PUERPERAL FEVER IN EIGHTEENTH-CENTURY BRITAIN*

Margaret DeLacy

Even to physicians accustomed to witnessing great suffering and frequent death, puerperal fever was a particularly horrible disease. "I almost shudder with horror when I consider the excruciating torments that must rack the distressed patient under these dreadful circumstances," wrote Nathaniel Hulme, physician to the City of London Lying-in Hospital, in 1772.[1] In the same year, John Leake of the New Westminster Lying-in Hospital commented:

> If those diseases which have been found most dangerous and mortal in their effects ought principally to be considered by physicians, none will more deservedly engage their attention than the *Childbed Fever,* as there is not, perhaps, any malady ... where powerful remedies of every kind have been tried with more diligence and less success.[2]

The eighteenth century marked a new epoch in the history of this affliction. Although we have reports of four possible epidemics from the seventeenth century, the first well-documented epidemic of what was probably streptococcal puerperal fever occurred in the Hôtel-Dieu in Paris in 1745, and the first known British epidemic took place at the British Lying-in Hospital in Brownlow Street in 1760.[3] In the eighteenth century, epidemic puerperal fever was for the first time recognized as a distinct disease, and for the first time treatises were devoted to the subject. At the end of the

*This paper was first read at the sixty-first annual meeting of the American Association for the History of Medicine, New Orleans, Louisiana, 6 May 1988. Research for this paper was conducted with the aid of a grant from the American Council of Learned Societies for 1986–87. I would like to thank Robert Kimbrough, Chief Professor of Infectious Diseases and Assistant Chief of Medicine of Good Samaritan Hospital, Portland, for advising me on current medical views about the transmission of streptococcal infection; Judith Schneid Lewis and Irvine Loudon for advice on the argument; Mary Pauli of the Clackamas County Library, Douglas Tallman of the Multnomah County Library, Jerome DeGraff of Portland State University Library, and the staff of the National Library of Medicine for providing material; and my husband, John DeLacy, for technical assistance.

[1] Nathaniel Hulme, *A Treatise on the Puerperal Fever* ... (1772), reprinted in *Essays on the Puerperal Fever and Other Diseases Peculiar to Women Selected from the Writings of British Authors Previous to the Close of the Eighteenth Century,* ed. Fleetwood Churchill (London: Sydenham Society, 1849), p. 66. (Essays found in Churchill's collection will hereafter be noted as FC.)

[2] John Leake, *Practical Observations on the Childbed Fever* (1772), FC, p. 117.

[3] For three seventeenth-century epidemics, see James Hawley Burtenshaw, "The fever of the puerperium (puerperal infection)," *New York Med. J.,* 1904, *79:* 1073–78, 1134–38, 1189–94, 1234–38, and *80:* 20–25 (pp. 1137–38). August Hirsch, in his *Handbook of Geographical and Historical Pathology,* trans. Charles Creighton, vol. 2 of 3 (London: New Sydenham Society, 1885), gives a table of epidemics (pp. 422–24), which lists a fourth. Both of them note the epidemic in Paris (1664).

century the first step was taken toward understanding its etiology when Alexander Gordon demonstrated that birth attendants could be carriers.[4]

Many historians have associated the rise of puerperal fever with the rise of the male obstetrician in general and the maternity hospital in particular. In a seminal article of 1965, Thomas McKeown and R. G. Brown wrote:

> When first introduced, and for many years after ... institutional confinement had an adverse effect on mortality.... with few exceptions hospital death rates were many times greater than those for related home deliveries.... it may be asked whether results of institutional delivery at the earlier period were equally bad. There is reason to believe that they were worse.[5]

Feminists soon adopted this argument. For example, Ann Oakley commented in 1980:

> The achievements of male obstetrics over those of female midwifery are ... argued ... from the double premise of male and medical superiority. More recent investigations ... are now revealing a different picture, in which the introduction of men ... brought special dangers to mothers and babies. The easier transmission of puerperal fever in male-run lying-in hospitals is one example; the generally careless and ignorant use of technology another. In Britain in the eighteenth and early nineteenth centuries many of the male midwives' innovations were often fatal.[6]

These contentions have been accepted by many historians,[7] but the available evidence has not been fully brought to bear on this issue.

[4] Alexander Gordon, *A Treatise on the Epidemic Puerperal Fever of Aberdeen* (1795), FC, pp. 445–500.

[5] Thomas McKeown and R. G. Brown, "Medical Evidence Related to English Population Changes in the Eighteenth Century," in *Population in History: Essays in Historical Demography,* ed. David V. Glass and D. E. C. Eversley (1965; reprint, London: Edward Arnold, 1969), pp. 287–88.

[6] Ann Oakley, *Women Confined* (Oxford: Martin Robertson, 1980), pp. 10–12. See also Barbara Katz Rothman, *In Labour: Women and Power in the Birthplace* (London: Junction Books; New York: W. W. Norton, 1982), pp. 52–55.

[7] See Gail Pat Parsons, "The British medical profession and contagion theory: puerperal fever as a case study, 1830–1860," *Med. Hist.,* 1978, *22:* 140–41; A. Clair Siddall, "Bloodletting in American obstetric practice, 1800–1945," *Bull. Hist. Med.,* 1980, *54:* 107; Cecilia C. Mettler, *History of Medicine* (Philadelphia, Pennsylvania: Blackiston, 1947), p. 907; David Charles and Bryan Larsen, "Streptococcal puerperal sepsis and obstetric infections: a historical perspective," *Rev. Infect. Diseases,* 1986, *8:* 411; Harold Speert, *Iconographia Gyniatrica: A Pictorial History of Gynecology and Obstetrics* (Philadelphia, Pennsylvania: F. A. Davis, 1973); Jean Donnison, *Midwives and Medical Men: A History of Inter-Professional Rivalries and Women's Rights* (New York: Schocken, 1977); Lawrence D. Longo, introduction to his recent edition of Charles White's *A Treatise on the Management of Pregnant and Lying-in Women* (London: Edward and Charles Dilly, 1773; reprint, Canton, Massachusetts: Science History Publications, 1987), p. ix; and Roderick E. McGrew and Margaret P. McGrew, *Encyclopedia of Medical History* (New York: McGraw-Hill, 1985), s.v. "Puerperal Fever" (pp. 291–92).

For the opposing view, see Edward Shorter, *A History of Women's Bodies* (New York: Basic Books, 1982), and *idem,* "The Management of Normal Deliveries and the Generation of William Hunter," in *William Hunter and the Eighteenth-Century Medical World,* ed. William F. Bynum and Roy Porter (Cambridge: Cambridge University Press, 1985), pp. 371–84. Shorter's article offers compelling evidence that midwives intervened routinely in normal labor in early modern Europe, to the extent of regularly removing the placenta manually immediately following birth. It also documents the increasingly conservative management of eighteenth-century obstetricians. My own research confirms Shorter's conclusions. Shorter anticipated the present paper by suggesting that the incidence of epidemic puerperal fever should be compared with the incidence of epidemics of streptococcal pharyngitis. See also Mabel C. Buer, *Health, Wealth, and Population in the Early Days of the Industrial Revolution* (1926; reprint, New York: Howard Fertig, 1968), pp. 139–47, and Irvine Loudon, "Deaths in childbed from the eighteenth century to 1935," *Med. Hist.,* 1986, *30:* 18.

Historians have cited the appalling rates of postpartum sepsis found in nineteenth-century institutions and have claimed that eighteenth-century rates could hardly have been any better. Even when eighteenth-century rates are cited, they are often averaged over periods of many years, thus obscuring changes that took place during the course of the century. Finally, some historians have accepted McKeown and Brown's dismissal of reported rates as "frankly incredible."[8]

This paper will reassess the incidence of puerperal fever in the eighteenth century to determine the relationship between the rise of the maternity hospital and maternal mortality. Part 1 discusses problems in defining and understanding the disease. Part 2 analyzes the timing of epidemics in maternity hospitals and concludes that the connection between the rise of the maternity hospital and the appearance of epidemic puerperal fever may have been indirect: other factors may have been involved, including a change in the incidence or virulence of the pathogens that caused puerperal fever. Part 3 examines maternal mortality figures and finds that there was a general improvement in mortality rates during the course of the century—an improvement that was more marked in hospital figures than in figures for home births. Part 4 argues that the development of contagionist disease theory may have helped fend off some epidemics in maternity hospitals toward the end of the century.

WHAT IS PUERPERAL FEVER?

It is easier to understand the difficulties and confusion that confronted the eighteenth-century physician if we realize that puerperal fever is still a disease in search of a satisfactory definition. Among historians there is a working understanding that puerperal fever is similar to, if not synonymous with, postpartum infection and involves some bacterial invasion of the birth canal following childbirth. On closer examination, however, a precise delineation of the disease becomes entangled in ambiguities and complexities. Following childbirth, women may coincidentally contract fevers from diseases such as typhoid or from bladder infections which are largely unrelated to any damage to their reproductive systems. (These fevers may still partly be a result of pregnancy and birth insofar as these events may depress immunity, expose patients to cross-infection, or disturb other parts of the body.) Furthermore, postpartum infection can result from a range of infective organisms, many of which may also cause other diseases. Some of these pathogens may cause illnesses such as trembling, heart disease, and arthritis: since these ailments are usually the result of other factors, the link between them and postpartum infection was not appreciated until very recently. In some cases the initial infection is silent and the damage appears

[8] McKeown and Brown, "Medical Evidence," p. 289.

later, or affects the child more than the mother. Some of these organisms usually come from outside the body, but some are endogenous: they are normal inhabitants of the female genital tract or of other parts of the body. Finally, women may experience ephemeral fevers that are unrelated to any identified infection and are possibly due to hormonal changes associated with parturition and lactation.[9]

Since postpartum infection in hospitals is notoriously a nosocomial (hospital-generated) disease, researchers face a further problem in the form of the reluctance of medical personnel to admit that it exists and thus accept responsibility for causing illness. The "fudge factor" caused by the inclination to assign cases of postpartum infection to some unrelated condition finally compelled British researchers to adopt a strict definition of puerperal fever based only on symptoms: "A fever of 100.4°F [or more] over a period of 24 or more hours during the three weeks after childbirth."[10] (Americans adopted a slightly different definition.) However, what is considered a relevant period after childbirth is still a problem, as it was in the eighteenth century, and this renders different series of postpartum morbidity and mortality rates incompatible. Modern hospitals frequently send patients home within one to four days after delivery and do not keep track of them thereafter. Moreover, this definition is not based on etiology and thus includes a large number of unrelated fevers. For example, an investigation at Queen Charlotte's Hospital in 1951 found 1,423 cases of fever associated with 2,701 deliveries, but only 141 of those cases were defined as "true genital tract infections."[11] Other researchers, attempting a more precise measurement, have relied on bacterial cultures, only to experience difficulties because of the large number of positive cultures in asymptomatic patients.

Postpartum infection, in the broad sense of any genital tract infection following childbirth, was a significant cause of maternal mortality in the eighteenth century. Estimates vary, but it may well have accounted for half of all maternal deaths. Because many of the causative organisms were endogenous or very common in the environment, women were often resistant to them and only became ill if they sustained extensive damage to the birth canal or excessive trauma. Although they might be fatal, these postpartum infections were also often mild. Because their incidence depended to a large degree on what happened in the course of a particular

[9] For general works on postpartum infection, see Sebastian Faro, ed., *Diagnosis and Management of Female Pelvic Infections in Primary Care Medicine* (Baltimore, Maryland: Williams and Wilkins, 1985), and William J. Ledger, *Infection in the Female,* 2d ed. (Philadelphia, Pennsylvania: Lea and Febiger, 1986). I would like to thank Michael Collins for supplying me with copies of these works. David Charles and Thomas A. Klein, "Postpartum Infection," in *Obstetric and Prenatal Infections,* ed. David Charles and Maxwell Finland (Philadelphia, Pennsylvania: Lea and Febiger, 1973), contains useful history but is now outdated.

[10] As cited in Loudon, "Deaths in childbed," p. 25.

