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SEMMELWEIS AND HIS PREDECESSORS

by

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IGNAZ SEMMELWEIS is generally known for identifying puerperal fever as an infectious disease preventable by aseptic methods. While no-one today would contest the importance, the practicality, or the independence of his contribution, its originality has been disputed from the beginning. When Semmelweis first reported his ideas in Vienna, a veterinarian claimed priority for the discovery.[1] 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 recommended.[2] Since the early decades of the twentieth century there have been intermittent debates concerning the relative priority of Semmelweis and Oliver Wendell Holmes.[3] Recently it was claimed that a Finnish physician preceded Semmelweis in his discovery.[4] In fact, there is a basic methodological distinction between Semmelweis and all these figures. Semmelweis employed a strategy that was later fundamental to many important developments in medicine; his use of that strategy connects his work with modern medicine and makes him a contributor to our current research tradition in a way that the others simply were not. Semmelweis may not have had a perfect understanding of his own method, and it is possible that the method was anticipated by earlier writers. But recognizing his method is absolutely essential to appreciating his place in the history of medicine, and so far as I can determine, his most important methodological contributions have been totally ignored in the numerous discussions of his work.

I

Preventive medicine is generally concerned with identifying necessary causes for

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[1]The veterinarian was Anton Hayne see 'Protokol der allgemeinen Versammlung der k. k. Gesellschaft der Aerzte zu Wien, vom 15 Juli 1850', *Zt. k. k. Ges. Aerzte Wien*, 1851, **7**: iii–ix; reprinted in Tiberius von Györy, *Semmelweis' gesammelte Werke*, Jena, Fischer, 1905, pp. 54–58, p. 55. Hayne, like several other early critics, claimed priority for Semmelweis's discovery while at the same time rejecting it as false.

[2]Ignaz Philipp Semmelweis, *Die Aetiologie, der Begriff und die Prophylaxis des Kindbettfiebers*, reprinted from the 1861 ed., New York and London, Johnson Reprint Corporation, 1966, p. 282.

[3]For the references see Frank P. Murphy, 'Ignaz Philipp Semmelweis (1818–65), an annotated bibliography', *Bull. Hist. Med.*, 1946, **20**: 692–703. For a more recent treatment see M. J. Busby and A. W. Rodin, 'Relative contributions of Holmes and Semmelweis to the understanding of the etiology of puerperal fever', *Tex. Rep. Biol. Med.*, 1976, **34**: 221–237.

[4]A. Turunen, 'C. R. Ehrström, the Finnish predecessor of Ignaz Semmelweis, the defeator of puerperal fever', *Centaurus*, 1967, **12**: 197–201.

specific diseases and insuring that those causes do not obtain.[5] In order for such a procedure to be possible, diseases must be characterized so that they *have* necessary causes, and this is by no means a trivial requirement. The most obvious way to characterize a disease is by signs and symptoms or, given pathological anatomy, by morbid structural modifications; these were precisely the methods most commonly used in early nineteenth-century medicine. For example, one typical characterization of hydrophobia was a "complete horror of fluids, reaching to such a degree, that their deglutition become almost impossible".[6] But as physicians recognized, there were many ways in which a horror of fluids could come about – some physiological, some psychological. Given this characterization, there was no necessary cause of hydrophobia that could be isolated and controlled. Indeed, early nineteenth-century thought still allowed that some diseases, including hydrophobia, could occur spontaneously.[7] In general, there was little precision or method in discussion of causes; in textbooks the aetiology of diseases was frequently not discussed at all, and specific causes were identified in the most casual way.[8] Since every set of symptoms could be caused in radically different ways, or perhaps occur spontaneously, it was impossible *in principle* to identify consistently reliable therapeutic or prophylactic measures. Steps that corrected one case of a disease may have no positive effect in other cases of the same disease. This situation could only be corrected by conceptual innovations.

Typically the innovations were achieved as follows: one began with a specific disease characterized symptomatically or by pathological modifications. By looking carefully at controlled situations, a specific condition was identified that was necessary for many or most of the recognized cases of the disease. Next, the disease was given a new characterization in terms of that specific necessary condition. This, of course, entailed reclassifying many particular cases since the newly defined disease included some cases with different symptoms (but the same cause) and it excluded other cases with the same symptoms (but different causes). Finally, measures could be adopted to prevent the definitive causal condition. Of course, precisely the same cases of disease

[5]The distinction between necessary and sufficient causes, which is crucial to the argument in this essay, is treated in most elementary logic texts, e.g. Michael Scriven, *Reasoning*, New York, McGraw-Hill, 1976, pp. 61–65.

[6]Gabriel Andral, 'Perversions of sensibility: hydrophobia', *Lancet*, 1832, **1**: 805–809, p. 806.

[7]For example, in his *Lectures on the principles and practice of physic*, (Philadelphia, Lea & Blanchard, 1858; 1st ed., London, J. W. Parker, 1843, 2 vols.), Sir Thomas Watson says that both tetanus (p. 376) and hydrophobia (p. 396) can arise spontaneously. But most writers stop short of saying that for spontaneous cases there are no causes; "Diseases sometimes occur when no exciting cause, when no cause at all, has been apparent. All that we can say of such cases (which are not, however, very frequent) is, that the causes have not hitherto been discovered." (p. 77) By 1866 one reads that the term "spontaneous" is "to be taken in a conventional, not a literal sense. . . . Every disease must have its adequate determining cause. But many diseases are developed without our being able, in the existing state of knowledge, to refer them to their causes." Austin Flint, *A treatise on the principles and practice of medicine*, Philadelphia, Lea, 1866, p. 94.

