit.*, note 36 above, p. 504; and Murphy, *op. cit.*, note 47 above, p. 21.

108. See below, p. 175; German edition, p. 285.

109. Murphy, *op. cit.*, note 47 above, p. 19.

110. In the Paris lecture, Arneth said only that decaying organic matter caused the excessive mortality in the first clinic; in neither lecture did he suggest that all cases of the disease were due to this cause. *Op. cit.*, note 58 above, p. 284f.; *op. cit.*, note 69 above.

even commended his work on etiology, but Kehrer still acknowledged the existence of epidemic childbed fever and he warned physicians that overcrowded hospitals, humid weather, and a prevalence of typhoid-like diseases were especially to be feared.¹¹¹ One cannot avoid the impression that there was much more agreement between Semmelweis's critics and his supporters than between Semmelweis and his supporters. The crucial difference between his critics and his supporters was how seriously they took his claim that every case of childbed fever was caused by the resorption of decaying matter. Almost without exception,¹¹² no one believed that he had identified a necessary cause. His contemporaries either failed to see that he claimed to have identified such a cause—in which case they may have thought they agreed with him—or they saw that he made this claim and they rejected it. Perhaps this was the reason why at least two of his critics noted that in all the literature on puerperal fever there was nothing that supported the view Semmelweis was advancing.¹¹³ Semmelweis denied this,¹¹⁴ but in this respect it was certainly true.

In his publications of 1858 and 1860 Semmelweis's views are made fully explicit. In his lectures published in 1858 he asserted without equivocation that every case of childbed fever was due to the resorption of decaying matter.¹¹⁵ His essay explaining the

111. Kehrer, op. cit., note 49 above, pp. 214–16.

112. The most likely possible exceptions are Friedrich Wieger, as mentioned above, and Lajos Markusovszky, who was a longtime friend and colleague of Semmelweis's. Markusovszky defended Semmelweis against various critics. See Gortvay and Zoltán, op. cit., note 1 above, pp. 137–39.

113. Braun, op. cit., note 105 above, p. 921; and H. Silberschmidt quoted in Semmelweis, op. cit., note 76 above, p. 407.

114. See below, p. 170; German edition, p. 275. In the Budapest lectures of 1858 Semmelweis seemed much less concerned to establish the existence of supporting literature than he was in his book. In his lectures he did not refer to Skoda, Hebra, or Haller. Moreover, in the book there are some differences in his treatment of critics. For example, Tilanus claimed in his letter to agree with Semmelweis, but he continued to acknowledge the operation of atmospheric influences as the primary factor in the disease. In his lectures, Semmelweis pointed out the difference in their opinions (Györy, op. cit., note 59 above, p. 68), but in the book he simply quoted Tilanus's letter without comment.

115. Ibid., pp. 70, 78.

difference between himself and the British ends with these comments: "The important difference between my opinion and the opinion of the English physicians consists in this: in every case, without a single exception, I assume only one cause, namely decaying matter, and am convinced of this. The English physicians, while believing that childbed fever can be caused by decaying matter, recognize in addition all the old epidemic and endemic causes that have been believed to play a role in the origin of the disease."¹¹⁶ In the *Aetiologie* Semmelweis says quite explicitly that resorption of decaying matter is a necessary cause for puerperal fever: "In order for childbed fever to occur, it is a *conditio sine qua non* that decaying matter is introduced into the genitals."¹¹⁷ He frequently asserts that every case of childbed fever, without a single exception, comes about in this way.¹¹⁸ Semmelweis also distinguishes quite explicitly between the necessary cause that he had identified and the purportedly sufficient causes that his contemporaries were claiming to have identified. He cites a passage in which Joseph Hamernik specified three conditions that must be satisfied in identifying the cause of any disease: "Does this cause always have the same effect? As an experiment can one always bring about the disease in this way? In those cases in which the cause does not bring about the specified disease, can the same reason for failure always be identified?"¹¹⁹ These criteria are all for causal sufficiency—not one of them is satisfied by a cause that is necessary but not sufficient. Semmelweis rejects the first criterion and gives this as his reason: "I have injected rabbits with decaying matter; some consequently died from pyemia and others did not. Could we deny that the decaying matter was the cause of pyemia in the rabbits that died, simply because the matter did not occasion pyemia in all the rabbits?"¹²⁰ In this passage he is using 'cause' in the sense of necessity. Sem-

116. Ibid., p. 94.

117. See below, p. 149; German edition, p. 196.

118. See below, for example, pp. 109, 114, 120, 141; German edition, pp. 87, 102, 114–16, 179.

119. Joseph Hamernik, *Die Cholera epidemica* (Prague: Calve, 1850), pp. 247f.; also quoted by Semmelweis, see below, p. 216; German edition, p. 418.

120. Ibid.

melweis claims to have fulfilled the second condition by his experiments with rabbits—he ignores Hamernik's stipulation that the disease is always produced in this way. Finally, while admitting that he has not satisfied the third condition, Semmelweis notes that "instead, my etiology of childbed fever satisfies another condition that Hamernik has not posed, but one that constitutes a condition for a true etiology. Namely, I have reduced the disease by making harmless that which I have identified as its cause."¹²¹ This assertion is not totally unambiguous, but taken together with his repeated claims that every case of the disease comes from the resorption of decaying matter, Semmelweis seems clearly to be saying that in any true etiology one must identify that necessary condition whose prevention will eliminate the disease, just as by preventing the resorption of decaying matter Semmelweis himself eliminated childbed fever in all those cases where resorption could be prevented.