[11] Leonard Colebrook, "Puerperal Infection," in J. M. Munro Kerr, R. W. Johnstone, and Miles H. Phillips, eds., *Historical Review of British Obstetrics and Gynecology, 1800–1950* (London: Livingstone, 1954), p. 224. See also Joan Taylor and H. D. Wright, "The nature and sources of infection in puerperal sepsis," *J. Obstet. Gyn. Brit. Empire,* 1930, n.s. *37:* 213–32.

labor, they tended to be incidental or endemic rather than epidemic.[12] Every eighteenth-century obstetrician saw many women with fevers resulting from traumatic labors or from such problems as a retained placenta. In some cases, the midwife or physician introduced the infection, in other cases it came from the woman's own body or from her home, but these infections were a part of every practice. It was the frequency of such fevers which hindered the acceptance of the theories of Oliver Wendell Holmes and Ignaz Semmelweis in the next century, since many cases of these fevers could not be traced to carriers; nor could any common precautions eliminate puerperal infections entirely.[13]

When they spoke of puerperal fever, however, most eighteenth-century authors meant more than simply any infection or fever following childbirth. They described a characteristic pattern of symptoms which delineated the most severe and frequently epidemic form of the disease. Hulme, for example, wrote that puerperal fever "is as much an original or primary disease as the ague, quinsy, pleurisy, or any other complaint."[14] It could be distinguished from after-pains, from milk fever (resulting from engorgement and an infected breast), from miliary fever (probably typhoid), from ileus, from colic, from local infections of the uterus, and from cholera. Charles White distinguished it from a "simple inflammation."[15] In 1790, Joseph Clarke distinguished puerperal fever from ephemeral fevers lasting only twenty-four hours following birth, and he further distinguished between epidemic puerperal fever and the incidental fevers that resulted from inflammation.[16] John Leake also differentiated between incidental and infectious cases.[17]

Nevertheless, considerable confusion remained. Thomas Kirkland commented that the term *puerperal fever* could be applied to any fever arising in consequence of pregnancy or delivery and occurring during the lying-in period. The fever itself was merely a symptom, not a disease, and could result from a number of different causes such as an infected uterus or an absorption of putrid effluvia from outside. He added, however, that puerperal fever should not be confused with diseases such as smallpox and typhus coincidentally contracted by women in the period after delivery.[18] John Clarke complained in 1793 that:

> the name of puerperal fever having been given indiscriminately to every febrile disease attacking women in childbed, has thus become a source of much incon-

[12] Shorter, *History of Women's Bodies,* chap. 6.

[13] For the importance of Group B infections see Dorothy I. Lansing, W. Robert Penman, and Dorland J. Davis, "Puerperal fever and the Group B beta hemolytic streptococcus," *Bull. Hist. Med.,* 1983, *57:* 70–80, and J. Wister Meigs, "Puerperal fever and nineteenth-century contagionism: the obstetrician's dilemma," *Trans. Stud. Coll. Phys. Philadelphia,* 1975, *42(3):* 273–80.

[14] Hulme, *Treatise,* FC, p. 61.

[15] White, *Treatise,* ed. Longo, p. 10.

[16] Joseph Clarke, *Observations on the Puerperal Fever, more especially as it has of late occurred in the Lying-in Hospital of Dublin* (1790), FC, pp. 351 and 362.

[17] Leake, *Practical Observations,* FC, p. 153.

[18] Thomas Kirkland, *A Treatise on Childbed Fevers and the Methods of Preventing Them* (1774), FC, p. 283.

> venience. Practical men, misled by this false bias, have persuaded themselves that the form of disease, which respectively they may have most frequently met with, is the only one.[19]

Some medical writers also included diseases that, to a modern eye, do not belong. Thus, Kirkland and White both seem to have confused the disease on some occasions with typhus, and many if not all of the cases in William Butter's 1775 tract seem to have been due to worms or other intestinal infections.[20] There was also fierce argument over whether peritonitis or hysteritis was the more characteristic form of the disease.

Despite this uncertainty, most authors depicted a particular cluster of symptoms that they believed distinguished puerperal fever both from other epidemic diseases such as smallpox and from less severe postpartum infections such as mastitis or uterine abscesses. A typical attack of puerperal fever occurred shortly after birth, generally on the second, third, or fourth day. The shorter the interval between birth and onset, the more dangerous the disease. It began with chills and shivering and was accompanied by a fast pulse and headache. Intense abdominal pain was frequent but not invariable. The patient usually remained conscious and often showed great fear or despair. The face was often flushed and the patient took short, quick breaths. Thomas Denman, in 1768, seems to have been the first English author to comment on the "erysipelatous appearance" of the joints.[21] There might be vomiting or suppression of the milk or lochia. The fever was dangerous, killing most of those attacked, but there was some variation in its severity. Autopsies showed large accumulations of pus and fluid in the abdominal cavity with other signs of inflammation which might extend to the lungs. Epidemics were more common during the cold months of the year.

The insistence of many experienced physicians that puerperal fever was not a simple infection, a local inflammation, or an incidental fever; the symptoms they recorded, such as the sudden onset of a high fever and flushing; the very high mortality rate, the autopsy descriptions, and the epidemic incidence—all strongly suggest that most of these cases were caused by one particular pathogen out of the many that may cause postpartum infections: the Group A beta-hemolytic streptococcus. This caused the terrifying epidemics that led obstetricians to take drastic measures and to publish their experiences. As Edward Shorter has pointed out, however, these epidemics caused fewer deaths overall than the sporadic infections that were often caused by other organisms, including Group B streptococci.

[19] John Clarke, *Practical Essays on the Management of Pregnancy and Labour; and on the Inflammatory and Febrile Diseases of Lying-in Women* (1791), FC, p. 389.

[20] William Butter, *An Account of Puerperal Fevers as they appear in Derbyshire* ... (1775): "Worms, either alive or dead, frequently come away in the stools," and "This puerperal fever is so far from being of a peculiar nature, that it is exactly analogous to the worm fever so fatal to children" (FC, pp. 337 and 343).

[21] Thomas Denman, *An Essay on the Puerperal Fever* (1768), FC, p. 48.

Severe infection with Group B strains may cause symptoms that are difficult to distinguish clinically from symptoms of infection with Group A, but Group B is usually a sporadic, rather than an epidemic, infection.[22]

The group letters were assigned in the late 1920s by Rebecca Lancefield, who also developed a way to distinguish among different strains within Group A.[23] This made it possible for researchers to investigate the differences between the Group A strains that normally cause pharyngitis and those that cause impetigo. Pharyngeal strains of Group A are now known to cause several different diseases which on a cursory inspection would appear to be unrelated. They include wound infections and septicemia, puerperal fever, sore throat, scarlet fever, erysipelas, thrombophlebitis, pneumonia, rheumatic fever and carditis, subcutaneous nodules, erythema marginatum, and chorea. While both throat and skin strains may cause kidney disease, only the pharyngial strains normally cause rheumatic fever, which was sometimes called "acute rheumatism" or "acute arthritis" in the eighteenth century. Occasionally the skin strains are found in the throat. The throat and skin strains have a different incidence. Skin strains tend to occur in warm climates and in warm months. Throat strains, on the other hand, are common in colder climates, such as that of northern Europe, and in the colder months of the year. The more virulent a strain is, the more infectious it appears to be. Scarlet fever is caused by particular strains of streptococci, but the scarlet fever strains vary in their virulence.

Because infection with any virulent strain of the Group A streptococci confers permanent immunity to that strain and may confer resistance to some less virulent strains, streptococcal pharyngitis is primarily a disease of children. If a virulent strain is of an uncommon type, however, it will attack persons of all ages. The bacteria rapidly lose virulence outside the body, and there are no significant animal hosts except for cows, who may transmit the disease in milk. To spread, the bacteria therefore require direct contact between a susceptible host and an infected person or a healthy carrier. Many people may contract subclinical infections or become healthy carriers, and most of those who experience untreated infections will continue to carry the disease for several weeks in their noses or throats. There are healthy carriers who retain the disease indefinitely, for months and maybe years, in many parts of the body, including the fingernails. Following an epidemic that attacked twenty women in the Boston Hospital in 1965, epidemiologists found that about 5 percent of the hospital staff and 10 to 15

[22] See nn. 12 and 13.

[23] Most of the following two paragraphs paraphrases Gene H. Stollerman's *Rheumatic Fever and Streptococcal Infection* (New York: Grune and Stratton, 1975), pp. 12–100. See also Daniel M. Musher, "The gram-positive cocci: I. Streptococci," *Hosp. Prac.*, 1988, *23*: 63–76. I would like to thank Robert Kimbrough for this reference, and the Infectious Diseases Department of the Oregon Health Sciences University for supplying a copy. See also Lewis W. Wannamaker, "The Streptococcal Siren," in *Infectious Disease Review*, ed. William J. Holloway, 10th Infectious Disease Symposium (New York: Futura Publishing, 1974), pp. 167–83.

percent of the neighboring community were asymptomatic carriers of hemolytic streptococci. Of the forty hospital carriers, four had positive throat cultures for Group A and one had a positive skin lesion.[24] Because it is a contact disease, streptococcal infection is closely related to urban crowding and poor housing conditions, and the rapid transfer of the disease from person to person under such circumstances results in especially virulent epidemics.

Lancefield's work also made it possible to trace individual cases of streptococcal infection to a probable source. In the 1930s, Dora Colebrook began a series of epidemiological investigations at Queen Charlotte's Hospital. She was able to culture pathogenic strains from many sources within the hospital, including dust, surgical gowns, and bedding. Several historians read this work and concluded that hospital fomites helped transmit the infection in the past, perhaps through airborne dust particles. For example, Leonard Colebrook wrote in 1954 of "dissemination through the air in dust particles, perhaps via bedding," and added that "streptococci could remain alive in dust and retain their virulence for several weeks."[25] Hospital administrators carried on a comprehensive campaign to expunge the bacteria from clothing, dust, and air. As late as 1973, David Charles and Thomas Klein wrote in a medical textbook:

> Postpartum staphylococcal and ... streptococcal infections ... are often airborne, either in droplets or dust. Viable bacteria can remain suspended in the air for long periods in minute droplets.... Larger droplets will contaminate the floor, bedside utensils, and fomites.... Inoculated dust may linger in the ward long after the source of contamination has departed, despite conventional floor mopping and waxing.... A more effective means of control is adequate ventilation.[26]

In fact, however, Colebrook had traced most actual infections to nasopharyngial carriers, not to hospital dirt. As high a proportion as half of all carriers may be asymptomatic. Following the Second World War, Charles Rammelkamp and his associates carried out a series of studies on airmen using streptococci found in the environment. They concluded that such bacteria do not remain viable for long outside the body.[27] Current infectious disease officers recommend a hospital practice that places less emphasis on the control of fomites and more emphasis on the isolation of carriers and the washing of hands between patients. This is designed to prevent the

[24] John Figgis Jewett et al., "Childbed fever: a continuing entity," *JAMA*, 1968, *206*: 344–50.

[25] Colebrook, "Puerperal Infection," in Kerr, Johnstone, and Phillips, eds., *Historical Review*, pp. 208–9 (quote on p. 209).

[26] Charles and Klein, "Postpartum Infection," p. 257.

[27] Stollerman, *Rheumatic Fever*, p. 15; and for Rammelkamp's group, see William Perry et al., "Transmission of Group A Streptococci: I. the role of contaminated bedding," *Amer. J. Hygiene*, 1957, *66*: 85–95.

direct transmission of infection from patient to patient and from attendant to patient.[28]

The unspoken comment of many historians on the physicians of the past seems to be "If only they had washed!" In fact, however, as Gail Parsons has pointed out, physicians who were carriers could have taken baths in chloride of lime without effect. Because carriers constantly recontaminate their own hands, handwashing, although essential, is not fully effective unless it is accompanied by methods to prevent recontamination such as the wearing of masks and gloves. Even after Ignaz Semmelweis instituted handwashing with chloride in Vienna, he experienced a maternal mortality rate of more than twenty-nine per thousand. The 1965 epidemic in Boston took place "without any evident breach of technique or good practice" and was traced to a small lesion on the hand of a physician-anesthetist.[29]

Even under the very different conditions in eighteenth-century hospitals, deficiencies in institutional cleanliness were probably overshadowed as a cause of infection by the presence of carriers and the extent of crowding in the wards. Once a case was introduced into a room, it would have required great luck and extraordinary vigilance to prevent the other patients in the room from contracting the infection, particularly since patients frequently tended to each other. Closing the ward entirely may have been the only effective measure available to eighteenth-century physicians. When a medical attendant became a carrier, even that course may not have been effective. Women could also contract the disease from infected children or visitors in their own homes, and some women brought the disease into the hospital with them.

THE INCIDENCE OF EPIDEMICS

The likelihood that cases would be introduced into hospitals depended in part on the prevalence and virulence of streptococcal infections in the surrounding community. Such infections were probably not new. There are several seventeenth-century descriptions of acute arthritis, a common symptom of rheumatic fever, which is caused by streptococcal infection. Sydenham's chorea, first described in 1686, is also a complication of rheumatic fever. Seventeenth-century accounts, however, suggest that at that time scarlet fever and other pharyngeal infections were usually mild.[30]

In the eighteenth century, England contained a much higher proportion

[28] F. D. Daschner, "The transmission of infections in hospitals by staff carriers: methods of prevention and control," editorial review in *Infect. Control*, 1985, *6*: 97–99. Elaine Larson, "A causal link between handwashing and risk of infection? Examination of the evidence," *Infect. Control*, 1988, *9*: 28–36, draws the same conclusion after a review of 423 articles published between 1879 and 1986.