[8]For example, in 1811 all the workmen in one gallery of a coal mine became ill while the workmen in surrounding galleries did not. In a discussion of the incident the sickness was identified as anaemia and the symptoms and attempted treatments were carefully described. The author then notes "the want of sun and the want of air seem to have been the causes of this singular malady." Marshall Hall, 'Anaemia', in Sir John Forbes, Alexander Tweedie, and John Conolly (editors), *The cyclopaedia of practical medicine*, (1st ed., London, Sherwood, Gilbert & Piper, 1833–35, 4 vols.), reprinted Philadelphia, 1849, vol. 1, p. 93. Semmelweis criticized a contemporary who proposed to investigate the aetiology of puerperal fever by requiring physicians to submit reports identifying the cause of each case they encountered. Semmelweis, op. cit., note 2 above, p. 350.

could have been controlled by exactly the same measures even if the disease were still defined symptomatically. But so 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 aetiological characterizations was absolutely necessary for systematic medical procedures. There were also theoretical advantages in the adoption of aetiological characterizations; given such characterizations it became possible to connect symptomatology and pathological anatomy and to explain many facets of diseases that had previously been observed and regarded as coincidental.

This new strategy first became prominent in work on the infectious diseases where its use is obvious. In earlier papers I have argued that the discovery of vitamins and Freud's early work in psychopathology are also applications of this basic method.[9] In this paper I shall discuss one of the earliest uses of this strategy – its use by Semmelweis in his work on puerperal fever. To emphasize the novelty of his approach, we will first review contemporary British and American medical literature on puerperal fever.

II

Here is a typical nineteenth century characterization of puerperal fever:

> 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 lochiae and milk, much accelerated pulse, furred tongue, great heat of skin, that peculiar pain in the sinciput . . . short breathing, the knees drawn up, and great anxiety of countenance.[10]

This characterization is entirely symptomatic. Attempts to derive a more precise characterization from pathological anatomy usually focused on structural modifications of the uterus, but because puerperal fever was associated with a variety of morbid alterations such attempts were not particularly successful. Through the middle decades of the nineteenth century, physicians frequently stressed the difficulty of obtaining an adequate characterization of the disease.[11]

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.[12] 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."[13] Holmes's conclusions and most of the specific case histories on which the conclusions are based are drawn from earlier British literature. But the

[9]"The germ theory, beriberi, and the deficiency theory of disease', *Med. Hist.*, 1977, **21**: 119–136, 'Germ theory, hysteria, and Freud's early work in psychopathology', ibid., **24**: 259–274.

[10]C. M. Miller, 'On the treatment of puerperal fever', *Lancet*, 1848, **ii**: 262.

[11]"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." James Young Simpson, 'Some notes on the analogy between puerperal fever and surgical fever', *Mthly J. med. Sci.*, 1850, **11**: 414–429, pp. 425f.

[12]The essay originally appeared in the *New Engl. Quart. J. Med. Surg.*, 1843; it was reprinted in Holmes's collected *Medical essays*, New York, 1883, pp. 103–172.

[13]Ibid., p. 131.

Holmes essay is a particularly clear and articulate statement of the British view, and it has been credited with anticipating Semmelweis, so the essay continues to have some historical interest. 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."[14] But Holmes felt that the existence of the minority justified the essay.

Holmes cites nearly twenty cases in which physicians examined or otherwise treated patients with puerperal fever or in which they performed autopsies on persons who died from puerperal fever and in which other patients subsequently contracted the disease. At the end of his essay he warns obstetricians against taking "any active part in the post-mortem examinations 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 labour ... as in danger of being infected by him".[15] Holmes considers cases suggesting that puerperal fever can be produced "by an infection originating in the matter or effluvia of erysipelas".[16] But he finds the relation of puerperal fever with 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," these cases are so relatively rare that they "hardly attract our notice in the midst of the gloomy facts by which they are surrounded".[17] Holmes never suggests that patients suffering from other diseases or corpses of persons who died from other diseases present any special danger to the delivering woman. 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.[18]

Each time Holmes states precisely his main point he says that the disease is *sometimes* (or *frequently*) carried from one patient to another.[19] "It is not pretended that the disease is always, or even, it may be, in the majority of cases, carried about by attendants; only that it is so carried in certain cases."[20] 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."[21] In the chronologically later introductory note, he writes that his theory "makes full allowance for other causes besides personal transmission, especially for

[14]A year before the first appearance of Holmes's essay, *Lancet*, (1842, i: 879) reported that in a discussion of puerperal fever in 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."

[15]Holmes, op. cit., note 12 above, p. 168.

[16]Ibid., p. 164.

[17]Ibid., p. 165.

[18]Ibid., p. 162.

[19]Ibid., pp. 112, 129, 131.

[20]Ibid., p. 123.