Having thus identified a necessary cause for childbed fever, Semmelweis gives a new etiological characterization of the disease; he defines it as "a resorption fever dependent on the resorption of decaying animal-organic matter."¹²² He characterizes pyemia as "disintegration of the blood through decaying animal-organic matter."¹²³ Several important conclusions follow from these definitions. First, it follows that every case of childbed fever is a case of pyemia; in other words, puerperal fever is not a separate species of disease but only a variety of blood poisoning.¹²⁴ Second, it follows that there never had been and never could be a single case of epidemic childbed fever, since atmospheric conditions neither create decaying organic matter nor convey it to potential patients.¹²⁵ Finally, it follows that Kolletschka, the male and female infants, and the puerperae all died from the same disease, and this is true regardless of what pathological remains, if any, may be found in autopsy.

121. Ibid.

122. See below, p. 114; German edition, p. 102; cp. Györy, op. cit., note 59 above, p. 70.

123. See below, p. 117; German edition, p. 106.

124. Ibid.

125. See below, p. 120; German edition, p. 116.

Given this new definition of puerperal fever, it follows trivially that every case of the disease is due to infection by decaying organic matter; but how could this definition (or equivalently the claim that childbed fever has this necessary cause) be justified? There were two kinds of justification for this new approach: one practical, one theoretical. First, the new approach clarified and unified the practical measures that could be used against the disease. There may have been cases in which patients were so terrified at the prospect of delivering in the first clinic that they began to suffer from the symptoms of the very disease they dreaded. Given the earlier symptomatic characterizations of the disease, such patients truly had childbed fever. But preventing these cases would obviously require prophylactic measures quite different from those required to prevent the cases that Semmelweis was considering. By recharacterizing the disease as he did, Semmelweis made it possible to focus attention on cases involving one particular cause, cases that could therefore be prevented by avoiding that one cause. If patients died from something else, say fear, that was lamentable but not his immediate concern. The recharacterization thus made it possible for there to be consistently effective prophylactic measures that, at least in theory, could be used to prevent all but the residual one percent who, Semmelweis believed, would suffer from self-infection. Of course, precisely the same cases of disease could have been controlled by exactly the same measures even if the disease were still defined symptomatically. But as long as the disease was defined symptomatically, those same measures would not work on other cases of the same disease, and the ensuing confusion made it virtually impossible for any effective measures to be identified. The introduction of etiological characterizations was absolutely necessary for the development of systematic medical procedures.

Second, the new characterizations had enormous theoretical advantages, and it is obvious from Semmelweis's book that he found these advantages at least as compelling as the practical advantages. As Semmelweis himself certainly recognized, the strength of his characterization lay in its explanatory power. Semmelweis drew from his account explanations for dozens of

facts that had been observed and recorded but never explained. To choose only a few examples, Semmelweis explained why infants never died from puerperal fever while their mothers remained healthy, why the mortality rates of infants changed in certain ways, why women who delivered on the way to the hospital or who delivered prematurely had a lower mortality rate, why the disease often appeared in particular patterns among patients, why the mortality rate was different in the two clinics and why it had changed in certain ways through history, why infections were rare during pregnancy or after delivery, why the disease appeared to be contagious, why it exhibited seasonal patterns, why the disease was concentrated in teaching hospitals, why some non-teaching hospitals had a much lower mortality rate than others, and why the disease appeared with different frequencies in different countries and in different historical periods.¹²⁶

In this respect the difference between Semmelweis and the others who wrote on the disease, for example Holmes or Lumpe, is the difference between day and night. In their essays Holmes and Lumpe use their accounts of the disease to explain nothing beyond the very facts that suggested those accounts. Thus, while Holmes's account was associated with certain useful practices, neither Holmes's nor Lumpe's account had any real theoretical or scientific interest. By contrast, Semmelweis provides not merely practical advice for avoiding certain cases of puerperal fever, but a complete scientific theory.

V

By the time Semmelweis published the *Aetiologie* his ideas had been widely disseminated and discussed. One disadvantage in the late appearance of his book is that by the time it appeared, everyone had an opinion (usually erroneous) about what Semmelweis believed and few bothered to read the only complete and authoritative exposition of his theory. Semmelweis hoped

126. See below, pp. 99, 100, 101, 115f., 118, 122, 123, 125, 133; German edition, pp. 67, 69, 69f., 70, 104f., 108f., 109, 121f., 123, 125, 145, respectively.

that his book would help to convince his opponents. The book not only failed in this, but also seems not even to have enlightened those who claimed to agree with him.¹²⁷ His reviewers responded to his opinions just as his earlier critics had done. In 1862 Carl S. F. Crede reviewed the *Aetiologie* in the *Monatsschrift für Geburtskunde*. According to Crede, Semmelweis calls everyone who disagrees with him an ignoramus and a murderer. The reviewer writes that Semmelweis's assertions "go too far and are too one-sided. In any case, Semmelweis owes us a proof that only the one etiological condition that he identifies is responsible. Nearly every obstetrician is still of the opinion that a large number of cases of illness remain that originate from a different cause, a cause admittedly yet unknown."¹²⁸ After what was obviously a more careful examination of the *Aetiologie*, August Breisky, an obstetrician in Prague, rejected Semmelweis's book as "naive" and he referred to it as "the Koran of puerperal theology."¹²⁹ Breisky objected that Semmelweis had not proved that puerperal fever and pyemia are identical, and he insisted that other factors beyond decaying organic matter certainly had to be included in the etiology of the disease.¹³⁰ From our point of view it is difficult to see how objections of this kind could still have been raised. In fact, Semmelweis's contemporaries were fully justified in their skepticism.

