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Puerperal Fever in Britain: Failed Models of Disease Causation

by

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A thesis submitted in partial fulfillment
of the requirements for the degree of
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Table of Contents

Abstract	ii
Introduction	1
Historiography	5
Etiology, Symptomology, Statistics	14
Early Accounts	20
Humoural Theory: Hippocrates and Galen	24
Miasma Theory: The Persistent Dead-End	27
Medical Education: The Resilience of a Dogma	31
Mechanism: A New Metaphor	33
Inflammation Theory: The Bleeding Continues	40
Pathology: New Techniques with Old Results	43
Contagionism and the Environment: Definitions and Confusion	47
Success: Treatments Break Free From Galen	59
Conclusion	62
References Cited	65

Abstract

In eighteenth- and nineteenth-century Britain, a bacterial infection which we now know to be caused primarily by a streptococcus, was killing women in childbirth at an alarming rate. The disease, called puerperal, or childbed, fever, was being transmitted primarily from doctor to patient by a doctor's unwashed hands and filthy, contaminated clothing and linens. Despite this evident and, in retrospect, obvious vector, the doctors of this period never discovered how to prevent their patients from dying a gruesome and painful death. Many physicians wrote extensive accounts of the illness but often ended their works in despair, unable to find the cause. Much of the historical literature blames this befuddlement on personality traits of the physicians, arguing that egos and professional hostilities prevented the kind of cooperation that could have led to progress.

This study attempts to show that this failure was not a product of personalities but of the modern physicians' assumptions and logic. The assumptions were the still-powerful, but often unnoticed, dictates about the human body handed down from ancient Greek medicine. The logical errors were a product of pre-scientific notions of definition, explanation, and evidence. The author argues that it was not a lack of data that thwarted the physicians, but a series of these intellectual roadblocks that prevented them from understanding and extended the terror of puerperal fever for another two centuries.

Introduction

Puerperal fever was the leading cause of maternal death prior to the twentieth century. It was a disease that, in the late eighteenth and early nineteenth centuries, defied modern medical understanding. The various theories of disease causation that made up the classical medical curriculum struggled unsuccessfully to explain a host of frustrating inconsistencies surrounding the disease. Puerperal fever was unpredictable in location, timing, and severity. It struck women of all classes and constitutions. It was frightening in tragic isolated cases, but in epidemic form it was a blind biological terror causing the deaths of countless mothers and babies in lying-in hospitals and homes all over the world.

The story of puerperal fever in the eighteenth and nineteenth centuries is one of failed logic. This is not a mystery of missing data: data that, had it been discovered, would have prevented or cured puerperal fever. In fact, the doctors had more than enough data, which they freely shared, to recognize that puerperal fever passed from doctor to patient and that basic sanitary procedures would have prevented the disease. The question at hand is why, in the face of clear, repeated associations of physician contact and puerperal fever, the medical establishment failed to incorporate this data into their model of disease causation. The answer is that a combination of ancient assumptions and pre-scientific logical deficiencies prevented them from making the conceptual breakthrough that would have prevented the disease.

Also known as childbed fever, puerperal fever is a post-partum infection that strikes lying-in women during or within a few days after childbirth. It can also be contracted during a miscarriage or abortion. Prior to the advent of antibiotic treatments in the mid-twentieth century, puerperal fever was a deadly complication of a significant percentage of births. In Great Britain it accounted for approximately half of all deaths related to childbirth.¹ It killed more women than cancer and ranked second only to tuberculosis in deaths of women of childbearing age.²

The emerging obstetric profession's attempts to apply theories of classical medicine, using concepts from Hippocrates and Galen, proved unsuccessful in either prevention or cure of the disease. There were scattered attempts to isolate causal factors: an early example of the scientific method making its way into medicine. These attempts to construct correlations that might have led to a new understanding of causation were handicapped by ambiguity in the meanings of key concepts and hidden assumptions about how the biological world worked. The inability to break free of long-standing assumptions led the medical community in circles and fostered intense debate over every aspect of this confusing disease. Because of this, progress was sporadic at best, even after the development of germ theory in the last half of the nineteenth century.

¹ Robert Thomas, *The Modern Practice of Physic* (London: Longman, Rees, Orme, Brown, and Green, 1828), 932. Charles Delucena Meigs, *Obstetrics: The Science and the Art* (Philadelphia: Blanchard and Lea, 1852), states on page 614, "...it is well known that child-bed fever destroys more women than all the other diseases and accidents of parturition put together. No physician can long practice the art of Midwifery without discovering that a constant and wise vigilance is necessary to obviate the causes of such attack, and cure the patient who has been unhappily seized with it. There is scarcely any form of dangerous disorder that is more insidious in its approach, or more rapid in its development when once its terrific train is set in motion; a development so rapid that the loss of a few hours, at the commencement, renders all after interposition fruitless and unavailing. Not a few of the victims are known to perish within twelve hours, and some even within six hours after the first manifestation of the symptoms."

² Irvine Loudon, *Death in Childbirth: An International Study of Maternal Care and Maternal Mortality, 1800-1950* (Oxford: Oxford University Press, 1992), 163.

Compounding these difficulties, puerperal fever was an especially perplexing disease. Compared to other contagious diseases, which doctors enjoyed modest success in explaining and treating during this period, puerperal fever remained a puzzle. Further, the developing focus on pathology in medicine, which might be expected to herald new insights, only compounded confusion about puerperal fever.

Most historical analyses have attributed these failures to personal shortcomings of the physicians involved, their turf battles, antagonistic personal relations, and a stubborn resistance to the few visionary heroes that could have led the way had the medical profession only been willing to listen. This is, at best, a simplistic and largely misleading interpretation of the issues of intellectual history at work during this period.

The history of puerperal fever is not a story of heroes and villains, of people who made glorious breakthroughs only to be shunned by ego-blinded colleagues. It is rather an account of physicians and midwives, dedicated but perplexed, daily confronting a predictably terrible disease against which they had little hope of triumph. Nor is it a story of a flash of insight, or a momentous discovery, that won the day. The brave people who tried to understand this scourge were intellectually disarmed. They were fighting an intractable foe with outdated conceptual tools, medical teachings that had varied little over the previous millennia.

Instead of petty and transient issues, this period was marked by dramatic disputes involving both substantial advances and persistent errors, most involving the hidden assumptions of a classical medical education and the worldview it promulgated. These assumptions were bolstered by the practice, employed by many physicians, of responding to any possible progress by crafting *ad hoc* addendums to their theories to show that,

contrary to first appearances, this latest discovery could be made to fit nicely into their established views.

Finally, pervasive confusion and ambiguities involving the definitions of key concepts related to the disease fragmented the modest advances that were achieved. This fragmentation, presented when advances in understanding transmission, sanitation, and even germ theory should have heralded major progress, is the sad story of the largely unsuccessful fight against puerperal fever.

Historiography

Failure is the unfortunate icon of the history of puerperal fever: failure in communication, failure in data collections, and failure of treatments. But, in retrospect, there were also substantial failures of logic. Moving from the ancients with their animated and spirit-ridden view of the world into the mechanistic, observable, verifiable analyses of the scientific revolution, we can see that the fundamental impediment to the triumph over this disease has consistently been an intellectual one. We find that all parties involved had roughly the same data. The task which commands the attention of historians has been to comprehend the inability of physicians to produce breakthroughs, either in prevention or treatment, that saved lives.

A majority of medical histories can be classified into two opposing camps. The progressive, Whig interpretations depict the “march of progress” in medicine by glorifying certain medical advances and the “heroes” associated with discovering them. These histories focus on the “Great Men” of medicine, doctors and researchers who took advantage of the steps laid before them by their equally heroic predecessors to climb the ladder of success and add their own contributions to medicine for the betterment of mankind. They chronicle the greatest inventions and innovations of medicine as a single trajectory of medical discovery on an inevitable march forward.³

³ Amanda Carson Banks, *Birth Chairs, Midwives, and Medicine* (Jackson, MS: University Press of Mississippi, 1999), xvi ; Mary Lindemann, *Medicine and Society in Early Modern Europe* (Cambridge, UK: Cambridge University Press, 1999), 1-2 ; Irvine Loudon, “Medical Practitioners 1750-1850 and the Period of Medical Reform in Britain,” in *Medicine in Society: Historical Essays*, ed. Andrew Wear

Revisionist histories claim that, rather than an intellectual march of progress, medicine has been marked by the tragic inability of doctors to do anything to alleviate human suffering, offering only treatments that more often added to a patient's pain. Revisionist histories claiming to debunk the Whig assumption of progress first became popular after the 1963 publication in French of Michel Foucault's *The Birth of the Clinic*, the first genuine critique of the role of medicine in society.⁴ Foucault broke with the traditional way of viewing the history of medicine by describing the origin of the clinic as a spontaneous creation distinct from the medical knowledge that preceded it. Instead, he described the medical knowledge of the clinic as being a re-presentation of knowledge and power within that particular society at that particular time. Illness became an identifier that lumped individuals into a category that needed to be monitored and treated in a way that reinforced norms of societal behavior and existing medical knowledge.

However revisionist histories of medicine are often guilty of similar personalization. Instead of searching the past for individuals that create links between concepts, forming a sequential timeline of discovery, revisionists often search for their own "heroes" who made amazing discoveries but were ignored by the medical establishment. In this way, revisionist histories often use their "Great Men" to explain medicine's often halting, unsure progress in much the same fashion as the traditional school celebrates its exciting achievements. Because of this, both versions of the story oversimplify the problems and the players, creating stories with bold characters but little nuance.

(Cambridge, UK: Cambridge University Press, 1992), 220 ; Richard W. Wertz and Dorothy C. Wertz, *Lying-In: A History of Childbirth in America* (New Haven: Yale University Press, 1989), xvii-xviii.

⁴ Michel Foucault, *The Birth of the Clinic: An Archaeology of Medical Perception* (London: Routledge, 1973).