[21]Ibid., p. 133.

epidemic influences".[22] In the literature of the time, epidemic influences were generally identified with miasmatic or atmospheric factors that could not be specified more precisely; the criterion for deciding whether a given case of puerperal fever was epidemic was the frequency of related cases in the surrounding area.[23] Sporadic cases were neither infectious nor epidemic; one discussion ascribed sporadic cases

> to difficult labour; 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; diarrhoea; liability to putrid contagion from changes in the humours during pregnancy; hasty separation of the placenta; binding the abdomen too tight; sedentary employment; stimulating or spare diet; or to fashionable dissipation.[24]

Some writers assert that puerperal fever can arise spontaneously, others invoked providence to explain certain cases of the disease.[25] So far as I can determine, prior to 1848 – the year in which Semmelweis's theories were announced in England – no one in England or in the United States published anything identifying a necessary cause for all cases of puerperal fever. Oliver Wendell Holmes certainly did not do so and his writings suggest quite clearly that he did not believe in the existence of such a cause.

III

In the early decades of the nineteenth century, two gratis maternity clinics were operated in connexion with the Viennese general hospital. Obstetricians were trained in one clinic, midwives were trained in the other. In 1847 Semmelweis supervised the former. For several years the incidence of puerperal fever in that clinic was about three times as great as in the clinic for midwives. Various commissions investigated the 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, and epidemic diseases were generally beyond human control. "Against childbed fever that is due to atmospheric, cosmic, terrestrial influences there can be no defense. The advocates of an epidemic theory secure themselves behind this indefensibility; they thereby escape all responsibility for the devastations of the disease."[26] On the other hand, the two clinics were subject to the same atmospheric influences – if the disease were epidemic, it should have occurred with the same frequency in both clinics. Semmelweis was absolutely convinced by the difference in mortality that the disease was not epidemic.[27] Semmelweis considered numerous endemic factors that had been associated with the disease. He decided that overcrowding, rough handling, specific medical practices, inadequate ventilation, dietary mistakes, as well as particular physiological or psychological conditions of the patients could not explain the incidence of puerperal fever since all these factors were operative in both clinics.

[22]Ibid., p. 107.

[23]Ibid., p. 113; see also W. Tyler Smith, 'Puerperal fever', *Lancet*, 1856, **ii**: 503–505, p. 503.

[24]J. M. Waddy, 'On puerperal fever', ibid., 1845, **ii**: 671f, p. 671.

[25]Holmes, op. cit., note 12 above, cites a long passage in which a Dr. Blundell observed that "this fever may occur spontaneously," (p. 140). Holmes also criticizes Charles Delucena Meigs who, by appealing to providence to explain the disease, minimized human responsibility for mastering its aetiology.

[26]Semmelweis, op. cit., note 2 above, p. 117.

[27]Ibid., p. 6.

The crucial event in Semmelweis's quest for the endemic cause of puerperal fever came in March 1847. Professor Jakob Kolletschka, who had been Semmelweis's friend and teacher, died from a minor injury incurred while dissecting a corpse. When Kolletschka's body was dissected, Semmelweis recognized the results as similar to those obtained in dissections of women who died of puerperal fever.[28] 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.[29] He had also observed that when women died of puerperal fever their newborn infants, both male and female, sometimes died of a fever that left similar anatomical 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 case of the maternity patients, but to deny that the identical findings in the corpses of the newborn were the result of the same disease, is to reject pathological anatomy."[30] Similar reasoning forced Semmelweis to conclude that Kolletschka also died of the same disease. "Day and night I was possessed by the image of Kolletschka's disease, and was forced to recognize, ever more decisively, that the disease from which Kolletschka died was identical with that from which I had seen so many maternity patients die."[31] Because Semmelweis bases his argument on the similarity of pathological remains, it is easy to overlook the originality of his position. As he pointed out, he was not the first to observe that infants of either sex could contract puerperal fever.[32] But nearly all characterizations of the disease referred to the puerperal state or to morbid alterations of the uterus; such characterizations excluded infants and especially adult males *by definition*. Second, no clear patterns emerged in

[28]Ibid., 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, Md., and London, Johns Hopkins University Press, 1977, p. 185. But Hebra and Skoda did not pretend to give historical accounts, their object was only 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 (Semmelweis, op. cit., note 2 above, preface). Moreover, the account of Semmelweis's lecture before the k.k. Gesellschaft der Aerzte zu Wien on 15 May 1850 clearly reports Semmelweis as having said that he was led to his theory by the difference in mortality *together with* the pathological similarities between childbed fever and pyaemia among surgeons and anatomists. This was certainly an allusion to Kolletschka that no one in Vienna could have missed (Györy, op. cit., note 1 above, p. 49). Incidentally, while Semmelweis's first account of the discovery was published eleven years after the event – as Lesky notes – she neglects to point out that the published version was taken from a series of lectures that were delivered in 1850, two years after the discovery. I see no reason for thinking that the Kolletschka case was any less important to the development of Semmelweis's thought than he himself tells us it was.

[29]Semmelweis, op. cit., note 2 above, p. 106.

[30]Ibid., pp. 43, 51. In both passages Semmelweis notes that this conclusion forced him to recognize that the whole concept of childbed fever was wrong.

[31]Ibid., p. 53.