127. After publication of the *Aetiologie* a few people mentioned Semmelweis favorably, but none of them seem to have accepted his etiological theories. See, for example, Kehrer, op. cit., note 49 above, pp. 212–16, Siebold, op. cit., note 41 above, pp. 346f. The same held true for those who subsequently adopted Semmelweis's views. For example, August Hirsch claimed credit for rediscovering Semmelweis and for recognizing the significance of his work, but Hirsch explicitly denied that decaying organic matter was the only cause of childbed fever. *Handbuch der historisch-geographischen Pathologie* (Erlangen: Enke, 1862–64), vol. 2, pp. 423f. Semmelweis also received a few letters congratulating him on the publication of the *Aetiologie*. Toward the end of his life he published these letters as part of an open response to some of his critics. The letters mention only his practical prophylactic measures. Györy, op. cit., note 59 above, pp. 465–67.

128. Crede's review appeared in *Monatsschrift für Geburtskunde und Frauenkrankheiten* 18 (1862), 406f.: 407.

129. The review appeared in *Vierteljahrsschrift für die praktische Heilkunde* 18 *Literarischer Anzeiger* (1861), 2:1–13. The remarks quoted above appear on p. 1.

130. *Ibid.*, p. 12.

No doubt many factors contributed to the opposition Semmelweis continued to encounter. First, there was the continuing hostility of the senior faculty members in Vienna. This hostility may have been generated by political rather than substantive issues, but it remained real. Carl Braun, Semmelweis's successor in the first clinic, achieved fame and influence both in Vienna and in Budapest by submitting entirely to the unenlightened but orthodox opinions of Johann Klein.¹³¹ Second, there was the fact that Semmelweis was consistently misunderstood and misrepresented. Hebra's first editorial suggested that Semmelweis believed himself to have discovered that cadaverous matter exerted harmful influences even through the uninjured skin of anatomists and surgeons. Routh and Skoda both described Semmelweis's discovery as concerning only cadaverous infection rather than infection from all decaying organic matter. Hebra, Routh, Skoda, and Arneth all suggested that Semmelweis's discovery applied only to the excess of mortality in the first clinic and to other cases that happened to be similar, rather than to every case of puerperal fever. It is possible that these misconceptions represent preliminary opinions that Semmelweis himself espoused in the course of formulating his ultimate views. In any case, by May 1850 Semmelweis held none of these positions; nevertheless his critics continued to argue as though he did. The ensuing confusion certainly retarded both the understanding and the acceptance of Semmelweis's views. Third, Semmelweis's discovery was subject to almost constant dispute regarding priority. The first foreign reaction to his work was a vitriolic letter from James Young Simpson of Edinburgh; Simpson complained that if Semmelweis were familiar with British medical literature he would know that the British had long regarded puerperal fever as contagious and preventable by precisely the methods that Semmelweis was claiming to have discovered.¹³² When Semmelweis first reported his ideas in Vienna, a veterinarian claimed priority for the discovery.¹³³ Even modern expositors continue

131. Gortvay and Zoltán, *op. cit.*, note 1 above, p. 162.

132. See below, p. 174; German edition, pp. 282f.

133. The veterinarian was Anton Hayne, see note 101 above.

to write as though there were questions about Semmelweis's priority.¹³⁴

There were more serious obstacles that prevented acceptance of Semmelweis's doctrines, however. His most important critics understood his claims, and they were not distracted by questions of priority or by political disputes; their objections were more substantial. For one thing, given any of the accepted characterizations of puerperal fever, it was simply false that every case of the disease was due to decaying organic matter.¹³⁵ Everyone had known for years that the disease could be caused by cadaverous infection, but it was also obvious to everyone that many cases of the disease could not have originated in this way. Semmelweis's account would not have been plausible unless it included more than this. Unfortunately, almost all of his evidence related to infection from cadavers. His only basis for assuming that other kinds of decaying matter could also cause the disease was subsequent outbreaks of childbed fever that he attempted to trace to other sources of decaying matter. He noticed, for example, that during October and November 1848, when the mortality rate again increased dramatically, there had been two women in the wards of the first clinic who were suffering from reeking and discharging lesions.¹³⁶ He speculated that the increased mortality rate during those months was due to infection from these sources. But as his critics immediately recognized, there was virtually no evidence for this interpretation.¹³⁷ Remarkably enough, neither

134. Since the early decades of the twentieth century there have been interminable discussions concerning the relative priority of Semmelweis and Oliver Wendell Holmes; the discussions suffer from an acute lack of firsthand knowledge of Semmelweis's writings. For some of the earlier references see Murphy, *op. cit.*, note 2 above, pp. 688–707.

135. As the disease was then characterized it certainly included (what would now be regarded as) a variety of inflammations and fevers, and probably did not have a necessary cause. See Smith, *op. cit.*, note 36 above, p. 503; and Kiwisch, *op. cit.*, note 23 above, pp. 600f.

136. See below, p. 93; German edition, pp. 59f.

137. Levy pointed this out in 1848 after first being notified of the discovery, *op. cit.*, note 88 above, pp. 204–6; also quoted by Semmelweis, see below, pp. 181, 185; German edition, pp. 294, 301. Breisky developed the objection more fully, *op. cit.*, note 129 above, pp. 6f.

of the women who supposedly infected the other women in the ward contracted puerperal fever. It seemed highly unlikely that a woman with a discharging lesion in her own uterus could have avoided childbed fever if her discharge was at the same time infecting the women around her.¹³⁸ These cases cast serious doubt on Semmelweis's hypothesis that decaying matter from persons who did not themselves have childbed fever could cause the disease in healthy persons, and also, therefore, on his claim that chlorine washing was responsible for the decline in mortality. As his critics pointed out, it seemed much more reasonable to ascribe the variations in mortality to changes in epidemic influences or in other unknown endemic factors. Since these cases were the only justification Semmelweis could give for believing in sources of decaying matter other than cadavers, his whole scheme must have seemed speculative and dubious. This may be the reason why even his closest associates continued to speak of cadaverous infection rather than accepting his notion that the disease could be caused by infection from other kinds of decaying organic matter.