Histories of puerperal fever offer a good example of this same personalization. The majority of these focus on selected heroes such as Ignaz Semmelweis, Alexander Gordon, and Oliver Wendell Holmes, crediting these men with the discovery of the contagiousness of the disease while demonizing known anti-contagionists such as Charles Meigs. A good example of this type of history is David Wootton's *Bad Medicine: Doctors Doing Harm Since Hippocrates* which sets itself apart in its introduction as being diametrically opposed to the Whig interpretation of progress in medicine. Instead, Wootton states, "what we need...is a history, not of progress, but of delay..." and he seeks to do just that, dividing his chapters into specific events wherein a medical non-hero is ignored or derided for, in retrospect, a revolutionary discovery.⁵ In his chapter discussing puerperal fever, Wootton pays homage to the familiar heroes and in doing so, overstates their innovation while quietly explaining away their logical failings.

The best secondary source on puerperal fever is Irvine Loudon's book *The Tragedy of Childbed Fever*.⁶ Although it is not without flaws, it is by far the most comprehensive and useful treatment of the disease. While this book is the least biased source on puerperal fever, it does not present a coherent analysis but is instead a report of relevant information describing the history of puerperal fever from the eighteenth to the twenty-first centuries.

While Loudon offers several minor arguments, they are often tangential to the main goal of his book which is to compile information about a relatively obscure and forgotten disease. The book is full of primary source material and was obviously

⁵ David Wootton, *Bad Medicine: Doctors Doing Harm since Hippocrates* (Oxford: Oxford University Press, 2006), 21.

⁶ Irvine Loudon, *The Tragedy of Childbed Fever* (Oxford: Oxford University Press, 1995).

carefully researched. But the crowning jewels of this book are its statistics. Loudon has compiled a massive amount of statistical evidence with which to support his assertions and they are conveniently catalogued in tables found throughout the book. He uses this data to present several excellent analyses, including his allegation that the adoption of Listerian antisepsis did little to affect the overall statistics of maternal death due to puerperal fever in Great Britain until after World War II.

His background as a medical doctor allows him to explain the biological effects of puerperal fever, its causes, and nomenclature in a clear and concise manner. Besides *The Tragedy of Childbed Fever*, he has also written *Death in Childbirth: An International Study of Maternal Care and Maternal Mortality, 1800-1950* and edited *Childbed Fever: A Documentary History*, both concentrating on puerperal fever, as well as several other books on medical history in the modern period.⁷

However, Loudon is not a historian; he is a general practitioner who turned to writing medical history after retirement. And not all of his assertions are borne out by evidence. He is, at times, guilty of the same personalization found in the Whig medical histories. For example, Loudon's "prematurity" argument centers on the fact that the discoveries of Alexander Gordon of Aberdeen, mainly that puerperal fever could not have been caused by a miasma and that it was related to erysipelas, predated germ theory. He states, "In science, an idea may be rejected 'if its implications cannot be connected by a series of simple logical steps to canonical, or generally accepted, knowledge.'" Unfortunately for Gordon, almost a century had passed before the bacterial basis of

⁷ Loudon, *Death in Childbirth* ; Irvine Loudon, *Childbed Fever: A Documentary History* (London: Garland Publishing, 1995).

puerperal fever was beginning to be accepted.”⁸ However, science has many examples when discoveries have been utilized without the scientific principles behind them being fully understood: electricity, selective breeding, etc. Loudon fails to admit that Gordon not only disregards his own most important achievements, discussing the prevention of puerperal fever in only 300 words, but instead spends the majority of space in his thesis arguing for the inflammatory nature of puerperal fever and bloodletting as the only appropriate treatment.

Christine Hallett has written a comprehensive overview of the eighteenth-century treatises on puerperal fever, and on the development and importance of inflammation and putrid theories.⁹ Rather than offering a Whig or revisionist argument, Hallett presents an excellent documentary analysis of eighteenth- and early nineteenth-century primary sources, describing each work as an example of two opposing theories of puerperal fever causation: inflammation theory and putrid theory. Hallett is currently the director of the UK Centre for the History of Nursing and Midwifery. She holds Ph.D.s in Nursing and in History and has written many articles on the history of nursing in the First World War and in industrial settings.¹⁰

Other excellent sources on puerperal fever are the works of Gail Pat Parsons. She has written a number of articles that truly discuss the differences between contagionist

⁸ Loudon, *The Tragedy of Childbed Fever*, 32-33.

⁹ Christine Hallett, “The Attempt to Understand Puerperal Fever in the Eighteenth and Early Nineteenth Centuries: The Influence of Inflammation Theory,” *Medical History* 49 no. 1 (2005). Inflammation theory was a disease causation model which focused on the buildup of pressure which led to stagnation of the blood while putrid theory characterized a chain reaction of putrescence wherein putrid material taken into the body would then cause healthy tissues to putrefy also. Both of these viewpoints will be more fully explained later in this thesis.

¹⁰ Christine Hallett, “The Personal Writings of First World War Nurses: A Study of the Interplay of Authorial Intention and Scholarly Interpretation,” *Nursing Inquiry* 14 no. 4 (2007): 320-329 ; Christine Hallett, M. Abendstern, and L. Wade, “Industry and Autonomy in Early Occupational Health Nursing: The Welfare Officers of the Lancashire Cotton Mills, 1950-1970,” *Nursing History Review* 14 (2006): 89-109.

and anticontagionist doctors.¹¹ Parsons explains how the disease causation model a doctor chose determined the type of prevention program that he would advocate and contextualizes his arguments for and against describing puerperal fever as a contagious disease.¹² Current historians who fail to comprehend these divisions within the medical community are liable to make false claims concerning the intent of their primary sources. Parsons' work is an example of well-done revisionist history in that she seeks to describe the medical practitioners as they truly were rather than taking a progressive or regressive stance. While she is critical of some secondary source work on puerperal fever, these are primarily revisionist histories that have claimed that the first half of the nineteenth century was characterized by anticontagionism and feminist histories that have suggested that male physicians deliberately allowed their female patients to die, either due to some "enmity" toward women or because "they were simply too 'busy' to wash their hands or change their clothing."¹³

Other secondary source material, both Whig and revisionist, unfortunately tends to idolize or stigmatize various individual doctors, developing theories of why isolated breakthroughs in preventing puerperal fever never influenced the practices of the greater medical establishment. Common heroes are Charles White of Manchester, Alexander

¹¹ Gail Pat Parsons, "The British Medical Profession and Contagion Theory: Puerperal Fever as a Case Study," *Medical History* 22 (1978): 138-150 ; Gail Pat Parsons, "Puerperal Fever, Anticontagionists, and Miasmatic Infection, 1840-1860: Toward a New History of Puerperal Fever in Antebellum America," *Journal of the History of Medicine* 52 (1997). Just as inflammation and putrid theories characterized the eighteenth century debates on causation, contagionist and anticontagionist camps characterized the nineteenth century. The contagionists believed that puerperal fever was caused by a specific contamination which could spread puerperal fever to others. Anticontagionists believed that a non-specific contamination led to an unhealthy state of disease being spread, though not specifically puerperal fever. A thorough description of these opposing theories will be provided later in this thesis. However, it is important to note that these theories were often contingent on the disease being discussed and that doctors could vacillate between the two theories depending on the evidence at hand.

¹² Parsons, "Puerperal Fever, Anticontagionists, and Miasmatic Infection."

¹³ Parsons, "The British Medical Profession and Contagion Theory," 138, 147n43, 148n49.

Gordon of Aberdeen, Oliver Wendell Holmes of Boston, and the Hungarian physician Ignaz Semmelweis.¹⁴ Many of the current claims that are made about the ingenuity of these doctors, by secondary source authors such as Kay Codell Carter, David Wootton, Hal Hellman, and Sherwin B. Nuland, are overstated while the insights of physicians such as Sir Thomas Watson and Robert Storrs of Doncaster are practically ignored in the secondary literature.¹⁵ Watson described puerperal fever as early as 1842 as “instances of direct inoculation”—

Recollect, that the hand of the accoucheur is brought, almost of necessity, into frequent contact with the uterine fluids of the newly-made mother. Recollect, . . . , with what tenacity the smell, which is thus contracted, clings to the fingers, in spite even of repeated washings; and, whilst this odour remains, there must remain also the matter that produces it. Recollect how minute a quantity of an animal poison may be sufficient to corrupt the whole mass of blood, . . . Recollect the raw and abraded state of the parts concerned in parturition; the interior of the uterus forming a large wound, and presenting, . . . , an exact analogy to the surface

¹⁴ Charles White, *A Treatise on Pregnant and Lying-in Women* (London: E. and C. Dilly, 1773) ; Alexander Gordon, *A Treatise on the Epidemic Puerperal Fever of Aberdeen* (London: G. G. and J. Robinson, 1795) ; Oliver Wendell Holmes, “The Contagiousness of Puerperal Fever,” *New England Quarterly Journal of Medicine and Surgery* 1 (1843): 503-530 ; Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*, trans. K Codell Carter (London: University of Wisconsin Press, 1983). Charles White of Manchester was perhaps the first English author on the subject to suppose that the retention of the lochia led to putrefaction which entered the bloodstream and caused puerperal fever. Alexander Gordon was among the first to show that he could trace which patient would contract puerperal fever by knowing which birth attendant delivered her. Oliver Wendell Holmes wrote an impassioned address citing many examples of apparent contagion of puerperal fever. Ignaz Semmelweis showed that hand washing in chlorinated lime solution significantly lowered the rates of infection in the Allgemeines Krankenhaus lying-in hospital in Vienna.