[32]The French called attention to the infant puerperal fever in 1855 and their discussions were reviewed in the *Mschr. Geburtsh.*, 1856, **7**: 152f, and in the *Wien med. Wschr.*, 1856, journal revue no. 3, pp. 22f. 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 erysipelas and typhus fever that could affect males. See, for example, Robert Storres, 'On the contagious effects of puerperal fever on the male subject', *Prov. med. surg. J.*, 1845, **19**: 289–294, p. 290. But, as far as I can determine, they never went so far as to identify diseases in the male as puerperal fever.

dissections of victims of puerperal fever; some corpses showed no morbid alterations whatsoever. Thus it was not clear how much weight to place on the similarities observed in dissections of surgeons who died of pyaemia (such as Kolletschka) and in the victims of puerperal fever. Finally, as Semmelweis admitted in other contexts, the pathological remains in Kolletschka and in the other surgeons were *not* identical to those found in the bodies of maternity patients.[33] In assimilating these cases, Semmelweis was as much transcending (repudiating) pathological anatomy as he was conforming to it. In this respect, Holmes provides an enlightening contrast. Holmes records that his interest in puerperal fever was occasioned by the case "of a physician who had made an examination of the body of a patient who had died of puerperal fever, and who himself died in less than a week, apparently in consequence of a wound received at the examination, having attended several women in confinement in the meantime, all of whom, as it was alleged, were attacked with puerperal fever."[34] But Holmes never suggests that the physician himself died of puerperal fever, and contemporary physicians typically concluded no more than that puerperal fever was *similar to* the diseases responsible for such deaths.[35] Semmelweis now took a remarkable and decisive step: "I was forced to admit that if we assume that Kolletschka's disease was identical with the disease which killed so many maternity patients, then this disease must originate from the same cause that brought it on in Kolletschka."[36] The cause of Kolletschka's disease was known; it was the introduction of decaying matter into his blood from the contaminated autopsy knife. Semmelweis quickly identified the contaminated hands of the examining physicians as the source of the decaying matter that induced the same disease in the maternity patients. Semmelweis recognized that decaying matter capable of causing puerperal fever could also be generated within the patients themselves. This could happen because of disease, because of retained lochia or placental remnants, or through the decay of body parts damaged during delivery. He estimated that in approximately one per cent of deliveries puerperal fever would arise from these factors and he regarded such cases as essentially beyond his control. As is well known, Semmelweis adopted chlorine washings to destroy the decaying matter and thereby reduced the mortality in his clinic almost to the one per cent that he calculated as optimal.

A decisive step in Semmelweis's reasoning was the assumption that various cases of a particular disease must share a common cause, in other words, that the disease has a necessary cause. Semmelweis gives no indication where this assumption came from, and it is not possible to be sure when he adopted it. In an essay published in 1858 he asserted that every case of childbed fever was due to the resorption of decaying organic matter.[37] By the time his main book was published, Semmelweis clearly

[33]Semmelweis, op. cit., note 2 above, pp. 40, 107.

[34]Holmes, op. cit., note 12 above, p. 104, cf. pp. 147, 161.

[35]See for example, Storres, op. cit., note 32 above, p. 290.

[36]Semmelweis, op. cit., note 2 above, p. 54, cf. pp. 40, 274, 338.

[37]György, op. cit., note 1 above, p. 78. The first four persons who announced Semmelweis's discovery, Ferdinand Hebra, C. H. F. Routh, Carl Haller, and Joseph Skoda, did not assert that *every* case of puerperal fever was due to the resorption of decaying matter, and the claim does not appear unambiguously in the report of Semmelweis's lectures before the k. k. Gesellschaft der Aerzte zu Wien in the spring of 1850.

believed that resorption was a necessary cause. He said so quite explicity: "In order that childbed fever should occur, it is a *conditio sine qua non* that decaying matter is introduced into the genitals."[38] He also frequently asserted that every case of childbed fever, without a single exception, came about through resorption of decaying matter.[39] Semmelweis also distinguished between the necessary cause, which he sought, and the purportedly sufficient causes generally identified by the physicians of his day. Semmelweis cites a passage in which Josef Hamernik specified three criteria that must be satisfied in identifying the cause of a disease: "Has this cause always the same effect? As an experiment can one always occasion the disease in this way? In those cases in which the cause does not occasion the specified disease, can the same cause of failure always be identified?"[40] 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: "We have injected rabbits with decaying matter and some consquently died from pyaemia and others did not. Could we deny that the decaying matter was the cause of pyaemia in the rabbits that died simply because the matter did not occasion pyaemia in all the rabbits?"[41] In this passage he is using "cause" in the necessary sense. Semmelweis claims to have fulfilled the second condition by his experiments with rabbits – he ignores Hamernik's stipulation that the disease *always* be produced in this way. Finally, while admitting that he has not satisfied the third condition, Semmelweis notes that "instead, we have satisfied another condition that Hamernick has not posed, but that constitutes a condition for a true aetiology and that my aetiology of childbed fever satisfies; namely, we have reduced the disease by making harmless that which we have identified as its cause".[42] 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 aetiology one must identify that necessary condition whose prevention will eliminate the disease, just as by preventing the resorption of decaying matter Semmelweis eliminated childbed fever in all those cases where the resorption could be prevented (i.e. in all cases except those in which the matter is generated internally). Having thus identified a necessary cause for childbed fever, Semmelweis gives a new aetiological characterization of the disease as "a resorption fever determined by the resorption of decaying animal-organic matter".[43] He characterizes pyaemia as "decomposition of the blood through decaying animal-organic matter".[44] From these definitions, it follows that every case of childbed fever is a case of pyaemia; in other words, puerperal fever is not a species of disease but only a variety of pyaemia.[45]

> In expounding the concept of puerperal disease as epidemic or endemic, one must disregard entirely the number of those patients who become ill or die. The cause, in consequence of which the illness or death

[38]Semmelweis, op. cit., note 2 above, p. 196.
[39]For example, ibid., pp. 87, 102, 114, 115, 179.
[40]Ibid., p. 418.
[41]Ibid.
[42]Ibid.
[43]Ibid., p. 102; cf. Györy, op. cit., note 1 above, p. 70.
[44]Semmelweis, op. cit., note 2 above, p. 107.
[45]Ibid., p. 106.