There was another related defect in Semmelweis's argument. Even if one granted the possibility that the disease could arise through infection from these other sources, it was obvious to everyone that other cases of disease would still remain for which there simply was no infection from any outside source. In order to maintain his thesis that every case of the disease was due to infection by decaying matter, Semmelweis hypothesized that in these cases decaying matter was generated internally. While one could easily imagine that this might happen, for example in a difficult delivery, Semmelweis was unable to present the slightest empirical evidence of its actual occurrence. Indeed, he admitted that because of the inadequate and unsanitary conditions in which he was forced to work, it was not possible to collect any evidence about self-infection.¹³⁹ Thus the assumption that the disease could come about in this way was entirely gratuitous; it was neither more nor less justified than the equally gratuitous

138. *Ibid.*, pp. 6f., 11.

✓ 139. See below, p. 118; German edition, pp. 109f.

assumption of other epidemic or endemic causal factors. There was certainly no basis for Semmelweis's further assumption that self-infection accounted for every case of childbed fever that was not caused by the introduction of decaying organic matter from external sources. To all appearances this assumption was simply a means of protecting his characterization of childbed fever from empirical counter evidence. In spite of his pages of tables, therefore, Semmelweis actually had remarkably little evidence for anything beyond the possibility of cadaverous infection—a possibility that nearly everyone accepted without question. Several of Semmelweis's critics recognized, more or less clearly, that his whole approach rested on changing the meaning of terms—on what must have appeared to be nothing more than a linguistic subterfuge. From their point of view, Lumpe and Karl Levy were exactly correct in objecting (respectively) that Semmelweis had created an egg of Columbus and that his argument rested as much on unstated a priori assumptions as on facts.¹⁴⁰ To them his whole approach seemed narrow-minded, wrong, and pointless. Moreover, by basing his conclusions on new definitions and on a priori assumptions, Semmelweis seemed to be reverting to the speculative theories of earlier decades that were so repugnant to Semmelweis's positivistic contemporaries. Semmelweis's critics objected that he was merely creating a new system in place of the older ones.¹⁴¹

Finally, Semmelweis's doctrine repudiated pathological anatomy—the very foundation of early nineteenth-century medicine. Semmelweis claimed that Kolletschka, the male and female infants, and the puerperae all died from the same disease. He supported this claim by observing that the pathological remains in all these cases were similar. Given the contemporary commitment to pathological anatomy, this was probably the only kind

140. Lumpe, *op. cit.*, note 68 above, p. 392; also quoted by Semmelweis, see below, p. 223; German edition, p. 443. Levy, *op. cit.*, note 88 above, p. 204; also quoted by Semmelweis, see below, p. 181; German edition, p. 293.

✓ 141. Lebert, *op. cit.*, note 100 above, pp. 759f.; also quoted by Semmelweis, see below, p. 221; German edition, p. 436. Levy also suggests this, *op. cit.*, note 88 above, p. 209; also quoted by Semmelweis, see below, p. 184; German edition, p. 301.

of argument that would have been persuasive, but because he argued in this way, it is easy to overlook the originality of this position. In fact, perhaps to make his position appear more acceptable, Semmelweis seems to minimize the differences between his findings and those of the pathological anatomists. For example, he pointed out that he was not the only one to observe that infants of either sex could contract puerperal fever;¹⁴² but in this respect he was definitely in the minority—almost no one else spoke in this way. Moreover, while at least one British physician suggested that surgeons died from a disease that was similar to puerperal fever,¹⁴³ only Semmelweis maintained that the diseases were identical. Lumpe, like most pathological anatomists, found it incredible that the cases should even be referred to as similar.¹⁴⁴ Lumpe had good reason to feel this way; Semmelweis himself admitted that the pathological remains in Kolletschka and in the other surgeons were not identical to those in the maternity patients—the remains were, in fact, totally different in the genital area, in the one area where all efforts to characterize puerperal fever necessarily focused.¹⁴⁵ Indeed, all existing characterizations of childbed fever referred either to the puerperal state or to certain morbid alterations in the uterus (and

142. See below, p. 77; German edition, p. 40. The French called attention to infantile puerperal fever in 1855, and their discussions were reviewed in *Monatsschrift der Geburtshilfe* 7 (1856), 152f., and in the *Wiener medizinische Wochenschrift* 6 (1856), Journal Review, no. 3, p. 22f. I have not been able to identify this claim in any publications before Semmelweis's work in 1847. This must at least raise the possibility that this recognition was not part of Semmelweis's original thinking.

143. The British sometimes claimed that puerperal fever caused (and could be caused by) a poison that could also cause (and be caused by) such other diseases as typhoid and erysipelas, and these, of course, affected males as well as females. See, for example, Robert Storres, "On the Contagious Effects of Puerperal Fever on the Male Subject," *Provincial Medical and Surgical Journal* 19 (1845), 289–94; 290. For a similar opinion, see below, p. 145; German edition, p. 188.

144. Lumpe, op. cit., note 21 above, p. 348. Lumpe cites an essay by Matthew Gibson to justify his claim that the British believed men could contract a disease similar to puerperal fever. In fact, Gibson's essay isn't about puerperal fever and he doesn't even mention the disease by name. "Epidemic Fever," *Lancet* 1 (1843): 330–34.