¹⁵ K. Codell Carter, “Semmelweis and His Predecessors,” *Medical History* 25 (1981): 57-72 ; K. Codell Carter, *The Rise of Causal Concepts of Disease: Case Histories* (Aldersot, UK: Ashgate Publishing Co., 2003) ; K. Codell Carter, *Childbed Fever: A Scientific Biography of Ignaz Semmelweis* (New Brunswick, NJ: Transaction Publishers, 1995) ; David Wootton, *Bad Medicine* ; Hal Hellman, *Great Feuds in Medicine: Ten of the Liveliest Disputes Ever* (New York: John Wiley & Sons, Inc., 2001) ; Sherwin B. Nuland, *The Doctor’s Plague: Germs, Childbed Fever, and the Strange Story of Ignác Semmelweis* (New York: W.W. Norton & Company, Inc., 2003). Ignaz Semmelweis, in particular, has received an enormous amount of attention for his analysis in 1847 that contact with decaying animal-organic matter in post-mortem dissections caused puerperal fever after it was introduced into the birth canal by the hands of attending medical students. While the hand washing program that he subsequently implemented saved countless women from death, the limitations of Semmelweis’ analysis, with its many mistaken assumptions and his unwillingness to modify his theory to fit incompatible data, has been blissfully glossed over by those who have promoted his contemporary veneration. While Semmelweis is, undoubtedly, a hero for pledging his life to saving the lives of these women, had he been less obstinate in his technically incorrect beliefs, he might have saved far more lives.

of a stump after amputation; . . . , that the hand which is relied upon for succor in the painful and perilous hour of childbirth, . . . , may literally become the innocent cause of her destruction; innocent no longer, however, if after warning and knowledge of the risk, suitable means are not used to avert a catastrophe so shocking.¹⁶

Storrs gave a lecture discussing how contact with puerperal fever victims and postmortem dissections evinced disease and death in medical practitioners, family members, husbands, and the recently delivered children of the deceased.¹⁷ In doing so, his work opened the possibility that a far broader range of people were at risk for the disease and, consequently, undermined the assumption that puerperal fever was exclusively a women's disease. The works of these doctors, despite their contemporary prominence, do not enjoy the status accorded to others in the secondary literature.

Instead of a discussion of the medical establishment's worldview and how individual breakthroughs were positioned in their greater intellectual context, the historiography on puerperal fever tends to focus on sensationalized accounts of troubled personalities and interpersonal disputes between doctors. This study offers an alternative interpretation of the primary sources, one that focuses on the deficiencies of the reasoning and the methodologies of the pre-scientific era and on the role of ancient medical

¹⁶ Sir Thomas Watson, *Lectures on the Principles and Practice of Physic: Delivered at King's College London* (London: John W. Parker, 1843), 423-430. Watson goes on to say, "Whenever puerperal fever is rife, or when a practitioner has attended any one instance of it, he should use most diligent ablution; he should even wash his hands with some disinfecting fluid, a weak solution of chlorine for instance: he should avoid going in the same dress to any other of his midwifery patients: in short, he should take all those precautions which, when the danger is understood, common sense will suggest, against his clothes or his body becoming a vehicle of contagion and death between one patient and another. . . . In these days of ready invention, a glove, I think, might be devised, which should be impervious to fluids, and yet so thin and pliant as not to interfere materially with the delicate sense of touch required in these manipulations. One such glove, if such shall ever be fabricated and adopted, might well be sacrificed to the safety of the mother in every labour." His suggestion of hand washing in a chlorinated disinfecting solution was made at least four years before Semmelweis, yet he is rarely given credit for it. It was not until 1890 that sterilized medical gloves were first used in surgery.

¹⁷ Robert Storrs, Esq., "On the Contagious Effects of Puerperal Fever on the Male Subject; or on Persons Not Child-Bearing," *Provincial Medical and Surgical Journal* 9 no.19 (1845): 289-293.

assumptions that were still current in the eighteenth and nineteenth centuries. Even those historians, such as Wootton and Carter, who address the problem of a reliance on ancient medicine, still do not adequately analyze issues of reasoning and language. This study attempts to fill that void.

Thomas Kuhn is not known for work related to puerperal fever, but his influence on the subject of theory formation and its relationship to the intellectual environment is extremely important. Starting with the publication of *The Structure of Scientific Revolutions* in 1962, Kuhn became a very influential thinker in science, including both “hard” and social sciences, as well as the philosophy of science and logic.¹⁸ His concept of the paradigm shift, describing the abrupt replacement of long-held assumptions and theories with new “paradigms” became a dominant area of study and debate over the past fifty years. His basic argument is that scientific progress is not smooth and linear but is instead characterized by long periods of steady accumulative scientific work under an accepted set of assumptions. Eventually this accepted “paradigm” becomes unable to accommodate the discovery of incompatible data, forcing an abrupt overthrow of the old intellectual framework, and the relatively rapid adoption of a new intellectual framework under which to work. He emphasized that each paradigm is both a guide to assist in gathering new information and a limiting factor, circumscribing those who work within it to understand data only as the paradigm allows. This appeared to many to be a form of relativism, a charge that Kuhn discussed and rejected. Although beyond the scope of this paper, the transition from the humoral framework to germ theory marks just the sort of dynamic that Kuhn analyzed and would be a fruitful topic for future historical work.

¹⁸ Thomas Kuhn, *The Structure of Scientific Revolutions* (Chicago, The University of Chicago Press, 1962).

Etiology, Symptomology, Statistics

To better understand puerperal fever's role in the history of medicine, it is important to understand what is now known about this disease: what causes it, how it progresses through the body, and how it can be treated. With the benefit of hindsight, historians today can use this knowledge to understand the paralyzing confusion about this disease that lasted well into the twentieth century.

Puerperal fever is a serious bacterial infection that can develop into puerperal sepsis as the infection spreads into a woman's blood stream. Depending on the pathogenic organism at fault and on how far the infection has progressed before treatment, puerperal fever can be fatal. Unfortunately, a great variety of organisms can cause puerperal fever and the virulence of the strains varies from year to year. This creates difficulties in interpreting the statistics of maternal deaths attributed to puerperal fever. Nevertheless, this data, when properly recorded, reveals cyclical patterns of maternal morbidity due to epidemic puerperal fever.¹⁹

The majority of the organisms responsible for causing puerperal fever, and all of those responsible for causing epidemic puerperal fever, are not normally found in the

¹⁹ Irvine Loudon, *The Tragedy of Childbed Fever*, 155. This cyclical pattern shows that, periodically, the active strains of the organisms which cause puerperal fever became especially virulent. These more tenacious strains were more likely to be spread to puerperal patients and, when infected, these patients were more likely to die. Carefully kept statistics in lying-in hospitals throughout Europe show that, for certain years, epidemics of puerperal fever claimed an unusually high number of women's lives. However, after a time, the bacterial strains would return to a normal, low-lying level of virulence and the epidemics would stop. Puerperal fever cases would continue but more sporadically and more often resulting in recovery. This pattern often led doctors to suppose that treatments they had used to combat the epidemic had eventually proved successful, when in actuality, the statistics were cyclical irrespective of treatment.

vaginal tract of lying-in women. They are introduced from an outside source. Directly after birth, the womb is particularly vulnerable to infection. A number of factors coalesce to create an environment wherein the woman's reproductive tract is temporarily unprotected against pathogens. First, the normal acidity of the vaginal secretions is rendered neutral during the birth process. Second, the cervix is widened and shortened, allowing accessibility to the uterus. Third, the endometrium, the inner lining of the uterus, which constitutes a natural, antibacterial barrier, is stripped away. Fourth, placental separation creates a particularly abraded surface. Fifth, blood vessels of the uterus are exposed, creating an environment ripe for septicemia.²⁰

A number of pathogenic organisms can cause puerperal fever: Groups A, B, C, D, and G Streptococci, Staphylococci, coliform bacteria, anaerobic bacteria, Chlamydia, mycoplasma, and very rarely, *Clostridium welchii*. Some have argued that fecal contamination can cause *E. coli* infections and that even normal vaginal flora can become infective in this opportune environment.²¹ However, the attention of medical historians has always been on the Group A beta hemolytic Streptococcus, abbreviated GAS, which is thought to have been the primary cause of puerperal fever epidemics throughout history.

There are many different strains of GAS, but all hemolytic streptococci are aerobic bacteria, that is, they grow in the presence of oxygen. Their easy communicability comes from the fact that they can live for a surprisingly long time in the

²⁰ Ibid., 7 ; Gail Pat Parsons, "Puerperal Fever, Anticontagionists, and Miasmatic Infection," 425.

²¹ Parsons, "Puerperal Fever, Anticontagionists, and Miasmatic Infection," 8, 11 ; Edward Shorter, *Women's Bodies: A Social History of Women's Encounter with Health, Ill-Health, and Medicine* (New Brunswick, NJ: Transaction Publishers, 1991), 127-128.

open air without losing their virulence.²² While less deadly strains of GAS are common causes of skin infections, the more dangerous strains, such as *Streptococcus pyogenes*, attack the throat. The problem with this strain of bacteria, particularly for birth attendants, is that *Streptococcus pyogenes* is regularly found in the human nasopharynx (the upper area of the throat which lies behind the nose). It may be communicated to the vaginal tract by the hands of the attendants, any family members present, particularly children, or even by the mother herself.²³ In addition to being a major cause of epidemic puerperal fever, Streptococci are also responsible for impetigo, scarlet fever, “inflammatory or malignant sore throat” and erysipelas, an infection of the skin which was often seen in tandem with epidemics of puerperal fever.²⁴

The symptomology of puerperal fever is surprisingly predictable if the infection arises from GAS organisms. Within the first to the third post-partum day, an increasing tenderness begins in the lower abdomen as the infection begins to invade the tissues of the uterus. The infective organisms begin to multiply in this oxygenated-blood rich environment. From here, they may move into the peritoneal cavity of the abdomen causing peritonitis evinced by severe pain, nausea, diarrhea, and fever. Shallow, quick breaths are a response to the pain of a full breath as the diaphragm presses against the stomach. As the infection slowly necrotizes the tissues, gasses are given off that distend and harden the belly. In advanced stages, extremely virulent strains will move into the

²² Shorter, *Women's Bodies*, 117.

²³ Parsons, “Puerperal Fever, Anticontagionists, and Miasmatic Infection,” 425.