[29] Parsons, "British medical profession," p. 148; Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*, ed., abridged, and trans. K. Codell Carter (Madison: University of Wisconsin, 1983), p. 143 (my calculation for the rate for 1847–49); Jewett et al., "Childbed fever," p. 344.

[30] Stollerman, *Rheumatic Fever*, pp. 2–3.

of young children than it does now, and its increasing population was crowding into large urban slums; these conditions favored the development and spread of virulent streptococcal epidemics. Although it seems likely that most women had become immune to common strains by the time they reached childbearing age, eighteenth-century accounts suggest that new, more virulent strains were attacking a population not yet widely immune to those strains.[31]

These accounts are ambiguous because of the difficulties in diagnosis, either contemporary or retrospective. Even with bacterial cultures, streptococcal pharyngitis may be difficult to diagnose, and in the eighteenth century it was commonly mistaken for diphtheria, typhoid and miliary fever, viral pharyngitis, and even mold poisoning. Scarlet fever is more distinctive, however, and the characteristic raspberry red rash and subsequent desquamation make a retrospective verdict of streptococcal infection probable.

There was a severe and highly fatal diphtheria epidemic in New England in the 1730s which apparently included some scarlet fever cases. There were reports from England and Scotland of similar epidemics in the same decade. When "epidemic sore throat" appeared in London in 1746, however, English physicians clearly believed they were confronting a disease previously unknown to them. John Fothergill's classic *Account of the Sore Throat Attended with Ulcers* described the English disease precisely enough to make it possible for us to conclude that it was streptococcal pharyngitis.[32] Fothergill's book also broke new ground in emphasizing the contagious nature of the disorder and in recommending a more conservative therapy, replacing extensive bleedings with prescriptions of cinchona bark and fluids.[33]

[31] Charles Creighton, *A History of Epidemics in Britain* (1894), 2d ed., with additional material by D. E. C. Eversley, E. Ashworth Underwood, and Lynda Ovenall, 2 vols. (London: Frank Cass, 1965), *2:* 678–744, esp. p. 685. See also R. Hingston Fox in *Dr. John Fothergill and His Friends: Chapters in Eighteenth-Century Life* (London: Macmillan, 1919). Fox comments on p. 51: "Heberden, writing in 1782, considered it highly probable that the malignant sore throat was a form of scarlet fever. And this must be our verdict today." He adds that earlier epidemics in Spain and Italy were probably diphtheria. In the eighteenth century, there were occasional scarlet fever epidemics of varying type. By the nineteenth century, they were more common (occurring every few years), and they were of greater average severity and mortality. Perhaps in Fothergill's time, Fox concludes, the disease was in the process of development.

[32] John Fothergill, *An Account of the Sore Throat Attended with Ulcers* (1748), republished as *An Account of Putrid Sore Throat* in vol. 1 of *The Works of John Fothergill, M.D.*, ed. John Coakley Lettsom (London: Charles Dilly, 1783). I would like to thank the National Library of Medicine (NLM) for supplying me with a microfilm copy of this work.

[33] *Ibid.*, esp. pp. 403–4. Fothergill was widely credited by contemporaries for encouraging milder treatments for many fevers but particularly for scarlet fever. For example, George Aspinwall wrote in 1798: "Blood-letting, purging, and the antiphlogistic regimen, in general, were formerly adopted as an universal practice; no wonder the disease proved a fatal one.... Dr. Fothergill [*sic*] wrote the first treatise on this disorder in England, and proposed a more rational method of treating the disease.... He condemned blood-letting, and the antiphlogistic regimen, till which time the lancet proved as fatal as the guillotine in the days of Robespierre." *A Dissertation on the Cynanche Maligna* (Dedham, Massachusetts: Mann and Adams), p. 5. I would like to thank the NLM for supplying a microfilm copy of this work. "How many lives were lost," William Withering asked, "until Dr. Fothergill and Dr. Wall taught us to withhold the lancet and the purge?" *An Account of the Scarlet Fever and Sore Throat* (London, 1779; 2d ed., Birmingham, 1793), quoted in Fox, *Dr. John Fothergill*, p. 53.

Was there any significance in the simultaneity of the appearance of puerperal fever epidemics in maternity hospitals and the appearance of community scarlet fever or sore throat epidemics? An epidemiologist was able to show such a relationship for the late nineteenth century. In 1879, G. B. Longstaff compiled graphs of the incidence of eighty-nine diseases for the years 1859 to 1878. Based only on the resemblance of their curves, he concluded that there was a relationship between scarlatina, erysipelas, puerperal fever, and rheumatic carditis. The relationship between puerperal fever and erysipelas was particularly close, and scarlatina was similar to the other three except that its curve anticipated theirs by about five weeks.[34] Some eighteenth-century commentators also noted that these diseases were found together: "It is a curious circumstance," wrote the obstetrician John Clarke in 1791, "that before the attack of the epidemic of lying-in women at Paris in the year 1746, in the month of January there had been an epidemic low fever, with an ulcerous sore throat."[35]

To determine whether this "curious circumstance" occurred more than once, I have compared the known epidemics of "ulcerated sore throat" and "scarlet fever" with the hospital epidemics of puerperal fever. The results, if not conclusive, are at least suggestive. For the incidence of "sore throat" I used the accounts compiled in the chapter "Scarlatina and Diphtheria" in Charles Creighton's *History of Epidemics in Britain.* I have added a few other references.

Creighton conceded that it was difficult and perhaps pointless to attempt to distinguish between scarlet fever and diphtheria in many eighteenth-century accounts, but he added that

> whether the disease were malignant scarlatina, or diphtheria, or a mixture of the two ... it was certainly new as a whole to British experience in that generation [of the middle third of the century], and, if we except the reference by Morton to certain cases which may have been sporadic, it was a disease hitherto unheard of in England since systematic medical writings began.[36]

In fact, Creighton did engage in retrospective diagnosis, and I have tried to follow his assessments. For the incidence of puerperal fever, I followed Fleetwood Churchill's introduction to the Sydenham Society's collection of eighteenth-century tracts on puerperal fever.

The accounts show that episodes of sore throat occurred in fairly well-defined epidemics. With each epidemic, physicians became more precise in their classification. London experienced such epidemics in 1746–48, 1751–52, 1755, 1770, 1772–73, 1777–78, and 1785–94. By 1796, "scarla-

[34] G. B. Longstaff, "On some statistical indications of a relationship between scarlatina, puerperal fever, and certain other diseases," *Trans. Epidemiol. Soc. London,* 1879, *4:* 421–30. I would like to thank Leonard G. Wilson for sending me a copy of this work.

[35] Clarke, *Practical Essays,* FC, p. 418.

[36] Creighton, *History of Epidemics,* 2: 702.

tina" seems to have become endemic. There was also an epidemic at the Foundling Hospital in 1763 which may have been measles. Epidemics took place in Sheffield in 1745, in Hertfordshire, Kidderminster, and Worcester in 1748–50, possibly in Plymouth in 1750–53, in Ireland in the 1750s, in Somerset in 1757, and in Yorkshire in 1759–60. No further reports appeared until 1770 in Manchester and 1771–72 in Ipswich, followed by an epidemic in 1777–79 in the areas around London, north Scotland, the Midlands, Newcastle, and Carlisle. In 1788 sore throat was reported in Buckinghamshire. This seems to have been streptococcal pharyngitis, but a report in 1793, this time of "croup," probably referred to diphtheria. With the exception of the area around London, England seems to have escaped without a serious epidemic between the late 1780s and 1801, but there were reports of "putrid sore-throat" in the North of Scotland, including Aberdeenshire, in 1790 and 1791.[37]

We know less about Ireland. James Sims, who practiced in County Tyrone from about 1764 until 1773, claimed he had not seen one instance of the malignant ulcerous sore throat as described by authors. Nevertheless, he reported epidemics of erysipelas in 1765–67 and 1772 and of "acute rheumatism" in 1767–68. John Rutty described what seems to have been diphtheria in 1743 and 1755 but what was referred to as "scarlet fever" in the winter of 1759–60.[38]

Between 1801 and 1805 severe sore throat epidemics were reported from such areas as Cornwall, Northampton, Cheltenham, Derby, Framlingham, Lancaster, Manchester, Liverpool, Yorkshire, and Edinburgh. In Dublin such an epidemic coincided with an epidemic of puerperal fever at the Rotunda in 1803. By the mid-nineteenth century, among the infectious childhood diseases, scarlet fever had become the leading cause of death. It was particularly fatal in England's large cities and industrial areas.[39]

Puerperal fever also occurred in epidemics. These epidemics were rarely confined to a single hospital. Reports of epidemics from widely scattered places throughout the British Isles and the Continent tend to cluster in a few "bad years," which also suggests that something other than conditions in individual institutions was involved. The first reported English epidemic of puerperal fever began in June 1760 in the British Lying-in Hospital in London and lasted until the end of December. The following spring, another London lying-in hospital experienced a fatal epidemic.[40] Maternal mortality rates in the city exceeded eighteen per thousand in 1761 and

[37] The 1770 Manchester epidemic is from Thomas Percival, *Observations on the State of Population in Manchester* (1789), reprinted in *Population and Disease in Early Industrial England,* ed. Bernard Benjamin (n.p.: Gregg International, 1973), p. 6. All others are from Creighton, *History of Epidemics,* 2: 678–722.

[38] James Sims, *Observations on Epidemic Disorders, with remarks on nervous and malignant fevers* ... (London, 1773), pp. 57–58 and 86; for Rutty, see Creighton, *History of Epidemics,* 2: 694.

[39] Creighton, *History of Epidemics,* 2: 719–28.

[40] Unless otherwise noted, all puerperal fever epidemics are listed in FC, pp. 3–42.

1762, the highest of the century, which suggests that these hospital epidemics were part of a larger epidemic in the city (see table 2). St. Thomas's Hospital experienced an epidemic of erysipelas in 1760 so severe it was rumored to be the plague, compelling the Governors to publish denials in the newspapers.[41] Sore throat epidemics were not reported for London in those years, but there was a puerperal fever epidemic in Aberdeen in 1760 and there were reports of sore throat in Yorkshire in 1761. The next London puerperal fever epidemic began in the late fall of 1769 and continued into 1771. It affected at least three hospitals and domiciliary births in the city, as well. There were sore throat epidemics in both London and Manchester in 1769–70.

There was only one more puerperal fever epidemic reported in London during the eighteenth century, in 1787–88. John Clarke wrote of this period that "in the year [*sic*] 1787 and 1788, the same year in which the disease seems to have been prevalent in Dublin, it was also exceedingly general through the whole of this country, but more especially in London, and in hospitals."[42] Indeed, the decade between 1784 and 1794 was a bad one throughout Europe, with epidemics in Vienna (1784), Copenhagen (1786), Lombardy (1786–87), Poitiers (1787), Aberdeen (1789–92), again in Copenhagen (1791–92), again in Vienna (1792–93), and in Amsterdam (1793), Rouen (1793), and Stockholm (1793 and 1794).[43] These coincided with sore throat epidemics in London in 1785–94 and Buckinghamshire in 1788, "acute arthritis" in Dublin in 1787, and "sore throat" in Aberdeenshire in 1790–91.[44] The records of the Aberdeen Dispensary, which were compiled by Alexander Gordon, show a high number of cases of erysipelas and sore throat in 1791 and of scarlet fever in 1792, when compared with the following two years. Gordon himself noted the connection between puerperal fever and erysipelas.[45]

There are two further possible puerperal fever epidemics in London. Fleetwood Churchill included 1783 in his table of puerperal fever epidemics, but I have not been able to find any further references to an epidemic in that year in the text of his book, either in his introduction or the treatises that follow, several of which include historical surveys.[46] I am therefore inclined to discard this as a printer's error. I am also discarding an

[41] William C. Wells, "Observations on erysipelas," *Trans. Soc. Improving Med. Chir. Knowledge,* 1800, 2: 213–28, esp. 218–19.

[42] John Clarke, *Practical Essays,* FC, p. 414.

[43] Hirsch, *Handbook,* 2: 422–24. A previously unknown puerperal fever epidemic in the Vaugirard hospital in 1791 is reported in Mireille Laget, "Childbirth in Seventeenth- and Eighteenth-Century France: Obstetrical Practices and Collective Attitudes," in *Medicine and Society in France: Selections from the Annales,* ed. Robert Forster and Orest Ranum, trans. Elborg Forster and Patricia M. Ranum, vol. 6 (Baltimore, Maryland: Johns Hopkins University Press, 1980), p. 151.