145. See below, pp. 77, 117; German edition, pp. 40, 107.

usually to both). These characterizations excluded infants and adult males by definition. Moreover, as we have seen, no clear patterns emerged in autopsies of victims of puerperal fever; some corpses revealed no morbid alterations whatsoever, and the alterations that were detected in others varied enormously from case to case. The pathological remains did not warrant classifying even all the cases of puerperal fever together, and the pathological anatomists did not do so.¹⁴⁶ To assimilate all these cases and then to include the infants and the anatomists as well was to go directly counter to the basic principles of pathological anatomy.¹⁴⁷

By now it is obvious by which principle Semmelweis was guided in his reevaluation of the evidence. It is possible that in the context of discovering the necessary cause of childbed fever, Semmelweis first noted a similarity in the pathological remains, he then inferred that the diseases were identical, and he concluded that the puerperae died because of infection by decaying matter. In the context of expounding and justifying his theory as a whole, however, he reasoned in precisely the opposite direction. If he had still taken pathological anatomy as fundamental, he could never have concluded that Kolletschka, the infants, and the puerperae all died from the same disease because they had different pathological remains. Given his etiological definitions, however, these differences could be disregarded as irrelevant. By the time he wrote his book, Semmelweis knew that the diseases were the same because they had the same causes. Moreover, Semmelweis had good reason to have more confidence in his etiological characterizations than in pathological anatomy. First, pathological anatomy could not prevent childbed fever—in a sense it caused the disease. The etiological characterizations made it possible to prevent almost every case of the disease. Second, while pathological anatomy could explain almost nothing, the new characterizations seemed to explain everything; they even provided a criterion for deciding which pathological remains were significant and which were not.

146. See above, note 21.

147. Breisky pointed this out, op. cit., note 129 above, p. 11.

In the face of these very fundamental objections, however, Semmelweis's critics were simply unable to accept his extravagant claims about childbed fever. Thus while most of them seemed perfectly willing to experiment with the practical prophylactic measures that Semmelweis recommended, virtually everyone either ignored or rejected his basic theoretical innovations. The practical and theoretical advantages of the etiological approach to childbed fever were not yet sufficiently obvious to warrant abandoning an entire medical system. This could come about only when subsequent work made it clear that the same new approach yielded similar advantages when applied to a whole range of different diseases.

VI

Because Semmelweis's theoretical strategy was so crucial in his own work and because the same strategy was so important to the subsequent development of medicine, it is interesting to consider the possibility that he may have drawn the innovations in that strategy from the thinking of his professors in the Vienna medical school. In her excellent monograph *The Vienna Medical School in the Nineteenth Century* and in an independent essay, Erna Lesky claims that Semmelweis "absorbed Rokitansky's and Skoda's methods of reasoning and investigation in a synthesis which brought forth revolutionizing new results."¹⁴⁸ Thus Lesky believes that Semmelweis obtained new results from the methods he learned from his professors. There is no doubt that Semmelweis was profoundly influenced by what he learned in Vienna; for example, as Lesky observes, his unremitting use of *modus tollendo ponens* may well have come from Skoda. Semmelweis's extensive use of statistics was also characteristic of the Vienna School and may have been learned there. For other crucial and decisive aspects of his method the case is less clear, however. Lesky claims that the training Semmelweis received in pathological anatomy while working with Rokitansky enabled him to

148. Lesky, op. cit., note 56 above, p. 183; and "Semmelweis: Legende und Historie," *Deutsche medizinische Wochenschrift* 97 (1972), 627-32: 629.

recognize that the anatomical findings in the patients and their infants were identical with those in Kolletschka, and so to conclude that childbed fever, previously thought of as a unique disease, was merely a variety of pyemia.¹⁴⁹ But neither Rokitansky himself nor any of his other students or associates—including Lumpe, Johann Baptist Chiarì, and Eduard Mikschik—recognized the findings as identical.¹⁵⁰ The French and the British, not the Viennese, stressed the similarity between the pathological remains in maternity patients and in infants and in victims of pyemia (respectively). Lumpe, who was also an assistant in the first clinic, expressed astonishment at the British idea that men could contract a disease similar to puerperal fever.¹⁵¹ Moreover, as we have seen, recognizing the similarity of the pathological remains required a reevaluation of the evidence and, to some extent, a repudiation of the basic principles of pathological anatomy. It is surely misleading to ascribe this aspect of Semmelweis's thought simply to his training in Vienna.

Lesky also claims that the Vienna school "provided Semmelweis with the intellectual tools to infer a single unified cause from identical anatomical remains."¹⁵² This claim obviously relates more directly to what I have identified as a crucial step in Semmelweis's thought. Unfortunately, Lesky provides no support for this claim; she goes on immediately to point out that Semmelweis learned from Skoda the so-called method of exclusion, and that in seeking the cause of the difference in mortality between the clinics, he took this procedure out of its original symptomatic-diagnostic context and applied it in a new and original way.¹⁵³ It seems very possible that Semmelweis's systematic exclusion of one possible endemic cause after another was an application of Skoda's method; but that is something quite different from the quest for a necessary cause common to all cases of an illness. Semmelweis seems to have been among the first to conceive of puerperal fever in a way such that it would

149. Ibid.

150. Lesky, op. cit., note 56 above, p. 185.

151. Lumpe, op. cit., note 21 above, p. 348.

152. Lesky, op. cit., note 148 above, p. 629.

153. Ibid.

have a necessary cause—all of his contemporaries seem to have been thinking in altogether different ways. It would be useful to know whether Skoda ever unambiguously asserted of any disease that it must have a necessary cause; he certainly did not make such an assertion for puerperal fever. In his first lecture announcing Semmelweis's results, he said that Semmelweis had discovered the cause of the unusually high incidence of the disease among the patients in the first clinic and the means of reducing this to the usual number. He also noted that the difference in mortality between the clinics precluded any thought that the generation of the sickness was the direct operation of an epidemic cause, and he concluded that, common opinion notwithstanding, the high incidence of disease in the maternity clinic could not be thought of as an epidemic.¹⁵⁴ These assertions did not preclude the possibility that various causal factors were responsible for the usual incidence of puerperal fever or that epidemic puerperal fever sometimes occurred. As we have seen, Theodor Helm, who heard Skoda's lecture, pointed out in a later discussion that nothing Skoda said excluded the possibility that such factors as difficult delivery or emotional trauma could also cause puerperal fever.¹⁵⁵ In the years immediately after Semmelweis's work, Skoda wrote official documents on Semmelweis's behalf. In these documents he is consistently ambiguous in describing Semmelweis's views; he consistently speaks of the causes (plural) of childbed fever, and he never describes Semmelweis's work as a quest for the one necessary cause.¹⁵⁶ By contrast, during the same period Johann Klein, who rejected Semmelweis's opinions, consistently described Semmelweis's work as a quest for the cause (singular) of childbed fever.¹⁵⁷ There is also the