> follows, determines whether the disease is to be conceived as epidemic or endemic. Epidemic puerperal fever is induced by atmospheric, cosmic, or terrestrial influences; the concept of an epidemic does not stipulate whether one or one hundred persons become ill.[46]

Given the above characterization of puerperal fever and this account of epidemics, it follows that there never had been a single case of epidemic childbed fever.[47] Of course it also follows that there are no spontaneous cases of the disease.[48]

As Semmelweis himself certainly recognized, the strength of his characterization of the disease lay in its explanatory power. In this respect, the difference between Holmes and Semmelweis is the difference between night and day. In his essay, Holmes uses his account of the disease to explain nothing more than the very facts that suggested that account. Thus, while the account led to useful practical procedures, it had no real scientific or theoretical interest. By contrast, 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 explains why children never died from puerperal fever while the mother remained healthy, the changing mortality rates of infants, the lower mortality rate of women who delivered on the way to the hospital and of premature deliveries, the particular pattern in which the disease appeared among patients, the changing mortality rates in the two clinics, the rarity of infections during pregnancy or after delivery, the apparently contagious nature of the disease, its seasonal pattern, the concentration of cases in teaching hospitals, the variation of incidence of the disease in certain non-teaching hospitals, and its relative frequency in different countries and in different historical periods.[49] Of course, each of these explanations deals with circumstances other than those which suggested the theory in the first place. Semmelweis has not given us merely practical advice for avoiding some cases of puerperal fever, but a complete explanatory scientific theory.

IV

Semmelweis's conclusions were announced as follows: first, editorials appeared in December 1847 and in April 1848 in the *Zeitschrift der k. k. Gesellschaft der Aerzte zu Wien*; these were written by Ferdinand Hebra and were clearly supportive of Semmelweis. Second, at about the same time Semmelweis and his friends wrote a series of letters to directors of various European obstetrical clinics; the letters announced the discovery and invited responses. Third, C. H. F. Routh (November 1848 in London) and Joseph Skoda (October 1849 in Vienna) delivered lectures articulating and endorsing Semmelweis's views; Carl Haller also mentioned Semmelweis enthusiastically and released supporting statistical data in a report published in 1849. Fourth, in May 1850 Semmelweis delivered a lecture at a general meeting of the *Gesellschaft*; the discussion continued in general meetings held in June and July. Reports of the meetings were published in the *Zeitschrift*, as was a critical response by Eduard Lumpe, but

[46] Ibid., p. 51, cf. p. 118.

[47] Ibid., p. 116.

[48] Semmelweis ridicules his contemporaries for saying that puerperal fever can arise by chance or without demonstrable cause. Ibid., pp. 401 (cf. p. 328), 495 (cf. p. 489).

[49] Ibid., pp. 67, 69, 70, 104, 108f, 121f, 124, 125, 145f, respectively.

Semmelweis's lecture was not published and we know of it only from the secretary's report. Fifth, in the spring of 1850 Semmelweis also delivered a series of lectures before the *Budapester königl. Aerzteverein*; these were published in 1858. In 1860 an essay was published in which Semmelweis explained the differences between his views and those of the British.[50] Finally, Semmelweis's major work, *Die Aetiologie, der Begriff und die Prophylaxis des Kindbettfiebers*, was published in November 1861. By the time Semmelweis wrote the book, his ideas had been widely disseminated and discussed. One disadvantage in the late appearance of the 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 the theory. On the other hand, Semmelweis was able to devote nearly half of the book to discussion of long passages extracted from critics and supporters. To the few who have ever read this discussion, the novelty of Semmelweis's approach should be totally clear. One striking fact is that almost unanimously *both his critics and his supporters end up saying the same thing*: puerperal fever can be caused by decaying organic matter exactly as Semmelweis claimed, but it can be caused in other ways as well.