[44] The "acute arthritis" in Dublin is from Joseph Clarke, *Observations,* FC, p. 354.

[45] Gordon, *Treatise,* FC, pp. 499–500.

[46] See FC, p. 31. That year saw one of the lowest maternal mortality rates for London in the century, 8.5 per thousand.

account by William Butter of the "puerperal fever" that appeared in Derbyshire between 1765 and 1775 because I do not believe from his account that he saw cases of streptococcal puerperal fever. Including these years would not affect the conclusion, however. The records of the British Lying-in also show unusually high mortality rates in 1774 and 1775: the death rate in 1773 was 6.3, but in 1774 it was 32.3 (see table 3). This followed a sore throat epidemic in London in 1772–73, and coincided with epidemics of severe puerperal fever in Paris and Dublin in 1774–75. The British Lying-in also experienced "bad years" in 1781–82 and 1784, when the mortality rate rose above twenty per thousand. This evidence is ambiguous: fourteen women died in 1781 compared with eight the previous year; with numbers this small the additional deaths could be due to bad luck or isolated fever cases. About five hundred women a year were delivered by the British Lying-in, about 3 percent of all the women delivered in the city of London.

To summarize: there were three reported London puerperal fever epidemics, in 1760–61, 1769–71, and 1787–88. Possibly there was an epidemic in 1774–75, and less possibly one in 1783. In Scotland, there were epidemics in Aberdeen in 1760 and 1789–92 and in the Edinburgh Infirmary in 1773. The London epidemic of 1760–61 coincided with "sore throat" reported in Yorkshire and Sheffield but not in London, with "scarlet fever" in Ireland, with erysipelas in a London hospital, and with the Aberdeen puerperal fever epidemic. The London epidemic of 1769–71 coincided with "sore throat" epidemics in London and Manchester. The London epidemic of 1787–88 coincided with a sore throat epidemic in London. The possible epidemic of 1774–75 followed a London sore throat epidemic in 1773. Both Aberdeen epidemics coincided with reports of sore throat, one rather far away, in North Yorkshire, the other in Aberdeenshire itself. The Edinburgh epidemic of 1773 is the exception, since there are no reports of sore throat from Scotland or even the North of England during this period. However, it may have been part of a pandemic that included sore throat in London in 1773 and puerperal fever epidemics in London, Dublin, and Paris in 1774–75. This epidemic was especially disastrous in Paris, where it attacked seven out of every twelve women who delivered and subsequently became endemic.[47]

The Rotunda in Dublin experienced between three and four epidemics in the eighteenth century, in 1767, 1774, and 1787 and 1788, depending on whether the latter two are counted as one or two epidemics.[48] As seen above, the 1774 epidemic and that of 1787–88 coincided with other puerperal fever epidemics. The 1767 epidemic coincided with reports of erysipelas and acute rheumatism in County Tyrone. The 1787 epidemic coincided with "acute arthritis" in Dublin. There was also an epidemic in 1803 which coincided with reports of scarlatina throughout the British Isles.[49]

[47] FC, pp. 8–9. The Edinburgh Infirmary also experienced an epidemic of erysipelas in 1773, but the disease did not appear in the town (FC, p. 226).

[48] O'Donel T. D. Browne, *The Rotunda Hospital, 1745–1945* (Edinburgh: E. and S. Livingstone; Baltimore: Williams and Wilkins, 1947). See also Joseph Clarke, *Observations*, FC, pp. 351–62.

The small number of hospital epidemics in the eighteenth century is in itself surprising, because the secondary literature suggests that hospital epidemics were a constant problem. I am inclined to believe that hospital epidemics of puerperal fever were in fact unusual events in Britain and did not account for a large number of deaths, and that few significant epidemics in major cities escaped comment. Comparing the London hospital reports with the total maternal mortality for the city (see below), one finds the two series to be very similar, increasing the persuasiveness of each. The London reports can also be compared with the comprehensive set of records for the Rotunda.

Comments from obstetricians of the time also suggest that puerperal fever epidemics were unusual in the eighteenth century. For example, Alexander Gordon wrote that puerperal fever was unknown in Aberdeen between the epidemics of 1760 and 1789–92, and that he was the only physician in the city who could recognize a case, because he had seen it in London.[50] Charles White, who had practiced in Manchester for more than twenty years, commented in 1773 that he had never lost a patient to puerperal fever following a natural labor, and that such fevers had greatly decreased.[51] His assertion is borne out by the fragmentary mortality data for Manchester.[52] (Both Gordon and White, however, refer to a much less dangerous affliction known in the North as "the Weed.")[53] "Happily for the Fair sex, it does not often occur," commented the statistician William Black in 1789.[54]

Some historians believe that the low hospital mortality rates from eighteenth-century British institutions were simply falsified.[55] This undoubtedly happened occasionally. White wrote of the London epidemic of 1760–61 that:

> a gentleman whose veracity I can depend on . . . attended a small private lying-in hospital . . . he himself delivered six women in a short time . . . and they all died:

[49] FC, p. 12; Creighton, *History of Epidemics*, 2: 721.

[50] Gordon, *Treatise*, FC, pp. 445–500, esp. pp. 447–48. On Gordon, see Ian Porter, *Alexander Gordon, M.D. of Aberdeen, 1752–1799* (London: Oliver and Boyd, 1958).

[51] White, *Treatise*, ed. Longo, p. 64. On White, see J. George Adami, *Charles White of Manchester (1728–1813) and the Arrest of Puerperal Fever* (Liverpool and New York: Paul B. Hoeber, 1923), and Charles J. Cullingworth, *Charles White, F.R.S., a Great Provincial Surgeon and Obstetrician of the Eighteenth Century* (London: H.J. Glaister, 1904).

[52] White, *Treatise*, ed. Longo, p. 138; and see Percival, *Observations*, p. 7: "Puerperal diseases also decrease every year amongst us."

[53] "The Weed" seems to have been an ephemeral fever.

[54] William Black, *An Arithmetical and Medical Analysis of the Diseases and Mortality of the Human Species*, 2d ed. (London: Charles Dilley, 1789), p. 217. I would like to thank the NLM for supplying me with a microfilm copy of this work.

[55] See McKeown and Brown, "Medical Evidence," p. 289.

> he was so shocked with the loss that he desired the gentleman who had the care of the hospital to deliver some of those who should next be in labour, which he did, but they met with no better fate. They buried two women in one coffin, to conceal their bad success.

White adds, however, "Several gentlemen of the faculty were invited to the hospital to inquire into the cause of this great fatality"—so apparently it was no secret to the profession.[56] Efforts at concealment became more subtle as time went on. At the beginning of the next century, a Master of the Rotunda was dismissed for sending women ill of puerperal fever home to die. Near the end of the century, American clinics simply bribed infected pregnant women to have their babies elsewhere.[57]

Nevertheless, as a group, eighteenth-century hospital statistics are in line with each other and with statistics from other sources, such as urban maternal mortality rates. Moreover, there is no reason to believe that hospital administrators were more anxious to conceal the presence of puerperal fever in the eighteenth century, when it seemed to appear inexplicably, than in the nineteenth century, when the administrators had at least some cause for suspecting that they themselves were to blame, or even in the twentieth century, when puerperal fever was universally conceded to be a preventable, nosocomial disease. Dishonesty may explain why all the figures are lower than they should be, but dishonesty is probably randomly distributed: there is no reason for believing that eighteenth-century English hospitals were unique in "fudging" their rates.

If puerperal fever epidemics appeared infrequently in British hospitals in the eighteenth century and usually coincided with the appearance of particularly virulent community streptococcal infections, then the traditional argument that puerperal fever was caused by the rise of the maternity hospital and of the male midwife with his autopsies, forceps, and dirty hands should be reconsidered. That simultaneous puerperal fever epidemics took place in widely separated places also suggests that something other than the practices of a particular physician or the cleanliness of a particular hospital was involved. It may have been a coincidence that more virulent strains of streptococci appeared in England in the second half of the eighteenth century, just about the time many maternity hospitals were established. These events may be causally linked, but probably because they are closely related to a third factor: changes in living conditions and particularly in the extent of urbanization, which made obstetrics more lucrative and hence attractive to male practitioners. The poverty and overcrowding that compelled women to enter a hospital also contributed to the virulence and infectiousness of the bacteria. The case is not conclusive, but it is sufficient to raise doubts about the traditional explanation. Maternity hospitals in the eighteenth century may have magnified epidemics, but they do not seem to have caused them.

[56] White, *Treatise,* FC, pp. 232–33.

[57] Lansing, Penman, and Davis, "Puerperal fever," p. 75.

MATERNAL MORTALITY

We will now turn from consideration of the number and timing of puerperal fever epidemics to an examination of the effect of these epidemics on total maternal mortality. Was puerperal fever killing a large number of women? Was its impact on mortality rates increasing? Did an increased risk of fever make hospital deliveries much more dangerous than home births? Recent work, including articles by Irvine Loudon and Edward Shorter, has argued persuasively that during the course of the eighteenth century the overall maternal mortality rate fell substantially, and that this trend was due in large part to improvements in obstetrical care.[58] It seems evident that this overall reduction could not have occurred without a reduction in the rate of postpartum infection. My own research fully bears out Shorter's assertion that birth attendants, whether male or female, were intervening less frequently in normal labor and were intervening more successfully in preternatural labor. It seems likely that this resulted not only in fewer deaths during labor but also in fewer deaths from infection following labor.

The overall trend can be seen in maternal mortality rates for the city of London. From the Bills of Mortality, Audrey Eccles obtained a rate of 21 per thousand in the second half of the seventeenth century.[59] Estimates from the Bills of Mortality reveal a rate of about 15 per thousand in the first decade of the eighteenth century, about 13 per thousand in the middle of the century, and about 9 per thousand in the last decade of the century (table 1).[60] This picture is not atypical for England as a whole or for Europe, although there were, of course, local variations. Some healthy villages with skilled midwives had achieved rates as low as 4 per thousand much earlier, but rates of between 10 and 20 per thousand were common, as was a decline by the end of the century. For example, Shorter has found a rate of 12 per thousand in Berlin before 1784, falling after that to 7.[61] Sweden had a death rate of between 8 and 10 per thousand in the second half of the century.[62] Manchester had a slightly better record: its rate, as reported by Charles White,

[58] See n. 7.

[59] Audrey Eccles, "Obstetrics in the seventeenth and eighteenth centuries and its implications for maternal and infant mortality," *Bull. Soc. Social Hist. Med.,* 1977, *20:* 8–11, cited in Shorter, *History of Women's Bodies,* pp. 98–99, and Loudon, "Deaths in childbed," pp. 13–14.

[60] My calculations are from William Heberden, *Observations on the Increase and Decrease of Different Diseases, and Particularly of the Plague* (London, 1801), reprinted in Benjamin, ed., *Population and Disease in Early Industrial England* (see appendix and table notes).

[61] Shorter, *History of Women's Bodies,* pp. 98–99. Shorter has added estimates for abortive and stillborn births to the denominator for this series.

[62] Ulf Högberg and Stig Wall, "Secular trends in maternal mortality in Sweden from 1750 to 1980," *Bull. World Health Org.,* 1986, *64:* 79–84.

was under 10 per thousand in the 1750s and 5.1 per thousand in 1771.[63] Edinburgh's rate was 14 in the 1750s and 6 in the 1790s.[64] By the mid-nineteenth century, the rate throughout Europe and Great Britain was between 4 and 5 per thousand.[65]

It is difficult to see how rates that low could be achieved without frequent and successful medical intervention, by either midwives or obstetricians. Loudon estimated that the death rate in unattended deliveries might have been between 25 and 30 per thousand deliveries.[66] Shorter does not provide a clear estimate, but his data suggest a higher rate. By the end of the eighteenth century the reported mortality rate for all deliveries was one-third to one-sixth that amount. Even when we allow for substantial inaccuracies in the rates, therefore, both the trend and the absolute magnitude suggest that the obstetrical revolution was contributing to improved outcomes. As Loudon commented:

> How was it possible for maternal mortality rates as low or lower than the national rates of the 1920s and early 1930s to be achieved before the introduction of anaesthesia, antisepsis, or twentieth-century methods? The answer is probably that the management of normal labour, and of the common complications, although remarkably poor at the beginning of the eighteenth century, was remarkably good . . . at the end.[67]

In hospitals, however, the story was very different. Continental hospitals in the nineteenth century were very dangerous places. The worst record was possibly that of the Paris Maternité, which in the years between 1861 and 1864 achieved an astounding maternal mortality rate of 184 per thousand.[68] Women must have been desperate indeed to face a nearly one-in-five chance of dying in each labor. Other European hospitals also experienced in bad years rates well in excess of one hundred per thousand, and in 1866 the average rate for a large number of European hospitals was 34.2 per thousand. Thus, a "typical" maternity patient in the mid-nineteenth century faced a chance of dying in hospital approximately seven times greater than the risk she would encounter in a home delivery. Nineteenth-century British hospital maternal mortality rates were considerably lower

[63] White, *Treatise,* ed. Longo, p. 138. White divided the deaths of mothers in childbirth by the figure for christenings without adding in an estimate for children dead before christening, the "abortive and stillbirth" figure. That is why his figure for London is slightly higher than Shorter's. Longo, in his introduction to White's *Treatise,* has printed a useful table of White's data (pp. xvi–xvii). The table includes a figure for the Manchester infirmary, but as far as I can tell from the text, White's figures for the infirmary are for all infirmary patients and have nothing to do with maternal mortality.