²⁴ D. B. Stewart and J. G. Williams, “Bleeding and Purging: A Cure for Puerperal Fever?” *Journal of Hospital Infection* 34 (1996): 85. Referring to a paper read to the Medico-Chirurgical Society of Edinburgh on November 12, 1845 by Dr. Peddie, reprinted in the *Provincial Medical and Surgical Journal*, edited by Robert J. N. Streeten (London: John Churchill, 1846), Dr. Gairdner notes on page 234, “It was also remarked, that almost every individual who had visited at Mrs. K.’s during her illness, complained soon afterwards of one kind or another, particularly with slight feverishness and sore throat; and it was at this time that Dr. Peddie himself became affected in the same way.”

blood stream causing an overall septicemia. The ominous chills associated with the onset of epidemic puerperal fever are a direct result of this blood poisoning.²⁵

The rapid decline of infected patients, as described in eighteenth- and nineteenth-century treatises on puerperal fever, was predictable and heartbreaking. “The patient is immediately seized with the strongest apprehension of her danger, and labors under vast anxiety, her countenance shewing (sic) indubitable marks of the great suffering both of body and mind.”²⁶ “The whole features, indicate anxiety, if not terror, and great debility...The secretion of milk stops, and the patient inquires very seldom about the child.”²⁷ “...there is vast prostration of strength with anxiety, depression of spirits, a disinclination to suckle, carelessness about her child, and watchfulness... an indifference to all external objects, denotes certain and speedy death.”²⁸ According to Alexander Gordon, a telltale final symptom of puerperal fever was a sudden cessation of pain. Often misread as a sign of recovery by relieved family members, Gordon explains that this was, in actuality, a sign that the tissues have necrotized and was the announcement of approaching death.²⁹ When fatal, most puerperal fever victims succumbed during the first week following delivery.³⁰ It was a horribly painful way to die and women in childbed, as

²⁵ Parsons, “Puerperal Fever, Anticontagionists, and Miasmatic Infection,” 438.

²⁶ Thomas Denman, *Essays on the Puerperal Fever, and on Puerperal Convulsions* (London: J. Walter, 1768), 10.

²⁷ John Burns, *The Principles of Midwifery* (London: Longman, Orme, Brown, Green, & Longmans, 1837), 599, 600.

²⁸ Thomas, *The Modern Practice of Physic*, 934, 936.

²⁹ Alexander Gordon, *A Treatise on the Epidemic Puerperal Fever of Aberdeen*, 12-13. This strange symptom was also mentioned by Thomas in *The Modern Practice of Physic*, 925, 929, 936 and C. H. F. Routh, “On the Causes of the Endemic Puerperal Fever of Vienna,” *Medico-Chirurgical Transactions* 32 (1849), reprinted in *The Edinburgh Medical and Surgical Journal* 75 (1851), states on page 249, “The pain generally disappeared an hour before death; the face gradually assuming a more sallowish, yellow, and cadaveric expression.”

³⁰ Meigs, *Obstetrics: The Science and the Art*, states, “The progress of puerperal fever is sometimes so very rapid, particularly in warm climates and hot seasons, as to destroy the patient in forty-eight hours. Even in cases seemingly the most favourable, we should look on the event as doubtful, as the complaint is apt to be

well as their birth attendants, were extremely fearful of this disease and sought every possible way to avoid it.³¹

Unfortunately, the risk of developing a fatal infection was inescapable for the majority of women. Edward Shorter has stated in his book, *Women's Bodies*, that during the era of traditional midwifery four percent of women were likely to contract a serious post-partum infection. He argues that if the average woman in this historical period were to survive to age forty-five, she would likely give birth six times. Using these average estimates, Shorter calculates the lifetime risk of these women to be twenty-five percent. This means that a woman in this environment had a one in four chance of contracting a potentially lethal case of puerperal fever at some point during her reproductive years.³²

However, puerperal fever statistics are difficult to evaluate. Records were not carefully kept in home deliveries and private practice; even in hospitals the numbers are often suspect. Differences of opinion amongst doctors of this period on what constituted puerperal fever have led some to believe the number of cases has been underestimated. Many puerperal fever deaths were also likely to have been misdiagnosed as enteritis or pneumonia.³³ Some have even accused hospitals of intentionally mislabeling the cause of death to hide the high incidence of epidemic mortality.³⁴

accompanied with delusive remissions; and indication arise in its progress which are by no means equal to the danger." 935-6.

³¹ Denman, *Essays on the Puerperal Fever, and on Puerperal Convulsions*, states on page 20, "A disease in which the dangerous symptoms come on with such impetuosity, and where the event is very often fatal, could not fail to alarm every man solicitous for the welfare of his patient. His anxiety would be increased by the want of a distinct history of the disease; and experience would convince him that the method of cure, generally directed, was not to be depended upon."

³² Shorter, *Women's Bodies*, 108.

³³ *Ibid.*, 106.

³⁴ Loudon, *The Tragedy of Childbed Fever*, 191.

The best statistics that can be offered, taking into account these limitations, indicate that in the eighteenth and nineteenth centuries, approximately six to nine cases of sporadic puerperal fever were found in one thousand deliveries. Less than half of these cases terminated fatally. During puerperal fever epidemics, the number of cases skyrocketed and the mortality rate grew toward eighty percent.³⁵ It is estimated that between a quarter and a half a million women died from puerperal fever in the eighteenth and nineteenth centuries in England and Wales alone.³⁶ Helpless in the face of this staggering mortality rate, at least one British physician told his students,

Of those attacked by this disease, treat them in any manner you will, at least three out of four will die. Upon examining the bodies, the uterus, the viscera, and every other part of the abdomen are found to be inflamed. There is a quantity of purulent matter in the cavity of the abdomen, and the intestines are all glued together. We tried various methods, (bleeding, refrigerants, stimulants, mithridate,) but every thing failed.³⁷

³⁵ David Wootton, 211 ; Christine Hallett, "The Attempt to Understand Puerperal Fever," 1 ; Loudon, *The Tragedy of Childbed Fever*, 190.

³⁶ Loudon, *The Tragedy of Childbed Fever*, 6.

³⁷ Robert Gooch, *An Account of Some of the Most Important Diseases Peculiar to Women* (London: John Murray, 1831), 9. Burns, *The Principles of Midwifery*, also states on pages 602-603, "With regard to the best mode of treatment, there has been a great difference of opinion, which partly depends, on giving the name of puerperal fever, to different disorders. I am sorry that I find it much easier to say, what remedies have failed, than what have done good."

Early Accounts

Historical interest in puerperal fever has centered primarily on the eighteenth and nineteenth centuries. However, the disease can be found in ancient Greek and Hindu medical texts dating back as far as 1500 BC. These texts refer to a potentially fatal fever in women who had just given birth and some, including one by the Greek physician Soranus, provide advice to birth attendants on proper hygiene, leading some to suggest that these early writers may have recognized the role that birth attendants played in spreading infection.³⁸

The well known story of Dromeades in Athens in the fifth century B.C. could have easily been written two thousand years later. It described how Dromeades' wife, after birthing a daughter, came down with chills and a high fever on the second day after delivery. Her symptoms included abdominal pain, irregular breathing, and thick, muddy urine. On the sixth day, her body wracked with spasms and delirious, she died. The description of her illness and the care with which the author recorded each symptom has led historians to confidently conclude that, not only was this puerperal fever, but that these symptoms were recognized as being a frequent complication of labor and delivery in the ancient world.³⁹

³⁸ Caroline M. De Costa, "The Contagiousness of Childbed Fever: A Short History of Puerperal Sepsis and its Treatment," *The Medical Journal of Australia* 177 no. 11/12 (2002): 668.

³⁹ Shorter, *Women's Bodies*, 103.

An Alsatian proverb states that “heaven stays open nine days for the woman in childbed.”⁴⁰ According to Edward Shorter, this refers to the realization that puerperal fever symptoms may not surface for a few days after delivery and the hope that, if the mother did not show signs of infection by this time, she had likely escaped the disease. Shorter also describes a Styrian belief that “if one woman dies in childbed, so must two others also die soon afterward.”⁴¹ He argues that this saying alludes to the communicability of the disease.

Despite the ease with which we find references to post-partum infections throughout the ancient and medieval periods, it was not until the seventeenth century that the scope of the disease became clear. And it was not until 1716 that the term “puerperal fever” was coined in Edward Strother’s *Criticon Febrium*.⁴² Until this time, the disease was termed “the weed” or “milk fever.” It was assumed that a mother’s milk originated in the blood and that the fever accompanied the milk when it left the blood and entered the woman’s breasts.⁴³ Indeed, throughout the early modern period, popular and medical characterizations of “milk fever” described it as a normal part of the birth process which resolved itself within a few days.⁴⁴ Still, it was also recognized that this “normal” fever could turn deadly and take the life of the mother. Apparently, a baseline infection rate of

⁴⁰ Ibid., 104.

⁴¹ Ibid.

⁴² Loudon, *The Tragedy of Childbed Fever*, 15. Edward Strother, *Criticon Febrium: Or, A Critical Essay on Fevers; with the Diagnosticks and Methods of Cure, in all the different species of them* (London: Charles Rivington, 1718), 169, 194, 203, 212.

⁴³ Ibid., 18-9 ; Shorter, *Women’s Bodies*, 105. However, Denman, *Essays on the Puerperal Fever, and on Puerperal Convulsions*, states on pages 15-16, “This fever is easily distinguished from the febrile disturbance, occasioned by the milk, if it comes on at the time the milk is expected, by the symptoms before mentioned, and by the flaccidity of the breasts; yet the consent between these and the *uterus* is so intimate, and the transition of the humours from one to the other so instantaneous, that an excuse may be readily accepted for those who have mixed diseases, in some respects similar, yet essentially different.”

⁴⁴ Shorter, *Women’s Bodies*, 105.

the less virulent bacteria that cause puerperal fever was ubiquitous in the population. Occasionally, more virulent strains produced fatal effects as the local uterine infection spread to produce peritonitis and septicemia.