154. Györy, op. cit., note 59 above, p. 36.

155. See above, note 92.

156. Lesky, op. cit., note 63 above, pp. 21, 26. One of these documents explicitly asserts that Semmelweis was "not seeking to explain all the causes of puerperal fever, but only to find and to circumvent the causes of the excessive mortality in the first clinic." (p. 27).

157. *Ibid.*, pp. 29, 43, 46. The contrast between Skoda and Klein may have been like the contrast between Semmelweis's other supporters and critics—the former did not take seriously his claim to have discovered a universal necessary cause, the latter admitted that he made this claim but rejected it.

interesting fact that long after Semmelweis had rejected the idea that corpses were the only source of decaying matter which could cause puerperal fever, Skoda continued to speak only of cadaverous poisoning.¹⁵⁸ Finally, we must recall that Skoda did not speak in Semmelweis's behalf in the discussions of Semmelweis's May 1850 lecture—the lecture in which Semmelweis first unequivocally claimed to have found the one necessary cause for puerperal fever. Skoda's silence can be interpreted in different ways; one possibility is that he was simply unable to accept this aspect of Semmelweis's work but, perhaps because of personal loyalty to Semmelweis, preferred not to make his disagreement explicit. Even in lectures on puerperal diseases given much later, Skoda did not assert that all cases of puerperal fever could be ascribed to a single, necessary cause.¹⁵⁹ Of course, we know that Skoda accepted Semmelweis's discovery that the high incidence of childbed fever in the first clinic was caused by the unclean hands of the medical staff and that chlorine washings could reduce the incidence of disease to the "normal" level. But there is no clear evidence that Skoda ever understood, agreed with, or shared Semmelweis's interest in a single necessary cause for all cases of the disease, or Semmelweis's strategy of recharacterizing the disease in terms of such a cause. Since this particular strategy was the crucial step in Semmelweis's work, his achievement certainly cannot be thought of simply as a new application of Rokitansky's and Skoda's methods of reasoning. Lesky and others have claimed that Skoda and Rokitansky were the "intellectual fathers of [Semmelweis's] discovery."¹⁶⁰ In fact Skoda and

158. Thus in his lecture given in 1849 Skoda spoke only of cadaverous poison, op. cit., note 67 above, while Hebra's announcement made two years earlier, op. cit., note 59 above, Schwartz's 1847 letter to Michaelis, op. cit., note 88 above, and even Wiegner's 1849 essay, op. cit., note 61 above, all assert quite clearly that other sources of decaying matter were involved. This cannot have been a simple oversight on Skoda's part.

159. Josef Skoda, "Über Krankheiten bei Puerpern," *Allgemeine Wiener medizinische Zeitung* 3 (1858), 20:1 and 21:1. Skoda does assert that puerperal fever is a kind of pyemia—he also said this in his 1849 lecture—but this is not conclusive unless one can show that he also ascribed pyemia to a single necessary cause.

160. Lesky says this, op. cit., note 56 above, p. 186. In a recent paper, Sherwin B. Nuland says the same thing: "Skoda and Rokitansky both recognized that the

Rokitansky probably regarded Semmelweis's logic as an incomprehensible and illegitimate mutation (as did everyone else), and while it may have borne a superficial resemblance to their own progeny, they neither could nor probably would have claimed any role in its paternity.

It is generally agreed that in later years neither Skoda nor Rokitansky ever mentioned Semmelweis in lectures or published works.¹⁶¹ This curiosity is usually explained as a consequence of Semmelweis's abrupt departure from Vienna in 1850; Skoda and Rokitansky were, presumably, offended by his ingratitude and by his unwillingness to persist in seeking the recognition that they had consistently sought on his behalf.¹⁶² But surely this explanation is inadequate. Even in lectures on puerperal fever Skoda not only failed to mention Semmelweis—he did not even adopt Semmelweis's most important theoretical innovation. It is hard to believe that a personal affront could have led Skoda to ignore something in these professional matters if he knew that it was true. The most plausible interpretation is that Skoda and Rokitansky, like everyone else, failed to see the value and justification for Semmelweis's innovation. They probably believed that associating themselves with a person who made such patently false and unjustifiable claims could only compro-

puerperal fever discovery was a logical outcome of their own teachings in the new methods of scientific logic. Erna Lesky . . . states that not only were these two rising giants the supporters of Semmelweis, but the 'intellectual fathers of his discovery.'" *Op. cit.*, note 17 above, p. 257. Neither Nuland nor Lesky bothers to explain how Skoda and Rokitansky could recognize Semmelweis's theory as a logical outcome of their own teachings and at the same time fail to accept it.