[64] Shorter, *History of Women's Bodies,* p. 99.

[65] William Gilliatt, "Maternal Mortality—Still-birth and Neonatal Mortality," in Kerr, Johnstone, and Phillips, *Historical Review,* pp. 264–65.

[66] Loudon, "Deaths in childbed," p. 18.

[67] *Ibid.*

[68] Gilliatt, "Maternal Mortality," in Kerr, Johnstone, and Phillips, *Historical Review,* pp. 264–65; Burtenshaw, "Fever of the puerperium," p. 1191 (for maternal mortality at the Maternité); Buer, *Health, Wealth, and Population,* p. 270.

than this, but they still rose on occasion to between 30 and 80 per thousand.[69]

The question is whether, as McKeown and Brown argued, hospital mortality rates in the eighteenth century were no better and probably much worse than these nineteenth-century rates. Was the advent of the hospital itself responsible for these tragic events? Charles White believed that hospitals did indeed increase the risk to mothers. In his *Treatise on the Management of Pregnant and Lying-in Women* (1773) he commented that "the fatality that attends the patients in some of the lying-in hospitals greatly exceeds that of any private practice, at least any that I have been acquainted with."[70] White then provided mortality figures for four unnamed London hospitals, figures that have been widely cited by historians. My tentative identifications of these hospitals are in parentheses in what follows. "A public lying-in Hospital" (Westminster Hospital) had a rate of 25.7 between 1767 and 1772. "Another Hospital" (the British Lying-in) had a rate of 21.5 for the period between 1749 and 1770 but 39.3 for 1770 itself. "Another Hospital" (possibly the Middlesex Hospital Lying-in wards) had a rate of 19.5 for 1747–72 and 35.4 for 1771. The last (the General Lying-in Hospital in Store Street) had a rate of 7.6 for 1767–72.[71] White, however, was writing immediately after the epidemic of 1770–71, which was one of the worst years of the century for the hospitals. His longer-term rates of about 20 per thousand may be compared with rates for the city of London and the Rotunda of between 10 and 15 per thousand for the middle of the century. This suggests that in London, hospital deliveries were somewhat more dangerous than home births in the 1760s and 1770s, but the figures are heavily weighted by the epidemics of 1760 and 1770–71 and tell us little about comparative risk in other years. There is a further difficulty with White's figures: his figure of 35 women of 890 who died in 1770, a figure that is also quoted by Fleetwood Churchill for the British Lying-in. "Another Hospital from its first institution in November 1749" can only be the British Lying-in, but the numbers themselves are considerably at variance with those of the hospital's own reports, which were published by William Heberden.[72] These are 28 dead of 472 delivered in 1770. Since the British Lying-in never had as many patients in one year as 890, White either is in error or is combining data from various years.

Additional information can be obtained from other sources. William Heberden published rates taken from the reports of the British Lying-in Hospital, which opened in 1749. These can be compared with rates for the city of London, the Rotunda in Dublin, and the Vienna Hospital maternity

[69] Gilliatt, "Maternal Mortality," in Kerr, Johnstone, and Phillips, *Historical Review,* pp. 261–63, 265.

[70] White, *Treatise,* ed. Longo, p. 135.

[71] *Ibid.*

[72] "The Account of the Women Delivered, and Children Born, in the British Lying-in Hospital from . . . 1749 . . . 1801," in Heberden, *Observations,* pp. 39–41.

wards. Annual figures are also available for the City of London Lying-in Hospital after 1790 (see table 1; see appendix for my sources).

How credible are these rates? The London rates are based on the "childbed" rates compiled from the London Bills of Mortality by William Heberden, a close friend of Fothergill's.[73] Heberden published maternal mortality rates as a percentage of all deaths: he did not give the total number of maternal deaths. I have recalculated them to give the maternal death rate in proportion to the number of christenings. All contemporary authors complained about the inadequacy of the London data. The main complaint was that the "searchers" who assigned the cause of death did not know what they were doing and came up with strange diagnoses such as "rising of the lights." There were also, however, intractable technical problems having to do with shifts in the urban population, undercounting of Dissenters (particularly in the case of christenings), and the removal of many infants from the city. Several authors complained that cases of postpartum infection were frequently misdiagnosed as other diseases such as measles. Despite these shortcomings, the London figures do at least provide an internally consistent long series.

The London figures compare the number of maternal deaths to the number of christenings. Multiple births increased the number of christenings relative to the number of mothers (the rate of multiple births at the British Lying-in was about twelve per thousand; applied to the entire city, that rate would yield about two hundred additional births). On the other hand, the large number of stillbirths and neonatal deaths lowered the number of christenings relative to the number of mothers, as did the rapid removal of infants from the city and the avoidance of Anglican christenings by Dissenters. Black estimated the "abortive and stillbirth" figure at about seven hundred a year, which would reduce the death rate in table 1 by a little less than one death per thousand. Both the practice of removing infants from London and the avoidance of christenings seem to have increased in the course of the century. It seems likely, therefore, that the real reduction in the rates in this period would have been greater than the recorded change.

[73] Heberden, *Observations.* There are significant discrepancies between Table I, "Of the Annual Christenings and burials in London for each Year of the Eighteenth Century; Together with the Proportion out of every Thousand, who have died by Bowel Complaints, Small Pox, Palsy, Measles, or Childbirth—from the Bills of Mortality," and Table II, "Of Ten different Articles extracted from the London Weekly Bills of Mortality, shewing their Variations every Week for Ten Years."

James Young, "Journals, 1800–1950," in Kerr, Johnstone, and Phillips, *Historical Review,* on pp. 324–25, supplies a series taken from the *London Medical and Physical Journal* for 1816 (vol. 36), which in turn took them from the Bills of Mortality. This supplies ten-year average mortality rates for London. Comparing these figures with my calculations from Heberden's annual rates yielded rates that were very similar to those of Heberden, but slightly lower. The difference varied, but it averaged about 0.6 per thousand births. No details are supplied, but this discrepancy is presumably because the *London Medical and Physical Journal* series used the same data as Heberden but added a figure for the "stillborn and abortive" births. I would like to hear from anyone who can account for the discrepancies in Heberden's rates, correct the Bills of Mortality figures for maternal mortality, or supply additional data.

Table 1. Maternal Mortality, Four Maternity Hospitals and London
(Five-Year Averages: Deaths)

	British Lying-in (deaths per 1,000 births)	City of London Lying-in (deaths per 1,000 deliveries)	Rotunda, Dublin (deaths per 1,000 deliveries)	Vienna Maternity (deaths per 1,000 births)	London (deaths per 1,000 christenings)
1750–54	32.5				12.8
1755–59	14.7		15.1*		13.2
1760–64	30.7		14.9		16.7
1765–69	11.4		14.3		12.7
1770–74	20.6		14.8		12.2
1775–79	15.3		8.6		11.4
1780–84	19.2		8.0		9.6
1785–89	12.4		12.3	5.7	10.5
1790–94	3.9	5.3	10.7	10.7	9.3
1795–99	3.0	4.6	6.0	9.2	9.0
1800–1804	4.6**	5.6	14.1	8.5	9.4‡
1805–1809		4.1	6.4	7.2	9.4
1810–14			14.0	16.9	9.1‡
1815–19			13.1	19.9	
1820–24			14.1	33.9	
1825–29			17.0	54.1	
1830–34		6.8†	7.5	52.9	
1835–39		17.3†	17.0	60.6	
1840–44		7.6†	10.4	100.8	
1845–49		27.7†	19.1	55.6	

SOURCES: The London figures are my calculations from Heberden, *Observations,* whose figures are from the London Bills of Mortality. See also appendix.
* 1758–59
** Figure for 1800–1807 from Buer, *Health, Wealth, and Population.*
† Figures for 1830–49 from *138th Report of the City of London Lying-in Hospital.*
‡ London Bills of Mortality for 1801–10 and 1811–14 from Young, "Journals," Kerr, Johnstone, and Phillips, *Historical Review,* p. 325.

Whatever their inadequacies, these rates are comparable to those of Edinburgh in the 1750s which were quoted by a modern demographer, somewhat higher than those of Edinburgh in the 1770s and 1790s, and higher than those of Manchester and Northampton which were quoted by Charles White.[74] When these rates are compared year by year with those of the British Lying-in, a complex story emerges (tables 2 and 3). From 1751 until 1755 the British Lying-in rates were much higher than those for the city: overall they were about twice as high. From 1756 until 1788 they fluctuated far more than did the city rates. In many years they were similar to or lower than city rates, but in epidemic years they rose much higher.

Although the average death rate was higher in the British Lying-in, most of the excess mortality was due to increased mortality in the years 1760–61, 1770, 1774–75, 1781–82, and 1784. If we use non-epidemic years as a guide to the overall level of obstetrical care at the hospital, then for most of the second half of the century it was comparable to that in the city as a whole. Even in its worst years, the British Lying-in had a good record compared with that of nineteenth-century hospitals.

There is some evidence that the British Lying-in and other hospitals took higher-risk cases. A report of 1805 commented, "Women who are the most deformed or who are in very bad health, in general take the most pains to procure letters of admission.... Many would have died of disease, if they had not been with child."[75] And John Clarke noted:

> It has been remarked, in the way of objection to lying-in hospitals, that the disease has not been so frequent among the poorer classes of women, who are delivered at their own habitations; but it is to be remembered that their situation is hardly ever so distressed as that of those who are the general objects of charity in hospitals; —women without a home, without friends, without husbands, without protection, and without the common necessaries of life before they were admitted.[76]

City death rates also increased in epidemic years, but not nearly by the same amount, increasing by about 5 per thousand instead of by 50. The epidemics of 1760–61, 1770, and 1787 appear clearly in the rates (table 2). Some of this increase is due to the very high rates of death in the hospitals in epidemic years. Between about 15,000 and 19,000 children were christened in London each year. Each 15 to 20 hospital deaths, therefore, would push the overall city death rate up by 1 per thousand. In the 1760 epidemic,

[74] Shorter, *History of Women's Bodies,* pp. 98–99.

[75] Cited in Buer, *Health, Wealth, and Population,* p. 145.

[76] John Clarke, *Practical Essays,* FC, p. 433. See also *An Account of the British Lying-in Hospital for Married Women ... 1749–1763* (London: C. Say, 1763): "Of the 121 women who have died in the Hospital, most of them came in not only under Circumstances of Distress and Poverty, in common with the Rest of the Patients, but also afflicted with dangerous Disorders, exclusive of their State of Pregnancy" (p. 15). Like the other maternity hospitals, the British Lying-in officially barred women with contagious diseases.

Table 2. Maternal Mortality, London
(Deaths per Thousand Christenings)

1701	14.3	1725	14.1	1750	15.8	1775	10.8
1702	14.2	1726	13.1	1751	12.0	1776	11.2
1703	14.1	1727	12.3	1752	10.6	1777	12.2
1704	16.7	1728	12.9	1753	11.4	1778	10.1
1705	17.8	1729	14.6	1754	14.6	1779	12.7
1706	15.4	1730	15.6	1755	13.7	1780	11.5
1707	16.0	1731	14.6	1756	12.1	1781	12.3
1708	15.4	1732	12.5	1757	12.7	1782	8.1
1709	14.0	1733	17.2	1758	13.1	1783	8.5
1710	14.5	1734	15.5	1759	14.2	1784	7.9
1711	13.2	1735	11.6	1760	15.9	1785	9.3
1712	13.3	1736	12.4	1761	18.4	1786	11.3
1713	11.1	1737	16.8	1762	18.2	1787	12.5
1714	17.6	1738	16.1	1763	17.1	1788	10.1
1715	16.1	1739	16.2	1764	14.1	1789	9.7
1716	13.2	1740	15.2	1765	15.3	1790	7.9
1717	13.1	1741	17.4	1766	12.5	1791	8.6
1718	14.3	1742	15.0	1767	11.0	1792	10.4
1719	15.9	1743	11.9	1768	13.3	1793	9.9
1720	14.8	1744	13.0	1769	11.2	1794	9.8
1721	16.4	1745	14.2	1770	16.0	1795	8.1
1722	16.0	1746	13.1	1771	10.5	1796	10.8
1723	15.2	1747	14.2	1772	10.9	1797	11.1
1724	12.7	1748	14.3	1773	11.6	1798	8.1
		1749	13.1	1774	12.2	1799	7.1
						1800	8.8

SOURCES: My calculations from Heberden, *Observations,* whose figures are from the London Bills of Mortality. See also appendix.