Despite these sporadic descriptions of post-partum fevers, very few tracts on fevers in puerperal women were published in Britain before 1760.⁴⁵ However, an epidemic of puerperal fever at the British Lying-in Hospital in that year generated a flurry of interest in the medical community.⁴⁶ Throughout the late eighteenth and early nineteenth centuries, many medical practitioners began publishing descriptions of puerperal fever cases they had attended, hypothesizing on the cause of the disease and recommending various methods of treatment. By 1860, an “extensive synthesis of knowledge” had been offered for the consideration of the medical community on what had, by then, become a fearful scourge to medical practitioners and their parturient patients.⁴⁷

Unfortunately, this abundance of new data was forced into the same old categories. Little had changed in models of disease causation from classical to modern

⁴⁵ Hallett, “The Attempt to Understand Puerperal Fever,” 2. Before the creation of lying-in hospitals in the seventeenth century, women gave birth at home. The low incidence of maternal death in the early modern period can be traced to the fact that the majority of puerperal infections would be caused by infective organisms within the mother’s normal environment. It has been argued that women have a higher resistance to pathogens found in their own homes to which they are routinely exposed. Therefore, the mortality rate of home births attributed to puerperal fever was almost always lower than found in the hospital environment. Some historians argue that the rise in puerperal fever deaths can be directly correlated to the transition from traditional midwifery to obstetrics dominated by male doctors. The statistics are not available to prove this assertion, but it is highly unlikely that general practitioners were more or less likely to spread puerperal fever than midwives in a home-delivery setting. It was in the hospitals, that epidemic puerperal fever ranged out of control and inflated the mortality statistics.

⁴⁶ Ibid. The first epidemic of puerperal fever was recorded in 1646 at the Hôtel Dieu, a maternity hospital in Paris. John Leake, *Practical Observations on the Child-Bed-Fever: Also on the Nature and Treatment of Uterine Haemorrhages, Convulsions, and such other Acute Diseases As are most fatal to Women during the State of Pregnancy* (London: R. Baldwin, 1775), states on page 234, that twenty-four women died in the London epidemic between the 12th of June and the end of December. London lying-in hospitals had apparently never seen such mortality, but epidemics such as these soon began to recur frequently in Britain and on the Continent.

⁴⁷ Ibid., 3.

times. Many of the same basic tenets of ancient medicine were still held to be true, though they were often disguised in modern metaphor. In the pre-scientific world, the assumption was that there was an absolute truth to be found, one that no data would ever contradict. People were taught, often from ancient texts, that understanding these received final words about a subject was the key to operating in that realm. This attitude toward knowledge had powerful consequences. If it appeared that some discovery was incompatible with a “true” theory, either the data had to be wrong or the physicians were somehow misunderstanding the implications of how the theory would manifest itself in actual cases.

Humoural Theory: Hippocrates and Galen

The humoural theory, put forth by the Hippocratic school in ancient Greece, was based on four cardinal humours that were thought to exist in every individual at varying levels, dependent upon the person's disposition. These humours included blood, phlegm, cholera or yellow bile, and melancholy or black bile. A delicate balance of these four humours existed within each individual body as a whole and within each body part. The levels of these humours established mental and physical health as well as determining the individual's personality. Any imbalance of these humours would cause suffering in the form of physical disease or mental anguish. Hippocratic writings spent a great deal of time describing how diet and lifestyle contribute to healthy or unhealthy habits and dispositions. Treatment of these conditions involved making changes that would bring the humours back to their natural balance.⁴⁸

The Roman physician Galen of Pergamon went one step further. His description of anatomy was based on the dissection of monkeys, as human dissections were still taboo, but he was able to expand upon the humoural theory and advocate more elaborate treatments of disease. He correlated the four humours to the four elements: blood with warm, moist air; phlegm with cold, moist water; yellow bile with warm, dry fire; and black bile with cold, dry earth. He then used treatments that corresponded with these properties to influence the humours within the body, applying medicines with the

⁴⁸ Joan Lane, *A Social History of Medicine: Health, Healing, and Disease in England, 1750-1950* (London: Routledge, 2001), 2.

opposite - warm/cold, moist/dry - combination of whatever humour was overly dominant.⁴⁹ This primitive notion harkens back to the pre-Socratic thinkers, especially Anaximenes, in the century before Hippocrates and their attempts to explain all nature by reducing it to a few primary substances and then account for the motion of these substances as a product of the constant pull of opposites.⁵⁰

Galen staunchly defended and promoted the treatment of venesection, or bloodletting, for disease. Venesection involved making an incision in a vein and allowing the venereal blood to flow out of the body. This treatment was based on the belief that black bile was “not a pure liquid, but one that darkens other liquids, producing states of suffering.”⁵¹ The venereal blood, lacking oxygen, is much darker than arterial blood. Galen also compiled lists of medicinal preparations which contained a variety of ingredients “from which the body would select what the disease required.”⁵²

Unfortunately, the cornerstone of Hippocrates’ humoral theory was a reliance on data that was, in principle, unobservable. It yielded explanations with no test implications. As humours were characterized in most versions of the theory, including the mechanistic variation, one could not correlate humours with symptoms because humours were quasi-physical. Since one could not observe the phenomena in question, such as levels of humoral imbalance, it was impossible to single out causal agents. If, for example, a physician postulated that puerperal fever was produced by cold weather – an assertion that could be easily tested – then an investigation could be conducted to

⁴⁹ Ibid.

⁵⁰ Geoffrey Stephen Kirk, John Earle Raven, and Malcolm Schofield, *The Presocratic Philosophers: A Critical History with a Selection of Texts* (Cambridge, UK: Cambridge University Press, 1983).

⁵¹ Michael Worton and Nana Wilson-Tagoe, *National Healths: Gender, Sexuality, and Health in a Cross-Cultural Context* (London: UCL Press; Cavendish Publishers, 2004), 10.

⁵² Lane, *A Social History of Medicine*, 2.

ascertain whether the incidence of the disease was less in warm weather. If, on the other hand, one said that an excess of a given humor caused disease X when it was impossible to measure said humor, one was forced to assume that this humor was present in those that had the disease, i.e. that it was a necessary condition for the disease to occur. At this point, the humor is no longer a cause of the disease but one of its defining characteristics. Lacking any means, even theoretical, to remove the humor to see if the disease could still thrive, the humor becomes an unquestioned characteristic of the disease and not a cause. It has covertly become part of the disease's definition. There is nothing that could be examined or measured that would count for or against that explanation.

Another aspect of a humour's unobservability was that, in many of its formulations, a humour was characterized as internally generated by ill-defined processes, independent of external influences. Insofar as it was thought that puerperal fever could be internally generated, any new data was undermined by the fact that physicians believed that external factors could not be the "true" cause of the disease as long as a significant portion of the disease might have been spontaneously generated within the patient. This undermined the creation of effective prevention programs since it was assumed by physicians that no preventative treatment, no matter how well it may have worked in any one instance, was likely to stop another incidence of the same disease.⁵³

⁵³ Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*. This was an obvious flaw in the work of Ignaz Semmelweis as he used the category of "spontaneous auto-infection" to explain any residual cases that occurred after he had implemented his hand washing technique in the Allgemeines Krankenhaus. Semmelweis was also unable to account for the large number of cases that occurred in home deliveries in Britain with no possible connection to post-mortem dissections. Because Semmelweis had specifically argued that an introduction of "animal-organic matter" from these dissections was the ultimate cause of puerperal fever, he was forced to resort to "spontaneous auto-infection" to explain away the contradictions in his theory.

Miasma Theory: The Persistent Dead-End

Another subtle yet powerful element undermining a physician's ability to make sense of infectious disease was the curious theory of miasmas. Miasma theory, like humoral theory, was another popular view of disease causation that had its roots in ancient Greece. Less a formal theory than an ongoing accumulation of conjectures, this view held that foul-smelling air or some noxious quality of the atmosphere was responsible for causing disease. Revived during the middle ages, it was thought to be capable of causing almost any infectious disease and was used to explain all epidemics.⁵⁴

This theory was based on the observation that rotting items smelled bad. Ergo, smelly items must be rotting. It was also understood that coming into contact with rotting substances, eating rotting meat or coming into close contact with animal and human wastes for example, caused people to become violently ill. These observations gave rise to the theory that the decomposition of organic matter gave off foul smelling air that could, in turn, cause disease.

Throughout the early modern period, regional governments circulated letters decrying practices, such as the keeping of pigs and silkworms within certain residential areas, and of the overflow of ill-managed cesspits because the stench would spread disease.⁵⁵ The odiferous particles entering the air were referred to, in the modern period,

⁵⁴ Lindemann, *Medicine and Society in Early Modern Europe*, 179-184.

⁵⁵ Carlo M. Cipolla, *Miasmas and Disease: Public Health and the Environment in the Pre-Industrial Age* (Essex, UK: Yale University Press, 1992), 10.

as “putrid effluvium.”⁵⁶ Overcrowding in urban areas, hospitals, jails, etc. were recognized as producing infectious diseases. These environmentally produced diseases were explained using the miasmatic theory and fevers, in particular, that were spread by miasma were seen as routinely infectious.⁵⁷

In fact, the term “epidemic” at this time did not necessarily imply a large number of cases. Epidemics were diseases that were caused by “atmospheric, cosmic, or terrestrial influences” and could just as easily have been an epidemic involving only one patient.⁵⁸ It was the environment that created the conditions under which an epidemic could take place. This disease model helped to explain why diseases were often local occurrences and why many epidemics were seasonal. The “epidemic constitution” helped to explain why all people within a given area, and under the same atmospheric influence, did not get sick. Only those with certain predispositional factors would have their humours altered by the atmospheric influence.⁵⁹

The notion, used by many physicians, that there was such a thing as a “predisposition” to contracting puerperal fever, or any other disease, proved to be a

⁵⁶ Hallett, “The Attempt to Understand Puerperal Fever,” 3, 11-12.

⁵⁷ *Ibid.*, 13.