We have already noted that in his initial response to Semmelweis, Simpson failed to see any difference between Semmelweis's views and those of the British. As Semmelweis noted, Simpson gradually understood more perfectly what was at issue;[51] in an essay published in 1850 Simpson refers to Semmelweis positively, and it is possible that the essay reflects a greater debt to Semmelweis than Simpson acknowledged.[52] However, Simpson continued to believe that some cases of puerperal fever were epidemic in origin and were not due to the resorption of decaying matter.[53] By 1855 Semmelweis was frequently mentioned in British medical literature, but typically they took him to be in agreement with their general position and they overlooked his claim to have discovered the one decisive cause of all cases of puerperal fever.[54] Semmelweis mentions specifically Edward William Murphy as sympathetic to his view; Murphy does mention Semmelweis favourably, but he explicitly acknowledges a whole range of causal factors.[55] The second person to respond to Semmelweis's letters was Christian Bernard Tilanus of Amsterdam. Tilanus claimed to be in agreement with Semmelweis and was cited by Ferdinand Hebra in the April 1848 editorial as having written in Semmelweis's support. But while Tilanus acknowledged that he had long believed that puerperal fever could be spread in the way that Semmelweis identified, he, like Simpson, continued to believe that many cases of puerperal fever were due to epidemic factors.[56] Frederich W. Scanzoni, whom Semmelweis regarded as a principal opponent and to whom he devoted eighty-five pages in his book, clearly admitted the possibility that puerperal fever could sometimes come about in the way that

[50]All of the preceding are reprinted in Györy, op. cit., note 1 above, pp. 23–58.

[51]Semmelweis, op. cit., note 2 above, p. 285.

[52]Simpson, op. cit., note 11 above, p. 429.

[53]Ibid., p. 427; and 'Medical news', *Mthly J. med. Sci.*, 1851, **13**, 72–80.

[54]E.g., Smith, op. cit., note 23 above, p. 504. The reprinted version of Holmes's essay refers to Semmelweis as well as to Routh and Skoda, Holmes, op. cit., note 12 above, p. 170.

[55]Edward William Murphy, 'Puerperal fever', *Dublin Quart. J. med. Sci.*, 1857, **24**: 4, 19.

[56]Semmelweis, op. cit., note 2 above, pp. 310–313.

Semmelweis discussed,[57] yet he insisted 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.[58]

Gustav Liebig included a positive reference to Semmelweis in his *Chemical letters* and Semmelweis treated him as an ally, but Liebig's passage concludes "certainly other causes of childbed fever will be identified, however, no unprejudiced person can doubt that the cause identified so sagaciously by Dr. Semmelweis, at the maternity clinic in Vienna, is among the causes".[59] Hermann Lebert, Professor at Breslau, wrote "Semmelweis has made into a system the direct inoculation of poison taken from the corpses of those who have died from this disease. Whether such inoculation is possible is questionable. In any case this would be at most one of many possible ways in which the disease can be conveyed".[60] Anton Hayne, the veterinarian who claimed priority for Semmelweis's discovery, noted that animals frequently contract a disease corresponding to childbed fever and that this is a consequence of dietary errors, injuries, exposure to cold, etc. 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".[61] 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 claimed, and that even before delivery other factors predispose women to the disease.[62] Joseph Herman 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 labour process. He asked how Semmelweis's hypothesis could be reconciled with the observation that the disease occurs in relatively few normal deliveries. He than admitted that the resorption of decaying matter "may be one path that leads to puerperal fever, but it is certainly not the only one".[63] D. Everken wrote "it would never occur to me to identify this circumstance as the only cause, but is has certainly caused me no longer to undertake examinations of patients after investigating corpses". He then warned Semmelweis that nowhere is one more frequently tempted with the *post hoc, ergo propter hoc* fallacy than in medicine.[64] Carl Braun, Semmelweis's successor as assistant in the first clinic, identified thirty causes of childbed fever of which the twenty-eighth was cadaverous infection. Other causes included conception and pregnancy, uraemia, pressure exerted on adjacent organs by the shrinking uterus, emotional traumata, mistakes in diet, chilling, and epidemic influences.[65]

In 1862 an unsigned review of Semmelweis's book appeared in the *Monatsschrift für Geburtshilfe*. According to the reviewer, Semmelweis calls everyone who disagrees

[57]Ibid., p. 396.
[58]Ibid., pp. 348, 356, 365.
[59]Ibid., p. 422.
[60]Ibid., p. 436.
[61]Ibid., p. 442.
[62]Ibid., p. 458.
[63]Ibid., p. 463.
[64]Ibid., p. 467.
[65]Ibid., pp. 530f.

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 aetiological 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".[66] But Semmelweis owed no proof to anyone. That every case of puerperal fever was due to the resorption of decaying matter followed from Semmelweis's aetiological definition of "puerperal fever". Moreover, that particular characterization of the disease was justified not only by the systematic treatment and prophylaxis that it made possible, but also because of its great explanatory power.

In 1845 Lumpe published an informed survey of existing literature on puerperal fever. He called attention to similarities between puerperal fever and pathological states induced by the introduction of decaying matter into the blood.[67] He noted that puerperal fever could be caused by the retention of lochial discharge or of decaying placental remains.[68] He was aware of British literature in which the contagiousness of puerperal fever was stressed.[69] He was familiar with the British view that women who were not in the puerperal state, and even men, could contract a similar disease.[70] Nevertheless, Lumpe believed that the epidemic origin of puerperal fever was established beyond doubt by the frequent occurrence of simultaneous and similar cases of the disease in which no detectable change occurred in other possible causal factors.[71] He even cited the difference in mortality between the two Viennese clinics as evidence for the operation of local miasmatic influences.[72] He identified the usual causal conditions: emotional disturbances, mistakes in diet, chilling. Lumpe had all the facts before him, he knew everything that Semmelweis later knew, he had been trained by the same medical faculty as Semmelweis, but he could not break free from the accepted way of looking at things. His response to Semmelweis's theory is particularly interesting.