24 women died in the British Lying-in Hospital alone, thus contributing to some of the increase in the overall mortality rate. In the epidemic of 1770, John Leake found from the Bills of Mortality that the number of deaths in childbed rose by about one-third, or about 100 deaths, to a total of 272, and that this increase largely occurred between December and May—a typical pattern for streptococcal infections.[77] This raised the city's death rate from 11 to 16 per thousand. According to Heberden's summary of the British Lying-in reports, 28 died there of 472 delivered. This number represented a hospital death rate of nearly 60 per thousand and contributed about 1.6 per thousand to the city's mortality rate. There can be no question that a hospital epidemic could cause death rates to soar within the hospital to levels far above the average in the community.

[77] Leake, *Practical Observations,* FC, p. 125.

Table 3. Maternal Mortality, British Lying-in Hospital (Deaths per Thousand Born)

1750	16.9	1765	15.8	1780	14.1	1795	3.2
1751	35.2	1766	16.7	1781	26.4	1796	1.6
1752	32.0	1767	7.0	1782	23.3	1797	4.8
1753	34.8	1768	5.1	1783	8.4	1798	3.5
1754	36.8	1769	12.2	1784	25.2	1799	1.9
1755	24.0	1770	59.1	1785	13.5	1800	0
1756	8.1	1771	7.3	1786	14.8		
1757	14.5	1772	6.6	1787	15.7		
1758	15.1	1773	6.3	1788	17.1		
1759	12.5	1774	32.3	1789	1.7		
1760	59.9	1775	36.5	1790	11.1		
1761	30.4	1776	5.4	1791	1.6		
1762	17.6	1777	9.9	1792	1.6		
1763	23.7	1778	19.0	1793	1.7		
1764	19.0	1779	5.3	1794	3.4		

SOURCES: Heberden, *Observations*. See also appendix.

In the final years of the century, however, both the British Lying-in and the City of London Lying-in achieved a very low mortality rate (table 1). From 1790 until 1810, the City of London Lying-in Hospital rate was about 5 per thousand. Between 1791 and 1807 the rate at the British Lying-in was about 4 per thousand. These were about half the rate in London in the same period. The Rotunda achieved an average of 6.0 from 1795 to 1799, but it was unable to maintain this rate in the early nineteenth century (tables 1 and 4). The rates at the Vienna Maternity Hospital also fell to low levels for many individual years between 1786 and 1810, although epidemics in 1793 and 1795 raised the overall average for the 1790s to about 9.8 per thousand. In 1797 and 1798 the rate fell to 2.4. It is far more likely that these rates were genuine than that the management at several different hospitals simultaneously decided to falsify rates for a short time and then simultaneously decided to abandon the deception in the second decade of the nineteenth century. Such low rates could be achieved only by highly skilled obstetrical management, but they were attainable at that time. The Westminster Dispensary, which delivered women at home, had maintained an average rate of slightly under 4 per thousand since 1774.[78]

Because of the great variation in hospital rates in the course of the century, and the overall decline during the century, statements about the relative safety of hospitals and home births are not very informative when they rely on averages or on selected individual years. Overall, hospital deliveries were slightly more dangerous than those at home, but for women who had reason to believe they were at high risk and needed skilled delivery, a hospital delivery may have been a wise choice. In most years, the difference was not great. If a woman was unlucky enough to enter a hospital during an institutional epidemic she faced a considerable added risk, but such episodes were rare. Throughout the century, though particular years could be more dangerous, on average women were as safe at the Rotunda in Dublin as they would have been at home in the city of London (tables 2 and 4). By the end of the century, a London woman seems to have been

[78] Loudon, "Deaths in childbed," p. 16.

Table 4. Maternal Mortality, The Rotunda, Dublin

	Clarke, "Observations"			Semmelweis, *Etiology*			
Year	Delivered	Died	Deaths per 1,000 women delivered	Year	Delivered	Died	Deaths per 1,000 women delivered
1758	454	8	17.6				
1759	406	5	12.3	1784	1261	11	8.7
1760	556	4	7.2	1785	1292	8	6.1
1761	521	9	17.3	1786	1351	8	5.9
1762	533	6	11.3	1787	1347	10	7.4
1763	488	9	18.4	1788	1469	23	15.6
1764	588	12	20.4	1789	1435	25	17.4
1765	533	6	11.3	1790	1546	12	7.7
1766	581	3	5.2	1791	1602	25	15.6
1767	664	11	16.6	1792	1631	10	6.1
1768	655	16	24.4	1793	1747	19	10.8
1769	642	8	12.5	1794	1543	20	12.9
1770	670	8	11.9	1795	1503	7	4.6
1771	695	5	7.2	1796	1621	10	6.1
1772	704	4	5.7	1797	1712	13	7.5
1773	694	13	18.7	1798	1604	8	4.9
1774	681	21	30.8	1799	1537	10	6.5
1775	728	5	6.9	1800	1837	18	9.7
1776	802	7	8.7	1801	1725	30	17.4
1777	835	7	8.4	1802	1985	26	13.0
1778	927	10	10.8	1803	2028	44	21.6
1779	1011	8	7.9	1804	1915	16	8.3
1780	919	5	5.4	1805	2220	12	5.4
1781	1027	6	5.8	1806	2406	23	9.5
1782	990	6	6.1	1807	2511	12	4.7
1783	1167	15	12.9	1808	2665	13	4.8
1784	1260	11	8.7	1809	2889	21	7.2
				1810	2854	29	10.1
				1811	2561	24	9.8
				1812	2676	43	16.9
				1813	2484	62	24.9
				1814	2508	25	9.9
				1815	3075	17	5.1
				1816	3314	18	5.4
				1817	3473	32	9.2
				1818	3539	56	15.8
				1819	3197	94	29.4

NOTE: For more information on the sources, see appendix.

considerably safer in a maternity hospital than she was at home, although the home delivery charities seem to have had an impressive record.

These low rates, as low as those of the early twentieth century, could only have been achieved by a combination of excellent obstetrical skills and the increasingly successful avoidance of puerperal fever. Some of the improvement might have been due to a decline in the prevalence of community streptococcal infections, but this seems unlikely in the case of London hospitals, since scarlatina was reported as a constant problem in the London area during the last years of the century. Overall, streptococcal infections seem to have been increasing in incidence and virulence from the middle of the eighteenth to the middle of the nineteenth century.

EIGHTEENTH-CENTURY DISEASE THEORY

There is some reason to believe that English obstetricians and hospital management committees took measures that may have helped to reduce the risk of infection or cross-infection. English hospitals in the eighteenth century did not often perform extensive surgical operations, a factor that contributed to the comparatively low rate of wound infection and erysipelas. They generally refused admission to persons suffering from contagious diseases, including scarlet fever and ulcerated sore throat, which had been classified as contagious by Fothergill in 1748. They also frequently refused admission to children, the most frequent carriers of streptococcal infections.[79] Although they had difficulty controlling an epidemic once it appeared in a hospital, they may have been effective in minimizing the risk that such infections would be introduced in the first place.

Pregnant women in England received even more protection because they were often separated entirely from other patients in specialized institutions. There is some indication that this was a deliberate policy in England and was developed by some doctors with the idea that it might help prevent cross-infection. The case of William Hunter and his associates is particularly interesting. He was one of a group of obstetricians who resigned from the Middlesex Hospital in 1749 when it refused to establish separate maternity wards or to turn over the whole hospital to maternity cases. The group then formed the British Lying-in Hospital in Brownlow Street. Ironically, it was at the latter institution that the first recorded epidemic of puerperal fever took

[79] See John Woodward, *To Do the Sick No Harm: A Study of the British Voluntary Hospital System to 1875* (Boston, Massachusetts: Routledge and Kegan Paul, 1974; reprint, 1978), p. 45. See also E. M. Sigsworth, "Gateways to Death? Medicine, Hospitals, and Mortality, 1700–1850," in *Science and Society, 1600–1900,* ed. Peter Mathias (Cambridge: Cambridge University Press, 1972), pp. 97–110; Guenter B. Risse, *Hospital Life in Enlightenment Scotland: Care and Teaching at the Royal Infirmary of Edinburgh* (Cambridge: Cambridge University Press, 1986), esp. pp. 287–91; and S. G. Cherry, "The hospitals and population growth: Part I, the voluntary general hospitals, mortality, and local populations in the English provinces in the eighteenth and nineteenth centuries," *Pop. Stud.,* 1980, *34:* 59–75.

place, in 1760. This seems to be one epidemic in which it is possible that the infection was introduced by an obstetrician who performed autopsies, although the epidemic was by no means confined to that hospital.[80] The experience of that terrible epidemic seems to have left a lasting impression on Hunter. One author has attributed to it Hunter's extreme conservatism in obstetrical management. We know that when he established his Great Windmill Street School in 1767 he did so on separate premises from his midwifery practice and that when he was attending Queen Charlotte he even discontinued his anatomical lectures entirely. George C. Peachey has suggested that a fear of communicating fever led Hunter to take these actions. Moreover, although he attended the birth of Charlotte's child, the actual delivery was performed by a midwife.[81]

Hunter was one of a group of four men who had been friends from their student days in Scotland, when Hunter studied with William Cullen and Cullen became a close friend of John Fothergill's at Edinburgh. Cullen went to Glasgow and then returned to Edinburgh, where he trained hundreds of physicians, while Fothergill and Hunter moved to London. In London, Hunter studied midwifery with Cullen's friend and former neighbor William Smellie, who in turn became a friend of Fothergill's.

These men became leading members of a circle of Scottish physicians, foreigners, obstetricians, and Dissenters who had not received degrees from Oxford or Cambridge and were not acceptable to the Royal College of Physicians because of their specialty, their nationality, or their religion. As a group, they held many distinctive views on the nature of fevers and particularly on the possibility of transmission through contagion. As they increasingly dominated the obstetrical profession, their views helped shape hospital practices in general, to the benefit of maternity patients.[82]

[80] Both Hunter and his associate Francis Sandys were distinguished anatomists. Sandys died in 1771 and Hunter retired in 1782. Following Hunter's departure, the average mortality rate at the British Lying-in dropped considerably. On Sandys, see Zachary Cope, *William Cheselden, 1688–1752* (London: E. and S. Livingstone, 1953), and John Glaister, *Dr. William Smellie and His Contemporaries: A Contribution to the History of Midwifery in the Eighteenth Century* (Glasgow: Maclehose, 1894). On Hunter, see W. F. Bynum and Roy Porter, eds., *William Hunter and the Eighteenth-Century Medical World* (Cambridge: Cambridge University Press, 1982), esp. chaps. 1 and 2, which contain extensive further citations; George C. Peachey, "William Hunter's obstetrical career," *Ann. Med. Hist.,* 1930, n.s. *2:* 476–79; *idem, A Memoir of John and William Hunter* (Plymouth, England: Brendon, 1924); John Kobler, *The Reluctant Surgeon: The Life of John Hunter* (London: Heinemann, 1960); and Judith Schneid Lewis, *In the Family Way: Childbearing in the British Aristocracy, 1760–1860* (New Brunswick, New Jersey: Rutgers University Press, 1986).

[81] Peachey, "William Hunter's obstetrical career," pp. 476–77, 478–79.

[82] In 1744, Fothergill became the first Englishman with an Edinburgh M.D. to be permitted to apply for a license to practice medicine in London from the Royal College of Physicians. (The first Scottish graduate of Edinburgh licensed was William Schaw, in 1752.) Fothergill was at the center of the unsuccessful effort by the Licentiates to obtain the privileges of Fellowship from the College. The College refused to admit the holders of Edinburgh degrees to Fellowships, which were by law reserved for the graduates of Oxford, Cambridge, and Trinity College, Dublin. Since Dissenters were excluded from Oxford and Cambridge, this boycott of Scottish degree holders effectively excluded most Dissenters from Fellowship and, until the middle of the century, from the right to practice as a physician in London at all. Obstetricians were also refused Fellowships. The prejudice against Scots, and the hardships experienced by those excluded by the medical establishment, welded the newcomers into a close community with ties to Edinburgh and other Dissenting provincial medical communities. Nearly every distinguished physician in London who was not a Fellow of the Royal College

Fothergill's emphasis on the contagious nature of some diseases, particularly streptococcal pharyngitis, has already been noted, although there is no evidence that he connected pharyngitis with puerperal fever. Cullen also emphasized the importance of contagion in many fevers and the value of conservative therapy.[83] Fothergill, his disciple John Coakley Lettsom, and his friend John Howard were constant campaigners for improved institutional hygiene: Lettsom, who was also a pupil of Cullen's, is known to have emphasized the importance of personal as well as institutional cleanliness. Lettsom and Nathaniel Hulme served as staff physicians to the City of London Lying-in Hospital, which had an admirable mortality rate of five per thousand during Lettsom's tenure at the end of the century.[84] (See table 1.)