⁵⁸ Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*, 86 ; Carter, “Semmelweis and His Predecessors,” 65. Samuel Kneeland, “On the Contagiousness of Puerperal Fever,” *The American Journal of Medical Science* 11 (1846), states on page 49, “Another point which has much confused this question is the vague signification of the word ‘epidemic,’ which by many is understood as implying some mysterious quality of a disease, in virtue of which it attacks a great number of individuals...—Again, a disease is said to be ‘epidemic,’ when its propagation is attributed to a morbid principle contained in the air. Strictly speaking, the term ‘epidemic’ implies a *number* of patients that such an affection is judged to be *epidemic*; and it is also by the *epidemic* quality attributed to the affection that the *number* of patients is explained—here, then, is a circle of confusion, in which the cause becomes the effect, and the effect the cause, according to the will or caprice of the observer.”

⁵⁹ Thomas, *The Modern Practice of Physic*, states on pages 932-933, “Some have been accustomed to look on [puerperal fever] as only a simple modification of the known species of fever, taking its origin from the leaven of the prevailing epidemic constitution, whether inflammatory or putrid, modified by the habit of body, the mode of living, the age and temperament of the patient, the preceding causes, the season of the year, and temperature of the air, &c.”

powerful impediment to understanding the causal framework of disease. Typically, a doctor would have a clear notion of several causal factors that produced the disease, but no way of explaining why its effect was not universal. Thus, they created the notion that some patients had a predisposition for the disease and that they, and only they, would contract the illness when exposed to the other causative agents.

The logic of this is clearly defective. If one asserts that a given ailment is caused by X, Y, and Z in only those patients “predisposed” to getting the disease, they are, at best, saying that there is another cause for the disease. Even more troublesome: If one has no independent way of determining which potential patients have such a predisposition, then the predisposition becomes part of the definition of the disease. Patients with puerperal fever, *a priori*, have the disposition, those without puerperal fever, do not. This renders the concept of the “predisposition” logically incapable of explaining the ailment, or of assisting in the discovery and analysis of its true cause. It creates a dead-end for those who use the concept, covertly characterizing the disease as inherently mysterious and unsolvable.

Miasmatic theory has often been incorrectly regarded as leading physicians toward a theory of droplet infection wherein disease could be transmitted through the air. But in the writings describing miasmas as the cause of disease, there were no references to particles transferring diseases through the atmosphere; miasmas were instead characterized as: “poisonous vapors,” “foul air,” and “morbid influences,” often attributed to astrological influence, “mineral exhalations,” “fermenting vegetable fluids,”

etc.⁶⁰ Like humours, miasmas were not a useful method of explanation because a miasma was not observable. Although it was sometimes spoken of as marginally tangible, much like a foul smelling air, it was more commonly described in more abstract terms of noxious influences. As such, the miasma often took on the character of a supernatural entity, powerful yet hidden and yielding no possible methods by which to isolate, and thereby test, its effects. This spirit-like “influence” represented a dead-end for any inquiry into disease. If it could not be isolated and identified then it could be invoked as the cause of anything and no data could be relevant to the claim.

⁶⁰ G. Motherby, *A New Medical Dictionary; or, General Repository of Physic* (London: J. Johnson, 1775), 257.

Medical Education: The Resilience of a Dogma

The tenets of this ancient, “classical” worldview were fundamental to eighteenth-century medical education. Such an education included extensive study of Hippocrates and Galen who, despite their ancient origins, were still regarded as the foundation of eighteenth century medicine. Discussing the Oxford curriculum, M. D. Warren quotes Dr. Campbell Hone as writing,

...the licenses for the practice of medicine and surgery were issued by the University without any adequate inquiries as to the qualifications of application, and the requirements for the degree of Bachelor of Medicine were meagre indeed. The candidate must have taken his M.A., have spent three years in medicine and attended lectures of the Regius Professor on Hippocrates and Galen twice a week...The D.M. degree could not be taken till four years after the B.M. degree, and during those four years the candidate had to attend the lectures of the Regius Professor and also to give three or four lectures himself on Galen.⁶¹

University education, even one specializing in medicine, was primarily grounded in classical learning, particularly in Latin and Greek. Hippocrates furnished the primary metaphor of humoural-based health; Galen promoted bloodletting as a primary treatment to reestablish humoural balance. Neither seriously considered the idea of contagion, instead arguing that environmental factors bred disease which preyed upon individuals with certain predisposing factors.⁶² Their combined doctrines were so pervasive that they formed a powerful intellectual roadblock to medical advances in the eighteenth century.

⁶¹ Warren, M. D. “Medical Education During the Eighteenth Century.” *Post Graduate Medical Journal* 27 (1951): 306.

⁶² Lindemann, *Medicine and Society in Early Modern Europe*, 9-17.

This encouraged *ad hoc* interpretations of the humoral theory. An *ad hoc* hypothesis is a fallacious technique whereby an established idea is saved in the face of fundamentally incompatible data, data that would normally mark the overthrow of the theory. In its purest case, it takes the form of a new hypothesis, added to an established theory, *after* an apparently conflicting piece of data is discovered. The new *ad hoc* hypothesis claims that the established theory is now compatible with this new data.

Until the data was discovered it was not previously thought to be a consequent of the theory. Perhaps it was even thought to be a falsifying instance of that theory, but in order to save the theory the *ad hoc* hypothesis extends the theory to include the otherwise negative discovery. The effect is to “save” but seriously weaken it. If the theory can accommodate discoveries that had been thought to falsify it, little of the original theory’s explanatory power will remain.

For contemporary scientists, it is an error of logic, but to physicians of the period, working before the details of scientific logic had been developed, it was a way to subsume newly discovered data within the ancient humoral theory, a theory they were not yet inclined to fundamentally challenge. The *ad hoc* adjustment was often presented as an advance and was couched in the language of newly developed metaphors, utilizing the “vocabulary of machine culture,” to describe how Hippocratic and Galenic principles could still be considered true and their attendant treatments valid.⁶³ Modern medical treatises on puerperal fever reveal how many models of disease causation continued to be influenced by the classical, Hippocratic and Galenic worldview, while being expressed in the emerging modern, technical language of mechanism.

⁶³ Erin O’Connor, *Raw Material* (Durham: Duke University Press, 2000), 10.

Mechanism: A New Metaphor

While miasmas were openly blamed for causing disease well into the nineteenth century, the humoral theory maintained its hold on medical discourse in a more obscure fashion. Scientific advancement in biology in the early modern period had begun to chip away at the foundation of the humoral theory. For example, William Harvey's description of the circulation of the blood in 1628 directly refuted Galen's justification for bloodletting as, in a closed circulatory system, there was in fact no difference between venous and arterial blood.

The ancients were sure that blood was fundamental to human life, but its source, pathway, and exact function eluded them. The humoral theory, by the time it was espoused by Galen, maintained that there were two kinds of blood, arterial and venous, each following a separate path. Arterial blood was continually produced by the heart, flowed through the body, and was consumed by the organs. Venous blood followed a similar path: only its source, the liver, was different. The lungs were thought to cool the blood.

This theory was so entrenched, and portions of it so old, that it had achieved iconic status and was, for many, an unchallengeable maxim. William Harvey, a man who did the most to undermine this system noted,

We are too much in the habit, neglecting things, of worshipping specious names. The word blood, signifying a substance, which we have before our eyes, and can touch, has nothing of grandiloquence about it; but before such titles as spirits, and

calidum inaatum or innate heat, we stand agape. But the mask removed, as the error disappears, so does the idle admiration.⁶⁴

Harvey's arguments and conclusions, derived from meticulous dissections and observations, were carefully presented in 1628 in a small volume titled *De Motu Cordis* (*On the Motion of the Heart and Blood*). After evaluating several characterizations of the bloodstream popular at the time, some of which Harvey traced back to Galen himself, Harvey revealed his empirical and experimental data to show that such theories were unworkable. For example, based on his measurements of the heart rate and the amount of blood it pushed out with each stroke, if estimated at one ounce, the heart, were it the source of all arterial blood, would need to produce 166 pounds of blood per hour.⁶⁵

His solution was that the bloodstream was a single, circulating, closed-loop system. There could be no good blood or bad blood because all blood flowed through the same system. This was a demonstrable conclusion, drawn on publically available observations. It was all, in the spirit of the new mechanism, entirely physical. Given the near-sacred status of the system it overthrew, Harvey's conclusions faced only modest opposition and were widely adopted.

Harvey's insights came at an auspicious time, for the next century erupted with new knowledge about fluids and their behavior. Fascination with hydraulics was about to begin. Within twenty years of Harvey's publication, Evangelista Torricelli invented the barometer enabling precise measurement of atmospheric pressures. In 1661, Christiaan

⁶⁴ William Harvey, "On Generation," in *The Works of William Harvey, M.D.: Physician to the King, Professor of Anatomy and Surgery to the College of Physicians*, trans. Robert Willis (London: C. and J. Adlard, 1847), 511.

⁶⁵ William Harvey, "An Anatomical Disquisition on the Motion of the Heart and Blood in Animals," in *The Works of William Harvey, M.D.: Physician to the King, Professor of Anatomy and Surgery to the College of Physicians*, trans. Robert Willis (London: C. and J. Adlard, 1847), 9-86.

Huygens measured the elasticity of gases, in 1676 Denis Papin developed the air pump, and by 1728 Stephen Hales had devised the means to measure blood pressure. Finally, in 1728, Daniel Bernoulli publishes his opus, *Hydrodynamics*.⁶⁶

These pioneers were discovering and developing the principles which describe the behavior of fluids under pressure, in the open air, and traveling through tubes. The topic captured the imagination of inventors and physicians alike and hydraulics became the new metaphor for understanding liquids, even in the human body. With it, Hippocrates and Galen's grip on the workings of the human body had been weakened, but not completely broken.