> When one thinks how, since the first occurrence of puerperal fever epidemics, observers of all times have sought in vain for its cause and the means of preventing its origin, Semmelweis's theory takes on the appearance of the egg of Columbus. I admit that I myself was originally overjoyed as I heard of the fortunate results of the chlorine washings; as everyone else, I too have had the misfortune of witnessing many blossoming young individuals fall before this devastating plague. However, during my two years as assistant at the first clinic, I observed incredible variations in the incidence of sickness and death. Because of this . . . any other possibility is more plausible than one common and constant cause.[73]

After numerous criticisms of Semmelweis, Lumpe concludes, "If adoption of the washings makes it possible to avoid even the least significant of the many concurring factors that cause puerperal fever, then [Semmelweis's] was a sufficient large service.

[66] *Mschr. Geburtsh.*, 1862, **18**: 406f.
[67] Eduard Lumpe, 'Die Leistungen der neuesten Zeit in der Gynaekologie', *Zt. k. k. Ges. Aerzte zu Wien*, 1845, **1**: 341–371, pp. 350f.
[68] Ibid., pp. 345, 348.
[69] Ibid., pp. 345f.
[70] Ibid., pp. 343, 348.
[71] Ibid., pp. 342f.
[72] Ibid., p. 347.
[73] Semmelweis, op. cit., note 2 above, pp. 443f.

However, whether this is in fact the case, only a later time will be able to decide. In the meantime, I believe we should wait and wash."[74] Lumpe's choice of metaphor could not have been more appropriate: Semmelweis himself noted "I am entirely of Dr. Lumpe's opinion when he says that my theory is like the egg of Columbus. I myself have often expressed amazement, not at my having become aware of the strident contradiction between theory and daily observation, but rather that it was not recognized long before me."[75]

One cannot avoid the impression that there was much more agreement between Semmelweis's critics and his supporters than there was between Semmelweis and his supporters. The key 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. With a few possible exceptions, no one believed that he had identified a necessary cause for the disease. 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 Semmelweis's critics noted that in all the literature on puerperal fever there was nothing that supported the view Semmelweis was advancing.[76] Semmelweis emphatically denied this, but it was true.[77] Indeed, given the symptomatic characterizations of the disease that were standard among his contemporaries, puerperal fever probably included various kinds of inflammations and very likely did not have a necessary cause.[78] From their point of view, Lebert, Lumpe, and Karl Levy were exactly right in objecting (respectively) that Semmelweis had created a system and an egg of Columbus, and that his argument rested as much on unstated *a priori* assumption as on facts.[79] But while his claims were partly analytic, they were certainly not trivial. Semmelweis's conceptual realignment was precisely what was needed if diseases were to be approached systematically and if medicine was to become an explanatory science.

V

Many of Semmelweis's specific ideas were known before he started his work, this is why the priority for his discoveries has been subject to almost constant dispute. As I have tried to show, it is Semmelweis's method that makes his work a genuine part of what we know as medical science and that differentiates him from his contemporaries. It would be interesting to know (a) to what extent Semmelweis himself initiated the method that he uses, and (b) to what extent Semmelweis directly influenced those who

[74] Ibid., p. 454.

[75] Ibid., p. 443.

[76] Carl Braun and H. Silberschmidt both said this. Ibid., pp. 275, 407.

[77] In the Budapest lectures (1850) Semmelweis seemed much less concerned to establish the existence of supporting literature than he was in his later book (1861). Ibid., p. 276. In the earlier lecture 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 nevertheless acknowledged the operation of atmospheric influences as the primary factor in the onset of puerperal epidemics. In the earlier lectures Semmelweis pointed out the difference in their opinion (Győry, op. cit., note 1 above, p. 68); in the book he quotes Tilanus's letter without comment.

[78] Smith, op. cit., note 23 above, p. 503.

[79] Semmelweis, op. cit., note 2 above, pp. 436, 443f, 294.

subsequently adopted that method. Very likely neither question can be answered conclusively. The second question is outside the scope of this essay; however, we must consider some evidence related to the first question.

In her excellent monograph, *The Vienna Medical School of the nineteenth century* and in an independent article, 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."[80] Thus Lesky sees Semmelweis as obtaining new results from methods he learned from Rokitansky and Skoda. 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. However, for other crucial and decisive aspects of his method the case is less clear. Lesky claims that the training he received in pathological anatomy while working with Rokitansky enabled Semmelweis to 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 pyaemia.[81] But neither Rokitansky himself nor any of his other students or associates – including Lumpe, Johann Baptist Chiari, and Eduard Mikschik – recognized the findings as identical.[82] In fact, as Semmelweis himself noted, the findings were *not* identical in the genital area – the area on which all attempts to secure an anatomical characterization necessarily focused.[83] The British, not the Viennese, stressed the similarity between the pathological remains in maternity patients and in victims of pyaemia. Lumpe, who was also an assistant in the first clinic, expressed astonishment at the British view that men could contract a disease similar to puerperal fever.[84] To what extent then can this aspect of Semmelweis's thought be ascribed to his training in the Viennese school?