Hunter and Smellie and their pupils trained a very large percentage of all English obstetricians, as well as many midwives. Smellie, for example, taught more than nine hundred men in the 1740s alone, along with an unknown number of women. They or their associates also served on the professional staffs of most of the London lying-in institutions. At the General Dispensary for Poor Married Women, for example, William Black and Gilbert Blane were both pupils of Cullen's, and Blane was a protégé of William Hunter's, who obtained for him his first naval appointment. The Irish-born Black was a friend of Lettsom's.[85]

At the Lying-in Charity, which delivered about five thousand patients in their homes in 1772 (more than a quarter of all the deliveries in London), twenty-seven midwives were overseen by the physician John Ford and his assistants, one of whom was Thomas Cogan.[86] Not much is known about Ford, except that he had an M.D. from St. Andrew's and was a Methodist lay preacher. Cogan, however, was not only a former Unitarian minister but also a friend of Lettsom's, with whom he founded the Royal Humane Society. He was an early member of Lettsom's Medical Society of London. In 1780, Cogan was replaced by John Sims (M.D. Edinburgh), a Quaker and

of Physicians was associated with one or more of these four men. Most had received a distinctive medical education at Edinburgh, which became even more distinctive as men like Cullen and Hamilton assumed professorships there.

[83] On Cullen's influence, see W. F. Bynum, "William Cullen and the Study of Fevers in Britain, 1760–1820," in *Theories of Fever from Antiquity to the Enlightenment,* ed. W. F. Bynum and Vivian Nutton (*Med. Hist.,* suppl. no. 1, 1981), pp. 135–47, and Guenter B. Risse, "'Doctor William Cullen, physician, Edinburgh': a consultation practice in the eighteenth century," *Bull. Hist. Med.,* 1974, *48:* 338–51.

[84] J. Johnston Abraham, *Lettsom: His Life, Times, Friends, and Descendants* (London: William Heinemann, 1933), p. 246. I would like to thank the NLM for supplying me with microfilm copies of Lettsom's works. On Howard, see Leona Baumgartner, "John Howard and the public health movement," *Bull. Hist. Med.,* 1937, *5:* 489–508, and John E. Ransom, "John Howard on communicable diseases," *Bull. Hist. Med.,* 1937, *5:* 131–47.

[85] Herbert R. Spencer, *A History of British Midwifery, 1650–1800* (London: John Bale, Sons, and Danielsson, 1927); William Munk, *The Roll of the Royal College of Physicians of London,* 2d ed., rev. enl., vol. 2 of 7 (London: Longman, Green, Longman, and Roberts, 1861); W. B. Howell, "Dr. George Fordyce and his times" *Ann. Med. Hist.,* 1930, n.s. *2:* 281–96, William Black, *A State of facts, relative to William Black* ([Edinburgh,] 1770).

[86] Stanley A. Seligman, "The Royal Maternity Charity: the first hundred years," *Med. Hist.,* 1980, *24:* 403–18.

also a close friend of Lettsom's. Sims served as president of the Medical Society in 1783.[87]

Dissenters such as the Quakers Fothergill and Lettsom, and Scots like Alexander Gordon or the professors of midwifery at Edinburgh Thomas Young (professor from 1756 to 1780) and Alexander Hamilton (professor from 1780 to 1800), differed from their English and Anglican colleagues in that they placed less emphasis on the importance of the climate—what Sydenham had called "the epidemic constitution of the atmosphere"—and more emphasis on the possibility of specific local contagions, which were spread by contact or fomites and could be transmitted only over a short distance by air.[88] Many English physicians, such as John Leake, William Butter, and John Clarke, believed that meteorological conditions were responsible for epidemic disease. There is considerable justification for their views, since streptococcal infections do indeed have a distinctive climatic and meteorological incidence. Miasmatic doctors were generally less alert to the importance of isolating surgical cases and patients with contagious diseases, and they sometimes believed that crowding was acceptable as long as there was adequate cross-ventilation within wards.[89]

For example, the founder of the first English maternity hospital was Sir Richard Manningham, the Cambridge-educated son of a bishop. Manningham was a dedicated anti-contagionist. In 1744 he wrote a book entitled *The Plague no Contagious Disease* and in 1758 reissued it as *A Discourse Concerning the Plague and Pestilential Fevers: plainly proving, that the general productive causes of all plagues of pestilence, are from some fault in the air: or from ill and unwholesome diet: and that the air is the principal cause of spreading the infection.* This book argued that any attempt to enact a general quarantine to prevent the introduction of the plague would do more harm than good by disrupting trade and raising the price of food to the poor. There was abundant evidence, Manningham argued, that the plague was not transmitted by contact or by tainted goods: "There is not any Corruption carried from the Body of the Sick, into the Body of the

[87] Abraham, *Lettsom,* pp. 140–41. John Sims should not be confused with James Sims, who practiced in County Tyrone before settling in London.

[88] Joseph Clarke was an Edinburgh graduate and the nephew of Fothergill's close friend George Cleghorn as well as a friend of Charles White's. I have not been able to determine whether Clarke and Cleghorn were also Quakers. For Gordon, Young, and Hamilton, see FC, p. 38, and Spencer, *History of British Midwifery,* pp. 90–97. Francis Home, professor of materia medica at Edinburgh, also adopted a contagionist view of puerperal fever; see John J. Byrne, "Dr. Francis Home and puerperal sepsis," *New Eng. J. Med.,* 1954, *251:* 440–42. Hunter was both a Scot and a putative lapsed Unitarian, but his personal views on disease transmission, like his personal religion, are difficult to determine. In the absence of evidence to the contrary, I have assumed that John Clarke of London, who attended St. Paul's School before studying obstetrics in London, and John Leake (the son of a Glasgow curate who settled in Cumberland), who attended Bishop Auckland Grammar School and obtained his M.D. from Rheims, were Anglicans. Obstetricians and surgeons are more difficult to classify by religion than physicians since, unlike most physicians, they did not find it advantageous to attend Oxford or Cambridge.

[89] Margaret DeLacy, "Social medicine and social institutions in eighteenth-century Lancashire" (unpublished).

Sound."[90] Manningham did not explicitly apply his views to his own field of obstetrics, perhaps because epidemic puerperal fever had not yet appeared in England, but he did argue that all fevers were products of the same underlying cause, a "lentor" of the blood, and that plague was merely the most extreme result.

The English-educated John Leake believed that the original cause of puerperal fever was a "distemperature of the air" combined with a mechanical change produced in the body by delivery, although puerperal fever might at last become infectious like dysentery or ulcerous sore throat. Along with early and copious bleeding, therefore, Leake also recommended cleanliness, ventilation, and the disinfection of the air by the burning of brimstone.[91] In 1793 John Clarke argued that "plethoric" women were more susceptible to fevers and recommended that they avoid animal food, breathe pure air, and remain horizontal for some days after delivery, but he attributed epidemic puerperal fever to a constitution of the air which resulted from a long succession of seasons whose "peculiar properties ... are infinitely too subtle for our investigation."[92]

An exception must be made for the Anglican Charles White, who demanded not only perfect cleanliness but also the complete separation of parturient women from each other. White was convinced that crowded hospitals—indeed, all hospitals—were dangerous. Although he was an Anglican, White had been trained by William Hunter and was a member of a group of hospital reformers centered in Warrington and Manchester.[93] Fothergill, who came from Warrington, spent his summers nearby. White advised Joseph Clarke on the administration of the Rotunda; perhaps it was because of his advice that the wards were small. White had strongly recommended that each patient have a separate room if possible. In fact, the Rotunda had four divisions, each with one room holding seven beds and two rooms with two beds each. A separate nurse and maid was assigned to each division.

[90] Richard Manningham, *A Discourse Concerning the Plague* ... (London: J. Robinson, 1758), p. 18. Spencer, *History of British Midwifery*, pp. 14–18.

[91] Leake, *Practical Observations*, FC, p. 126.

[92] John Clarke, *Practical Essays*, FC, p. 416.

[93] On this group, see Thomas Percival, *Essays, Medical and Experimental* (London: Lowndes, 1770); *idem, The Works, Literary, Moral and Medical of Thomas Percival ... to which are Prefixed Memoirs of his Life and Writings*, ed. Edward Percival, 4 vols. (London: Joseph Johnson, 1807); John Aikin, *Thoughts on Hospitals* (London: Joseph Johnson, 1771); *idem, A View of the Life, Travels, and Philanthropic Labors of the Late John Howard* (Philadelphia, Pennsylvania: John Ormrod, 1794). Aikin supplied Howard with the questions about the contagiousness of the plague which he used in collecting information for his work on Lazarettos. See also Michael W. Flinn, introduction to Edwin Chadwick's *Report on the Sanitary Condition of the Labouring Population of Great Britain, 1842* (Edinburgh: Edinburgh University Press, 1965); E. P. Hennock, "Urban sanitary reform a generation before Chadwick," *Econ. Hist. Rev.*, 1957, *10:* 113–20; B. Keith Lucas, "Some influences affecting the development of sanitary legislation in England," *Econ. Hist. Rev.*, 1953–54, *6:* 290–96; Edward Mansfield Brockbank, *Sketches of the Lives and Work of the Honorary Medical Staff of the Manchester Infirmary ... 1752 to 1830* ... (Manchester: Manchester University Press, 1904); and Lucy Aikin, *Memoir of John Aikin, M.D., with a selection of his Miscellaneous Pieces* (Philadelphia, Pennsylvania: Abraham Small, 1824). I would like to thank Dickinson College, Carlisle, Pennsylvania, for supplying a copy of Lucy Aikin's work.

Both groups agreed that thorough ventilation of hospital wards was essential to prevent cross-infection: Leake, in fact, was said to refuse to remain in a room that was not supplied with fresh air. The "nonconformist" group, because it was more willing to entertain the possibility of contagion, placed a greater emphasis on the importance of separating patients and maintaining institutional and personal cleanliness. If diseases could be transmitted only in a confined area, it was much easier to interrupt their progress through cleansing than if they were due to the atmosphere of an entire city, or to a mechanical cause, like the weight of the uterus. Contagionists were more alert to the possibility of transmission from person to person by a third party, to the need for clean hands and clothing, and to the danger of crowding. Thus, Lettsom argued:

> Matters ... are constantly arising ... from all breathing animals, and putrid animal bodies, which, under certain circumstances, are capable of producing the [putrid] fevers.... These diffusive active matters, appear in some stages of these fevers, to act as ferments on the fluids, and thereby to multiply themselves, and communicate contagion amongst a number of men.... [Contagion] does not in general extend to any considerable distance, or rise to any great height.... It is apt to remain in a concentrated state on the surface of the body retaining it, and on the garments and substances which have been in vicinity to the diseased, in the same manner as odors adhere to bodies in general.[94]

The Irish-born William Black, of the General Dispensary for Poor Married Women, differentiated between inflammatory and putrid fevers. The former were both infrequent and mild. The latter, which were common and severe, were not spread by climate, season, or any sensible qualities of the atmosphere, but could be communicated by

> imperceptible emanation or contagion from one infected person to another, by personal intercourse, by the medium of polluted goods, furniture, apparel, cloaths, and houses; in all which the noxious miasma may be concentrated and lodged.... Of what elementary nature miasma and contagion consist; the analysis of their minute atoms, whether animalcules, or to us invisible emanations, I pretend not to decide.... Neither marshy miasma, nor those from human effluvia, spread to any considerable distance through the air.[95]

The predisposing causes of such fevers included crowding, filth, rotting food or animal carcasses, "putrid sores and mortifications, gangrenous inoculation through wounds of the skin," and many other factors. Black, how-

[94] John Coakley Lettsom, *Reflections on the General Treatment and Cure of Fevers* (London: Printed for the author by J. D. Cornish, 1772), pp. 8–15. Like most of his contemporaries, Lettsom accepted the division of fevers into intermittent (primarily caused by marsh air), putrid, and inflammatory. Inflammatory fevers were not contagious, but the "nonconformist" doctors argued that they were also infrequent by comparison with the putrid fevers. One of the fiercest debates of the century was over the question of whether puerperal fever was putrid or inflammatory. This had implications for therapy, since the common treatment for inflammatory fevers was bleeding, while the common treatment for putrid fevers was cinchona and cordials.