The father of the new "mechanism" was Rene Descartes. Descartes was a powerful mathematician – he invented analytic geometry – and a philosopher who, more than any other, presented an elaborate and carefully reasoned attempt to explain and justify a mechanical understanding of the universe, including the human body. He argued that events in the physical world are exclusively a product of matter in motion. As such, they are observable and, to the limits of our understanding, predictable. In *Meditations on First Philosophy*, Descartes goes to considerable length to establish that the changeable, corporeal world is distinct from, and operates under a different set of principles than, the eternal, non-corporeal world comprised of God, minds and souls.⁶⁷ To understand the physical world, he argues, we have a powerful tool, mathematics, which is itself eternal but can be applied to the temporary items of the physical realm.

⁶⁶ Bryan Bunch and Alexander Hellemans, *The History of Science and Technology* (New York: Houghton Mifflin Co., 2004), 187, 191-2,199, 215, 229.

⁶⁷ René Descartes, *Meditations on First Philosophy: With Selections from the Objections and Replies*, trans. John Cottingham (Cambridge, UK: Cambridge University Press, 1996).

His descriptions, which isolated the human body from the soul, supplied the foundation which made a scientific, experimental medicine possible. Animals were purely physical, as was the human body. Spiritual entities were non-physical. Only a human possessed both: a spiritual, eternal, non-physical soul, and a physical, temporary, body. This hard “dualism,” the separation of things, including human beings, into distinct physical and non-physical categories, had an important consequence for medicine. Descartes went so far as to argue that the human body was an *automata*, albeit one with a soul.⁶⁸ But there was nothing about the body itself that was not matter-in-motion. All of it was corporeal therefore all of it was observable. Humans had souls but they were separate and distinct and not an appropriate topic for medicine. The thinkers of the eighteenth century did not have a fully-formed notion of experimental logic. Nor did they have the sophistication in hypothesis creation and testing that we now call science. But, after Descartes, they did have a solid structure for understanding the human body without resorting to unobservable, spirit-like entities in their explanations. While we find the primary literature full of physicians using the new mechanistic concepts the notion of non-observable entities hung on tenaciously.

Knowing that the humoral theory had practical disadvantages, the medical establishment sought to rework the theory so that it would be compatible with recent scientific discoveries. The first attempt to create a modern metaphor of disease causation came from this philosophical theory of mechanism. In the early eighteenth century, Hermann Boerhaave, began to incorporate Descartes’ mechanism into his descriptions of

⁶⁸ René Descartes, “The Treatise on Man,” in *The World and Other Writings*, trans. Stephen Gaukroger (Cambridge, UK: Cambridge University Press, 1998).

disease, particularly fevers.⁶⁹ The scientific revolution and early industrial revolution spurred an understanding of the body as a machine whose internal parts worked together in a mechanical system of healthy operation. If parts of the machine wore out or broke, or if corrosive substances entered the system, disease would follow.

Boerhaave's work proved highly influential. Later writers on puerperal fever, and on fevers in general, were particularly influenced by the mechanistic metaphor of the circulatory system as a fluid hydraulic device. By describing the circulatory system in such a way, humoral descriptions of imbalance could be understood as mechanistic descriptions of pressure. Any blockage in the hydraulic valves would cause backups and dangerous stagnation in the system. Pressure would build to an unhealthy degree. Blood would pool and stagnate causing the liquids to putrefy.⁷⁰

Thus, a return of this crucial system to normal functioning would ensure that the body would heal itself, as long as the treatment was delivered before too much damage to the system had occurred. Galen's justification for the use of venesection may have lost its theoretical underpinnings, but physicians were still able to maintain, *ad hoc*, the use of venesection as a popular treatment by pointing out that it was a perfect way to relieve the pressure of inflammation and allow for the return of proper blood flow.⁷¹ The

⁶⁹ Hallett, "The Attempt to Understand Puerperal Fever," 4-5.

⁷⁰ *Ibid.*, 4.

⁷¹ Thomas, *The Modern Practice of Physic*, states on pages 926 and 930, "By an early attention to the disease on its first approach, we may often subdue it, and prevent the inflammation from proceeding to any great height. Our immediate and speedy care ought, therefore, to be directed towards diminishing the quantity of the circulating fluids, and weakening the action of the heart and arteries; and this is to be done by drawing blood from the system, regulating the quantity which we take away by the violence of the symptoms, the state of the pulse, and the age and habit of the patient...Such a decision will soon be justified by a greater freedom in the action of the arterial system, by an abatement of the languor, and by a diminution of the pain and tenderness."

mechanistic metaphor allowed the medical establishment to maintain their time-honored traditions and admiration of ancient icons while appearing modern and scientific.

A number of conditions were posited as causing these blockages. According to her examination of eighteenth century documents on puerperal fever, Christine Hallett chronicles that stagnation of the blood, or of other important bodily fluids, was recognized as being caused by:

...pressure, contortion, or erosion, acrid substances either taken as food, or applied externally, severe cold, and contusions...anything acrid which entered the blood vessels themselves and caused them to contract...anything which caused the blood to 'concrete or cohere', such as 'too great motion; a consumption of the thinner parts of the blood by sweats, urine, spitting, or a diarrhoea.'⁷²

She also argues that it would have been easy for this wide explanation of the many causes of blockage, resulting in dangerous inflammation, to have led doctors to the realization that the natural course of pregnancy and delivery was likely to create the conditions under which blockages occurred.⁷³

There was, therefore, argument in the eighteenth century over whether puerperal fever was, in fact, a disease of the body, or a natural consequence of childbirth that should be left alone to resolve itself. Perhaps due to the staggering figures of maternal mortality attributed to puerperal fever in the lying-in hospitals throughout Europe, the majority of the medical establishment decided to advocate for intervention. The central problem came down to what kind of intervention would be applied and this was

⁷² Ibid.

⁷³ Ibid., 7-8. Denman, *Essays on the Puerperal Fever, and on Puerperal Convulsions*, states on page 16, "Some constitutions are naturally subject to diseases proceeding from a redundancy of the quantity, or an exaltation of the quality of the bile; and that the secretion of that humor is much disturbed during gestation, we need no other proof than the obstinate costiveness, or almost perpetual vomiting of bilious matter, with which many are afflicted at that time."

dependent on whether a doctor supported a view of puerperal fever being fundamentally an inflammatory or a putrid disease.

Inflammation Theory: The Bleeding Continues

If puerperal fever was primarily an inflammatory disease, then the mechanistic metaphor of circulatory distress would explain its disease process. Puerperal fever would be classified as a disease of the circulatory system, either of the blood's composition or of its action within the vessels, and early, "copious" bleeding would be prescribed as its cure. Inflammation theory, a body of ideas centered on this pooling and resultant stagnation of blood, was essential to the eighteenth-century understanding of puerperal fever and appeared in works by such preeminent authors as William Cullen, John Leake, Alexander Gordon, Thomas Denman, William Campbell, and John Clarke.⁷⁴

Treatments used under inflammation theory were based on the idea of "remote counter-excitement." With its theoretical foundation in humoural theory, treatments such as bleeding and blistering (also known as vesication, this treatment involved raising blisters on the patient's skin) were thought to treat internal inflammation that doctor's could not reach by "determining the inflammation to the external parts, and thereby lessening it on the internal ones..."⁷⁵ "...the manifest utility of blistering near the part affected in inflammatory diseases leads us to think, that blistering, by deriving to the

⁷⁴ William Cullen, *First Lines of the Practice of Physic* (Edinburgh: Bell & Bradfute, 1796) ; John Leake, *Practical Observations on the Child-Bed-Fever*; Gordon, *Epidemic Puerperal Fever of Aberdeen* ; Thomas Denman, *Essay on the Puerperal Fever* (London: J. Cooper and J. Walter, 1773) ; Thomas Denman, *Essays on the Puerperal Fever, and on Puerperal Convulsions* ; William Campbell, *A Treatise on the Epidemic Puerperal Fever: as it prevailed in Edinburgh in 1821-22* (Edinburgh: Bell & Bradfute, 1822) ; John Clarke, *An Essay on the Epidemic Disease of Lying-in Women, of the Years 1787 and 1788* (London: J. Johnson, 1788).

⁷⁵ Thomas, *The Modern Practice of Physic*, 931.

skin, and producing an effusion there, relaxes the spasm of the deeper seated vessels.”⁷⁶ Thus, blistering agents were used as counter-irritants that somehow pulled the blood away from the areas of congestion thereby relieving pressure in much the same way as did bleeding by venesection or the use of leeches.

As a theory which had the power to illuminate the development and progression of puerperal fever but also to validate a treatment that had ancient theoretical support, albeit one that required the employment of inaccurate assumptions, inflammation theory was held by the majority of the medical establishment as puerperal fever’s most valuable explanatory theory both in practical and academic circles.⁷⁷ However, as a direct descendant of humoral theory, this mechanistic classification would invite doctors to conceive of puerperal fever as a disease that was expected to behave in ways predicted by that theory. This fact elucidates the troubling underestimation of puerperal fever throughout the eighteenth century.

Putrid theory was the competing theory of disease causation in eighteenth-century treatises on puerperal fever. It described diseases such as puerperal fever as being caused by an introduction or absorption of putrefying matter that would in turn create putrefaction in the tissues with which it came into contact. Putrid theory in the eighteenth century was formed as a practical application of miasmatic theory in diseases which involved bodily systems other than the respiratory system. As miasmatic theory was initially fashioned to deal with illnesses that were thought to be taken in through

⁷⁶ *Encyclopaedia Britannica* 11 (Edinburgh: A. Bell and C. Macfarquhar, 1797), 131 ; Robley Dunglison, *A Dictionary of Medical Science* (Philadelphia: Lea Brothers & Co., 1895), states on pages 148-149, “...a blister plaster...when applied to the skin, irritates it and occasions serous secretion, raising the epidermis and inducing vesicles, as cantharides, mustard, ammonia, etc.”

⁷⁷ Hallett, “The Attempt to Understand Puerperal Fever,” 4.

respiration, putrid theory explained how that process occurred within the body but also extended the theory to describe how the “putrid effluvium,” or other acrid substances which entered the body through other means, would set off a chain reaction of putrefaction.