Lesky also claims that the Vienna school "provided Semmelweis with the intellectual tools to infer a single unified cause from identical anatomical remains."[85] 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.[86] 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. It would be useful to know

[80] Lesky, op. cit., note 28 above, p. 183, and 'Semmelweis: Legende und Historie', *Dt. med. Wschr.*, 1972, 97: 627–632, p. 629.
[81] Ibid., p. 629.
[82] Lesky, op. cit., note 28 above, p. 185.
[83] Semmelweis, op. cit., note 2 above, p. 40, 107.
[84] Lumpe, op. cit., note 67 above, p. 348.
[85] Lesky, op. cit., note 80 above, p. 629.
[86] Ibid.

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 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 precludes any thought that the generation of the sickness is 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.[87] These assertions do not preclude the possibility that various causal factors were reponsible for the *usual* incidence of puerperal fever or that epidemic puerperal fever sometimes occurs. 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 deliveries or emotional traumata could also cause puerperal fever.[88] 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 puerperal fever, and he never describes Semmelweis's work as a quest for one necessary cause.[89] By contrast, during the same period, Johann Klein, who was Semmelweis's supervisor and who rejected Semmelweis's opinions, consistently described Semmelweis's work as a quest for *the cause* (singular) of childbed fever.[90] Even in lectures on puerperal diseases given much later Skoda never unambiguously asserted that all cases of puerperal fever could be ascribed to a single necessary cause.[91] 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 it could be prevented by chlorine washings. My only point here is that 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 redefining the disease in terms of such a cause. Since this particular strategy was a crucial step in Semmelweis's method, and since he seems to have been the first to use this strategy clearly and unambiguously, his achievement cannot be thought of simply as a new application of Rokitansky's and Skoda's methods of reasoning.[92] Since this particular strategy is an important aspect

[87] Győry, op. cit., note 1 above, p. 36.

[88] Helm says, in fact, that neither Skoda nor Semmelweis had said anything to preclude this possibility and he said this in the discussion of Semmelweis's lecture before the k. k. Gesellschaft der Aerzte zu Wien. For Semmelweis this was contradicted by Heinrich Herzfelder's report of the meeting (*Zt. k. k. Ges. Aezte zu Wien*, 1851, **8**: vii). But even if it were true it would only indicate that Semmelweis did not decide until later that decaying organic matter was a necessary cause.

[89] See, for example, Erna Lesky, *Ignaz Philipp Semmelweis und die Wiener medizinische Schule*, Vienna, Böhlau, 1964, pp. 21, 26f.

[90] Ibid., pp. 29, 43, 46. The contrast between Skoda and Klein may be 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 his claim and rejected it.

[91] 'Ueber Krankheiten bei Puerpern', *Allg. Wien. med. Ztg.*, 1858, **3**: 20, 1; and **3**: 21, 1. Here Skoda does assert that puerperal fever is a kind of pyaemia, but this is not conclusive unless one can show that he ascribed pyaemia to a necessary cause.

[92] In a recent paper, 'The enigma of Semmelweis – an interpretation', *J. Hist. Med.*, 1979, **20**: 255–272, Sherwin B. Nuland follows Lesky's general interpretation of the relation between Semmelweis and the

of our contemporary medical research tradition, his achievement is connected with modern medicine in a way that the work of many of his teachers and so-called predecessors simply is not.

VI

It is generally acknowledged that the move toward aetiological characterizations of disease was one facet of a nineteenth-century revolution in medical thought and that this revolution was intimately connected with the rise of bacteriology. After noting that the work of Koch and Pasteur enabled physicians to adopt new characterizations of diseases in terms of specific micro-organisms rather than in terms of morbid anatomical modifications, 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."[93] But this approach, which ultimately spread to virtually every area of medicine, preceded Koch and Pasteur, and it existed in medicine quite apart from any serious interest in micro-organisms. It remains to be seen whether Semmelweis initiated this method and whether its use in his writings directly influenced those who so brilliantly explained the infectious diseases. Nevertheless, Semmelweis's place in the history of modern medicine is at least as dependent on his particular method as on any specific discoveries he made, and his use of that method provides a justification for his claim to priority that is simply not available to any of his so-called predecessors.

SUMMARY

Ignaz Semmelweis is generally known for identifying puerperal fever as an infectious disease preventable by aseptic methods. While no one today would contest the importance, the practicality, or the independence of his contribution, its originality has been disputed from the beginning. However, there is a basic methodological difference between Semmelweis' work and the work of all those who are sometimes said to have preceded him in his discovery. Semmelweis was very likely the first to identify a single necessary cause for all cases of a particular disease and to recharacterize the disease in terms of that necessary cause. This strategy, which until now has generally been regarded as a contribution of those who worked on the infectious diseases, was necessary if medicine was to become an explanatory science and it has since become pervasive in most areas of medicine. Yet it was precisely this aspect of Semmelweis's work that his contemporaries were unable to accept. Moreover, there is no reason to think that Rokitansky and Skoda, who have been identified as the intellectual fathers of Semmelweis's work, agreed with, adopted, or even understood this crucial strategy. Thus Semmelweis was associated with and contributed to our modern medical research tradition in a more fundamental way than any of his so-called predecessors.

Second Vienna Medical School. He notes that "Skoda and Rokitansky both recognized that the 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.'" (p. 257) Skoda and 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.

[93] Owsei Temkin, 'Health and disease', reprinted in *The double face of Janus*, Baltimore, Md., and London, Johns Hopkins University Press, 1977, p. 436.