[95] Black, *Arithmetical and Medical Analysis*, pp. 51 and 69.

ever, like Leake, believed puerperal fever was due to inflammation and became epidemic through "some unknown quality of the atmosphere and seasons."[96] Black's Scottish colleague Gilbert Blane, better known as a naval surgeon, is described by Margaret Pelling as a leading contagionist of the early nineteenth century.[97] The Quaker physician Anthony Fothergill wrote an article on puerperal fever in 1783 which assumed the fever was highly contagious.[98]

Thomas Young, professor of midwifery at Edinburgh, believed that puerperal fever arose from a local infection. His successor, Alexander Hamilton, commented that an outbreak of puerperal fever at Edinburgh Infirmary was due to a "specific contagion from the air of the wards [which] in surgical wards ... sometimes ... produces almost in every wound, even the slightest, symptoms of erysipelas and even mortification."[99] Hamilton saw puerperal fever as primarily an airborne contagion, but he believed it was so contagious it could be carried by a third person. At the Rotunda, Joseph Clarke attributed puerperal fever to a local infection. He followed the advice of Young, "the only writer who has recommended measures similar to what we pursued," and carried out a ruthless cleaning program, removing patients, whitewashing and painting the wards, and replacing the bedding.[100] When, at the end of the century, Alexander Gordon made a point of refusing to discuss the atmosphere, and instead demonstrated that attendants carried contagion from house to house, his work represented the logical culmination, although in more definitive and conclusive terms, of the work of many previous British writers.

According to Ignaz Semmelweis, the chief cause of the soaring mortality rates in nineteenth-century hospitals was the teaching of pathological anatomy and the large numbers of medical students coming into the maternity wards straight from the dissecting rooms with "cadaveric matter" on their hands. Autopsies were not performed as frequently in eighteenth-century hospitals, but they did take place. Many of the cases of puerperal fever reported by physicians included autopsy reports. Autopsies could be a source of some danger to the physicians themselves, who sometimes contracted septicemia. Lettsom died from such an infection. It seems likely, however, that the communication of puerperal fever to patients from this source occurred much less frequently in the eighteenth century than in the nineteenth. Few medical students came straight from the morgue to the ward, and medical students were often banned entirely from lying-in wards. For example, both the British Lying-in and the Middlesex Hospital barred

[96] *Ibid.*, p. 217.

[97] Margaret Pelling, *Cholera, Fever, and English Medicine, 1825–1865* (London: Oxford University Press, 1978), p. 27.

[98] Anthony Fothergill, "An account of an improved method of treating the puerperal fever," *London Med. J.*, 1782, *3:* 411–18. Anthony was a distant cousin of John Fothergill's.

[99] Spencer, *History of British Midwifery*, p. 97.

[100] Joseph Clarke, *Observations*, FC, pp. 360 and 357.

male students.[101] In at least one case, the physician who usually performed the autopsy did not deliver patients.[102] Pathological anatomy had not yet become fashionable.

Treatises of the time say a great deal about keeping the patients and their surroundings clean but little about personal cleanliness for attendants. Nevertheless, there is some evidence that obstetricians cleansed themselves and their instruments. Smellie was criticized by a leading London midwife for delivering in a "nightgown," a costume he recommended not only because it offered a way to conceal his forceps but also because it was easy to wash.[103] As for the forceps themselves, Smellie was again roundly criticized by John Burton, a York obstetrician, for his first design, which had the blades wrapped in leather. Burton pointed out that this made it impossible to clean the blades completely. Smellie rejoined that the leather was supposed to be removed and replaced after each use, but he soon substituted more easily cleaned metal blades.[104] The contagionist belief that the source of disease tended to adhere "in a concentrated state on the surface of the body" probably encouraged physicians to wash their hands and change their clothing.[105]

We also know that British hospitals, unlike Continental ones, preferred to keep maternity patients in small wards. For example, the Edinburgh General Lying-in Hospital had seven-bed wards for ordinary patients. A woman was admitted to one ward, was carried to the delivery room, and then, still in the same bed, was carried to a separate lying-in ward. There were also six private bedrooms for women "in a dangerous situation." When they left, their bedding was removed to a drying house for two weeks.[106] All the maternity hospitals tried to bar verminous or contagious patients and controlled visitors. For example, the Westminster New Lying-in required that patients be clean and free from infection, that patients suckle their babies, and that visitors see patients in the reception hall. No visitors were permit-

[101] *Laws, Orders, and Regulations of the British Lying-in Hospital* (London: E. Cox, 1781) and *An Account of the Middlesex-Hospital* (c. 1752).

[102] Hulme, *Treatise*, FC, p. 62.

[103] Spencer, *History of British Midwifery*, p. 148.

[104] Glaister, *Dr. William Smellie.* See also Gordon, *Treatise:* "The nurses and the physicians who have attended patients affected with the puerperal fever ought carefully to wash themselves and to get their apparel properly fumigated before it be put on again" (FC, p. 485).

[105] See, e.g., Louis H. Roddis, *James Lind, Founder of Nautical Medicine* (New York: Henry Schuman, 1950) pp. 105–6: "Scrupulous cleanliness, not only to the compartment but of linen and bedding, and the disinfection of the urine, sputum, or other discharges ... was part of his regular routine. He recommended that the surgeon wear special clothes when on duty and was in favor of waxed linen as being least likely to become soiled. A modern touch was in urging the frequent washing of hands on the part of the surgeon.... Even more important was his injunction against the use of sponges. Lind recommended in place of sponges the use of clean linen or cotton cloths that could be thrown away or thoroughly washed.... He had his nurses and attendants wear painted canvas jackets as these could easily be washed." Lind, a Scot, was educated at Edinburgh.

[106] James Hamilton, *Select Cases in Midwifery extracted from the Records of the Edinburgh General Lying-in Hospital with Remarks* (Edinburgh: G. Mudie and Son; London: J. Johnson, 1795).

ted within one week of delivery and after that were allowed only with written permission.[107]

In the early nineteenth century, contagionist views and the efforts they encouraged again fell out of fashion. They had never been generally accepted on the Continent. English visitors frequently described their disgust at the squalor and crowding they found in Paris. "That the Parisian malady was more highly malignant . . . will not appear wonderful to anyone who has ever visited that crowded receptacle of disease and contagion, the Hôtel-Dieu," commented Anthony Fothergill in 1782.[108]

As J. George Adami wrote in 1923 of British obstetricians in the late eighteenth and early nineteenth centuries, they

> first gained control over puerperal fever. They it was who introduced free ventilation and . . . cleanliness . . . , who laid stress upon disinfection, who . . . recognised the worth of chlorine . . . , who introduced the disinfection of the hands and drainage of the puerperal wound, who would have no truck with the . . . atmospheric, cosmic, or telluric theory—but held to the contagious. . . .
>
> By the early [1840s] other influences were at work. . . . Charles White's . . . methods had lapsed, even in Manchester itself, so that . . . mortality . . . became a matter of the gravest concern. This . . . [was due] to the diffusion of foreign, and particularly French teaching as to the nature of puerperal fever. . . . It was [this] . . . which, in 1826, on the ground that his procedure was old-fashioned and based on "contagionism," had at Vienna forced the resignation of Professor Boër, and had led to the disastrous introduction of Professor Klein and his "anatomical" teaching into the Allegemeines Krankenhaus.[109]

Semmelweis followed Klein. When Semmelweis demonstrated that maternal mortality rates had fallen since he had introduced chlorine handwashing, his opponents argued that the improvement was due instead to the introduction of a new system of ventilation into the hospital.

Because they believed that cross-infection could be avoided if wards were adequately ventilated, miasmatists were willing to relax the rules that had barred patients with contagious diseases from eighteenth-century hospitals. Changes in policy permitted hospitals to admit a much wider spectrum of patients. It was this, and not merely the growth in urban population, that created the appalling crowding of hospitals in the nineteenth century —which in turn contributed to appalling mortality rates. One of the most dedicated of English miasmatists was Florence Nightingale, who believed that the greatest requirement for hospital design was the promotion of free ventilation. This, in turn, was favored by the creation of large wards in place of small, separate rooms. She had hoped to establish a special program for nurse midwives but was forced to discontinue the school by an unacceptably high incidence of puerperal fever in the training wards. Her experience eventually persuaded her that maternity patients should be placed in separate rooms.[110]

[107] *Laws, Rules, and Orders of the Westminster New Lying-in Hospital* (London, 1793).
[108] Fothergill, *Account,* p. 415.
[109] Adami, *Charles White,* pp. 30–31, 41, and 50–51.

The shift toward miasmatism may help to explain why hospital mortality rates, which had fallen to very low levels by the end of the eighteenth century, rose steeply again in the early nineteenth century. The greater resistance to contagionism on the Continent may also explain why Continental hospitals were far more dangerous than British ones. In Paris, for example, puerperal fever became common in the 1770s and grew steadily in incidence thereafter, creating death rates unknown in Britain. Those few historians who have discovered that nineteenth-century hospital mortality rates were not better than those of the eighteenth—but far worse—have attributed the change to increasingly adventurous surgery and to crowding.[111] This nineteenth-century rise requires further investigation to evaluate the possible contributions of changing admissions policies and the steadily increasing virulence of community streptococcal infections.

In conclusion, British maternity hospitals in the eighteenth century may have magnified epidemics, but they probably did not generate them. Moreover, their record showed continual improvement during the second half of the century. By the end of the century a pregnant woman was as safe in a hospital as she was at home, and as safe as she would be at any time before the second half of this century. This comparatively low mortality was due to a combination of factors, including improved obstetrical care and a distinctive medical tradition.

APPENDIX: TABLE SOURCES

The sources for the tables herein are as follows. Figures for the British Lying-in (tables 1 and 3) are from "Account of the Women Delivered, . . . 1749 to . . . 1801," in Heberden, *Observations,* pp. 39–41. This gives figures for the number of women delivered and the number of children born including stillbirths and neonatal deaths. I have calculated the rates using the number born. Because of twin births this is slightly greater than the number of women delivered. Using the number of women delivered would raise the overall annual average by about 0.2 deaths per thousand. Buer, *Health, Wealth, and Population,* supplied the rate for 1800–1807 in table 1. Figures for the City of London Lying-in (table 1) are from William Gilliatt, "Maternal Mortality," in Kerr, Johnstone, and Phillips, *Historical Review,* p. 261, for 1790–1810, and from *The One Hundred Thirty-Eighth Report of the City of London Lying-In Hospital . . .* (London, 1889) for 1830–1848. Figures for the Rotunda (tables 1 and 4)

[110] Cecil Woodham-Smith, *Florence Nightingale, 1820–1910* (New York: McGraw-Hill, 1951), pp. 304–5. See also Sydney Selwyn, "Sir James Simpson and hospital cross-infection," *Med. Hist.,* 1965, *9:* 241–48.
[111] See, e.g., Woodward, *To Do the Sick No Harm,* p. 122, and Cherry, "Hospitals and Population Growth." See also Gilliatt, "Maternal Mortality," in Kerr, Johnstone, and Phillips, *Historical Review,* p. 262, and Buer, *Health, Wealth, and Population,* pp. 145–48.

are from Joseph Clarke, "Observations on some causes of the excess of the mortality of males above that of females," *London Med. J.,* 1788, *9:* 179–200, for 1758–1784, and Semmelweis, *Etiology, Concept, and Prophylaxis of Childbed Fever,* ed. Carter, pp. 142–43, for 1784–1848. Whereas Clarke specifically states that these figures are rates for the number of women actually delivered at the Rotunda (women who left before giving birth were subtracted), Semmelweis gives his Rotunda rates as percentages of "births." Since Semmelweis gives the number of "births" as 1,261 in 1784, however, and Clarke gives 1,260 as the number of "women delivered" for the same year, I believe Semmelweis's figures are actually for women delivered. Because of the incidence of twin births, Semmelweis's figure should have been higher if it was for all children born. Figures for the Vienna Maternity wards (table 1) are from Semmelweis, *Etiology,* pp. 142–43. I do not know whether this adjusts for twins. Figures for the city of London (tables 1 and 2) are my calculations from Heberden, *Observations,* for 1750–1799, and James Young, "Journals, 1800–1950," in Kerr, Johnstone, and Phillips, *Historical Review,* p. 325, for 1800–1814. In many instances I have converted the data to deaths per thousand from some other method of expressing the rate. I have also supplied five-year averages.