161. See above, note 71.

162. This is the universal interpretation; Gortvay and Zoltán (*ibid.*, pp. 72f.) and Lesky and Nuland (*Nuland, op. cit.*, note 17 above, pp. 264f.) even agree on this. It is also part of the legend that "the Semmelweis theory of puerperal fever stood on the verge of acceptance" when Semmelweis suddenly abandoned the fight and returned to Budapest. *Ibid.*, p. 264. The chlorine washings may have stood on the verge of acceptance, but there is no evidence whatsoever that anyone in Vienna ever understood the theory, much less was ready to accept it. Skoda, for example, still hadn't accepted the theory eight years later in 1858. See above, notes 158 and 159. The problem here, as with the endless debates regarding Semmelweis's priority, is that no one bothers to determine what the theory in question is, before deciding that Semmelweis didn't originate it or that the Viennese were on the verge of accepting it.

mise their own professional credibility. Thus they had to remain silent or reject that which they regarded as false. Had they entered into the discussion of 1850 or had they referred to his works in later years, they would have been obliged to reject those aspects of his work that they found objectionable. Since they seem never to have accepted his central idea that the disease could be recharacterized in terms of a necessary cause, if they had responded to his work they would probably have ended up saying the same things that Scanzoni, Braun, Lumpe, and all the others said. Perhaps they preferred simply to remain silent.

VII

It is difficult to determine precisely what impact Semmelweis had on his contemporaries. His use of chlorine was probably more influential, even during his lifetime, than he himself realized. Some of those who wrote to Semmelweis observed that his practices were influencing physicians throughout Europe, but that no one was willing to admit openly that this was the case.¹⁶³ If true, this was almost certainly because the practical results of Semmelweis's work continued to become progressively more obvious, while the theoretical basis for those results continued to appear speculative and false.

Because Joseph Lister's antiseptic procedures proved to be so famous and so successful, it would be of interest to determine whether he was influenced by Semmelweis. As early as 1894 Lister was quoted as having said, "Without Semmelweis I could not have succeeded, modern surgery owes much to that great Hungarian."¹⁶⁴ But Lister himself denied having made this statement, and intensive investigation suggests that the statement is probably spurious.¹⁶⁵ Lister's own account of his relation to Semmelweis is contained in a letter that was written on 2 April 1906: "When in 1865 I applied the principles of antiseptics to the treatment of wounds the name of Semmelweis was not yet familiar to me, and I confess, I had not even heard of his work.

163. See below, p. 186; German edition, pp. 306f.

164. Gortvay and Zoltán, *op. cit.*, note 1 above, p. 220.

165. *Ibid.*, pp. 220–22.

When twenty years later I visited Budapest, I was received with the warmest welcome both by colleagues and university students, and even at that occasion the name of Semmelweis was not mentioned, he seems to have been forgotten by his native country to the same extent as by everybody else throughout the world. It was much later that a Hungarian physician in London of the name of Dr. [Theodor] Duka, had called my attention to Semmelweis and his work. Although Semmelweis never influenced my own work, I think with the greatest admiration of him and his achievement and it fills me with joy that at last he is given the respect due to him.¹⁶⁶ There is a good possibility that Lister did hear of Semmelweis, however; at least he had several opportunities to do so. First, while Lister was a student, Semmelweis was often mentioned in British medical literature.¹⁶⁷ Furthermore, M. J. Duncan, who was Simpson's assistant and who may have persuaded Simpson to adopt Semmelweis's methods, had intimate ties with Lister.¹⁶⁸ When Lister married in 1856, he and his wife spent two weeks in Vienna. At that time they were guests in Rokitansky's home, and Rokitansky spent over three hours showing Lister his medical museum.¹⁶⁹ This was just about the time that Rokitansky and others in Vienna were recommending Semmelweis as the successor to Johann Klein, who had died in the same year.¹⁷⁰ Second, Lister spent three days in Budapest in 1883; by this time he had become famous for his work on antiseptics. Lister spent one long evening at a large public gathering at which Dr. Lajos Markusovszky presided.¹⁷¹ Markusovszky, a lifelong friend and colleague of Semmelweis's, may have been among the few persons in Europe

166. *Ibid.*, pp. 220f.

167. See above, note 107.

168. Gortvay and Zoltán, *op. cit.*, note 1 above, p. 223.

169. Owen H. Wangensteen, "Nineteenth-Century Wound Management of the Parturient Uterus and Compound Fracture: The Semmelweis-Lister Priority Controversy." *Bulletin of the New York Academy of Medicine* 46 (1970), 464-596: 586.

170. Lesky, *op. cit.*, note 63 above, pp. 83-93.

171. Owen H. Wangensteen and Sarah D. Wangensteen, "Lister's Books and the Evolution of Antiseptic Wound Practices," *Bulletin of the History of Medicine* 48 (1974), 106-132: 120f.

who understood and agreed with Semmelweis's theoretical approach in dealing with puerperal fever. Owen H. and Sarah D. Wangensteen called attention to a copy of Semmelweis's *Aetiologie* bearing the signature of Lajos Markusovszky that is now in the library of the Wellcome Institute in London. They suggest that this book may have been sent to Lister sometime after his visit to Budapest.¹⁷² It would have been very natural for Semmelweis to have been mentioned in either of these contexts.

In 1896 Lister finally abandoned his earlier procedures that involved wound irrigation and adopted instead prophylactic antiseptic practices—basically the same practices that Semmelweis had advocated. The Wangensteens conclude that Lister certainly knew and was influenced by the work of Semmelweis in making this change.¹⁷³

It would also be significant if one could show that Semmelweis's theoretical approach influenced subsequent work in medical theory. It is generally agreed that the move toward etiologic characterizations of specific diseases was one facet of a nineteenth-century revolution in medical thought. This revolution has generally been associated with bacteriology. After noting that the work of Koch and Pasteur enabled physicians to adopt new characterizations of diseases in terms of specific microorganisms rather than in terms of morbid anatomical modifications, Owsei Temkin writes, "Although bacteriology concerned infectious diseases only, its influence on the general concept of disease was great. Presumably, diseases could be bound to definite causes; hence the knowledge of the cause was needed to elevate a clinical entity or a syndrome to the rank of a disease."¹⁷⁴ As we have seen, this approach existed in medicine quite apart from any serious interest in microorganisms. In fact, however, neither Koch nor Pasteur ever mentioned Semmelweis in their published works. There seem to be only tenuous and indirect connections between Semmelweis and either of them.¹⁷⁵

172. *Ibid.*, pp. 119f.

173. *Ibid.*, p. 124.

174. Owsei Temkin, "Health and Disease," reprinted in *The Double Face of Janus* (Baltimore: Johns Hopkins University Press, 1977), p. 436.

175. For example, in his discussion of the etiology of infected wound diseases,

VIII

After returning to Budapest in 1850 Semmelweis was appointed as an unpaid director of the obstetrical clinic at the St. Rochus hospital. At that time the clinic was seriously troubled with childbed fever. Semmelweis achieved results similar to those achieved in the first clinic in Vienna. Even in Budapest, however, many of his colleagues seem not to have been persuaded by his success. Ede Flórián Birly, who was Professor of Obstetrics at the University of Pest while Semmelweis was working at the St. Rochus hospital, never adopted Semmelweis's methods, and when Birly died and the medical faculty was seeking his successor, Semmelweis received fewer votes than did Carl Braun, Semmelweis's arch rival and antagonist.¹⁷⁶

In 1857 Semmelweis married Mária Weidinhoffer (1837–1910), a daughter of a successful merchant in Pest. In the same year they were married, he received a call to become professor of obstetrics at the University of Zurich,¹⁷⁷ but decided to remain in Budapest. During these years Semmelweis was active in various projects. In addition to his publications on puerperal fever, he helped to found and also contributed several articles to a Hungarian medical periodical. He began writing a handbook of obstetrics. He was also active on various university committees, and functioned as the economic superintendent of the medical

Robert Koch gives considerable attention to puerperal fever. He bases this part of the discussion on Wilhelm Waldeyer. Robert Koch, "Untersuchungen über die Aetiologie der Wundinfektionskrankheiten," reprinted in Julius Schwalbe (ed.), *Gesammelte Werke von Robert Koch* (Leipzig: Georg Thieme, 1912), vol. 1, pp. 61–108. Waldeyer, in turn, cites Karl Meyrhofer. "Über das Vorkommen von Bacterien bei der diphtheritischen Form des Puerperalfiebers," *Archiv für Gynaekologie* 3 (1872), 293–306: 294. Meyrhofer wrote a series of papers between 1863 and 1865 in which he argued that a specific form of microorganism constituted a necessary cause for puerperal fever. The most complete paper was "Zur Frage nach der Aetiologie der Puerperalprocesse," *Monatsschrift für Geburtskunde und Frauenkrankheiten* 25 (1865), 112–34. At the time he wrote these papers, Meyrhofer was an assistant in the Viennese Obstetrical Clinic, and he must have known of Semmelweis. Meyrhofer, however, never mentioned Semmelweis in his papers. This may have been because at this time Carl Braun was Meyrhofer's chief.

176. Gortvay and Zoltán, op. cit., note 1 above, pp. 91–93.

177. Ibid., pp. 100f.

faculty. When his writings did not convince the medical world, he also wrote a series of open letters to various prominent obstetricians.¹⁷⁸ The open letters were highly polemical and superlatively offensive; they probably did little to persuade those to whom they were addressed. By the time of their publication, however, Semmelweis's prophylactic measures were probably beginning to influence medical thought rather significantly.¹⁷⁹

Beginning about 1861 Semmelweis suffered from nervous complaints. As time progressed he became irritable and was easily excitable; he suffered from severe depression and he became excessively absentminded. Paintings of Semmelweis over the years from 1857 to 1864 show that he aged very rapidly.¹⁸⁰ In July 1865 it was his duty to read a report in a faculty meeting. Semmelweis's former assistant József Fleischer relates that when he was called on to do so "he rose, took a piece of paper from his trouser pocket and, to the stupefaction of those present, began to read the text of the midwives' oath."¹⁸¹ There was no doubt about his condition, and his colleagues took him home. "This was the last function he performed in his faculty, which had lost the most accomplished, and the most illustrious of its professors."¹⁸²

Semmelweis was taken by train to Vienna where he was met by Professor Hebra. He was taken to the Lower-Austrian Mental Home and was confined to the ward for maniacs. The following day his wife visited the home to see him, but she was told that the night before Semmelweis had tried to get out and that it had required six attendants to hold him back. She was not allowed to see him. Within two weeks, on 13 August 1865, Semmelweis died.

Much has been written about the nature of Semmelweis's ill-

178. The letters are reprinted in Györy, op. cit., note 59 above, pp. 429–511.

179. Gortvay and Zoltán, op. cit., note 1 above, pp. 170–72. By 1864 Josef Späth, who had been among the outspoken critics of Semmelweis's ideas endorsed at least the practical measures suggested by those ideas. Murphy, op. cit., note 2 above, pp. 669f.

180. Gortvay and Zoltán, op. cit., note 1 above, pp. 6, 183–86.

181. Ibid., p. 187.

182. Ibid.

ness and about the cause of his death. In particular, it is usually reported that he died from pyemia, possibly because of a wound incurred during an autopsy just before his breakdown. After a review of the evidence, Sherwin B. Nuland recently concluded that Semmelweis probably suffered from Alzheimer's disease—a form of presenile dementia, the most prominent characteristics of which are deterioration of intellect, failure of memory, and a striking appearance of rapid aging.¹⁸³ Nuland also concludes that Semmelweis died of wounds inflicted when he was forcibly restrained by the attendants at the mental home—that he was, in effect, beaten to death.

Semmelweis's remains were originally buried in Vienna, but in 1891 they were transferred to Budapest. On 11 October 1964 they were transferred once more to a space in the garden wall of the house in which he was born. The house, in the meantime, had been converted into an historical museum and library, a monument to Ignaz Semmelweis, one of Hungary's most brilliant sons.

183. Nuland, *op. cit.*, note 17, p. 270.