Putrid theory would lead to some scientific insights in the nineteenth century, namely in Ignaz Semmelweis’ animal organic-matter thesis, but in the eighteenth century it was little understood as anything more than a mechanistic version of miasmatic theory, albeit with a more extensive application.⁷⁸ As in the argument over whether inflammation was normal or not, doctors also speculated whether putrefaction arose as a disease process or was simply the body’s attempt to get rid of these poisonous substances.⁷⁹

While doctors advocated for a more inflammatory or a more putrid understanding of puerperal fever, most sat somewhere in the middle as it was still unclear as to whether the putrefying bodily tissues were a result of some kind of infection with acrid matter or if it was caused by the suppression or obstruction of fluids.⁸⁰ Eighteenth-century treatises on puerperal fever were fixated on the question of which came first, the inflammation or the putrefaction.

⁷⁸ Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*.

⁷⁹ Hallett, “The Attempt to Understand Puerperal Fever,” 13-15.

⁸⁰ *Ibid.*, 11-13.

Pathology: New Techniques, Old Results

The nineteenth century brought further confusion for doctors dealing with puerperal fever with the ideological shift in academics toward the dominance of pathology.⁸¹ In an attempt to make the medical classroom more scientific, medical schools began using postmortem dissection to teach their students the physiological changes that took place within the diseased body. The dissecting room became the new arena where students of medicine would learn about the disease processes that the mechanistic theory had uncovered.

However, for doctors writing about puerperal fever, postmortem dissection became yet another avenue of divergence and disagreement. Those who postulated that the disease was fundamentally inflammatory began looking for the beginnings of inflammation in the bodies of the mothers they dissected.⁸² As the analysis of morbid structural changes was now the holy grail of nineteenth-century science-based medicine, the findings of these autopsies fueled further debate as to the “seat” of the disease or where and how it originated in the body.⁸³

Because puerperal fever is a bacteriological infection which ultimately leads to significant destruction of tissues, the type of organism and the amount of damage done to the internal organs before the patient’s death would produce differing degrees of

⁸¹ Meigs, *Obstetrics: The Science and the Art*, refers to both the “library of this discreditable warfare of opinions” on page 615 and the “scandalous confusion in Medicine as to child-bed fever” on page 617.

⁸² Hallett, “The Attempt to Understand Puerperal Fever,” 10-11.

⁸³ Loudon, *The Tragedy of Childbed Fever*, 15-20 ; Parsons, “British Medical Profession and Contagion Theory,” 139.

damage.⁸⁴ Nineteenth-century treatises on puerperal fever almost always published the findings of autopsies and many doctors argued over whether the disease was inflammatory or putrid and whether it really began in the uterus as had been previously supposed or if it originated in the omentum, perineum, or even the intestines. The debate yielded few areas of consensus among the many writers tackling the subject and led some to return to the view held in the early eighteenth century that puerperal fever was not a unique disease at all. It was instead declared that puerperal fevers were a group of many distinct illnesses that happened to occur during the puerperal period.⁸⁵

Pathological findings that were used to argue for the seat of puerperal fever originating in one organ or another fueled a return to growing lists of possible explanations and classifications of puerperal fever.⁸⁶ In 1846, Samuel Kneeland wrote,

...indeed since morbid anatomy has of late years so absorbed the attention of physicians, constant attempts have been made to localize the disease in question. Having been supposed from the time of Hippocrates to that of Boerhaave, to be dependent on an inflammation of the uterus, it was afterwards localized in the peritoneum by Johnston, in England, Walter, in Prussia, and Bichat, in France; and the name of peritonitis was given to it. Then as inflammation of the uterus was found combined with that of the peritoneum, the name was changed to metro-peritonitis; metritis was the next appellation; then uterine phlebitis, pus having been found in the veins of the uterus; as this was sometimes found only in the lymphatics, it was hence called lymphangitis; others, especially the Germans (with Boër at their head,) discarded all these as false, and substituted putrescence or softening of the uterus; this again did not satisfy the French pathologists, who,

⁸⁴ Thomas, *The Modern Practice of Physic*, states on page 934, “Under different circumstances, the disease assumes different appearances, and accordingly different distinctions have been laid down by writers between its various forms; but such distinctions are of no use in practice, and may, perhaps, be productive of embarrassment to the practitioner. We may conclude, I think, that the only essential difference in the cases that ought to be considered puerperal fever, consists in their degree of violence, and their being epidemic, or simply sporadic; for it seems to be admitted that whenever the disease exists epidemically, it is more urgent in all its symptoms.”

⁸⁵ Meigs, *Obstetrics: The Science and the Art*, states on page 618, “the disorder here to be treated of is observed only in pregnant and lying-in women; yet it is not one, but many...It is inflammation of the womb alone; too it is inflammation of the veins of the womb; or it is inflammation of the peritoneum; or it is metro-phlebitis, or metro-peritonitis; or else a combination of metro-peritonitis with phlebitis. These are the several forms of the disease.”

⁸⁶ Hallett, “The Attempt to Understand Puerperal Fever,” 10.

having met with pus in the cellular tissue, in the viscera, and the articulations, without lesion of the uterus or peritoneum, came to the conclusion that it was a purulent fever, which is the prevailing opinion in the present French school. *Thus, by attempting to localize and simplify, this system has only rendered still more confused and difficult this important subject.* Each observer has described faithfully his epidemic; each has been so far in the right: but all have been wrong in maintaining that the type of disease observed by them was the true type of puerperal fever, to the exclusion of all others.⁸⁷ (italics added for emphasis)

In this way, pathology acted as a hindrance to developing prevention practice for there was very little prevention that could occur if the disease was so ill-defined that it was not certain if it was a single disease at all. For if it was not it might be prevented in many different ways, again according to the specifics of each case. Describing the contradictory findings of autopsies in puerperal fever cases, Gail Pat Parson stated, “The Practitioner of 1830 had gained one advantage over earlier colleagues—science had legitimized his confusion about puerperal fever.”⁸⁸

This confusion can be further understood by remembering that the very definition of puerperal fever was based on an ancient understanding of disease as that affecting a certain population and was classifiable by a recognizable collection of symptoms. In this case, puerperal fever was defined as a fever that took place during the puerperium – the period between childbirth and the return of the uterus to its normal size. The fairly regular symptoms of a bacterial infection which turned septic in the population of women having given birth are what caused puerperal fever to be classified as a separate disease from other illnesses that might be caused by the same bacteria.

Diseases are classified today by their causative pathogen because in our current bacteriological paradigm, treatment for disease is geared toward killing or inhibiting the

⁸⁷ Samuel Kneeland, “On the Contagiousness of Puerperal Fever,” 45-46.

⁸⁸ Parsons, “British Medical Profession and Contagion Theory,” 139.

growth of that particular pathogen in the affected body. Before it was understood that germs played a vital role in disease, the disease causation models of the modern period continued to classify disease by population and symptomology because the available treatment was of ancient origin where the classification of symptoms and the predispositional factors of the population infected were what determined the correct treatment course.

Those doctors studying puerperal fever in the eighteenth and early nineteenth centuries could not have known about bacteria. And as puerperal fever can be caused by many different microorganisms, it can have very different disease courses with varying lengths of illness, symptoms, and outcomes. Some cases are more deadly than others. So puerperal fever was confusing because it was a collection of many different infections, but with the same ultimate cause: an introduction of bacteria into the birth canal of the mother. Therefore, prevention would have included making it more difficult for these bacteria to be thus introduced. Unfortunately, prior to the development of the germ theory, the pathological findings of the dissecting room acted more as a distraction than an arena of discovery and little achievement was made in overall prevention.⁸⁹

⁸⁹ Meigs, *Obstetrics: The Science and the Art*, states on page 615, “Diseases that are clearly understood, and methods that are proved to be salutary and successful, unite all voices in the proposition as to their nature and use; but, in our disorder, the utmost latitude seems to have been given to the imagination, so that a complete distraction of the professional mind appears, in the command of this one to regard it as a fever— of that one, as an inflammation—of another, as synocha or typhus; to bleed—not to dare to bleed—to salivate—to rely on opium—on ipecacuanha, on turpentine—on purgative drugs, on saline draughts; and so, of every possibly suggestion of treatment, until the Student, confused and baffled at last in his search after some sure foundation to rest upon, gives up the search for truth in despair, and resolves to wait until the conflict arises, and then to do as best he may.”

Contagion and the Environment: Definitions and Confusion

In the nineteenth century, the argument over disease causation shifted from inflammation theory vs. putrid theory to one of contagion vs. anticontagion. Actually, this debate was split many ways, though most historians of puerperal fever have ignored the subtle shifts in terminology which make this issue so perplexing to the careful reader of these primary sources. Most histories on puerperal fever have been concerned with what kept the contagionist camp from convincing the medical profession of the contagiousness of puerperal fever. They rarely discuss the anticontagionists, other than to quote their more extreme assertions so as to support their contemporary marginalization.⁹⁰ The real debate, as it took place in the nineteenth-century writings on puerperal fever, was not simply among contagionists and anticontagionists.

Intent on more clearly describing the various groups of contagionist and anticontagionist doctors, with each group further separated into environmental and iatrogenic factions, Gail Pat Parsons describes the oft-overlooked, yet fundamental differences between these viewpoints in her article on puerperal fever in antebellum

⁹⁰ Wertz and Wertz, *Lying-In: A History of Childbirth in America*, 122. For example, Charles Delucena Meigs, who is often vilified in the secondary literature because of his staunch anticontagionist philosophy is repeatedly quoted in revisionist literature as saying, "Doctors are gentlemen and gentlemen's hands are clean." This statement has been characterized as an extremely arrogant way of dismissing the need for hand washing and other cleanliness procedures. However, if one reads Meigs' works or those written by physicians who knew him well, he was a staunch believer in cleanliness to the point that his contemporaries remarked on his fastidiousness, "at all times ready to enter either the sick chamber or the drawing room," as quoted in Parsons. His words, taken in appropriate context then, read closer to, "Doctors are gentlemen and gentlemen's hands [should always be] clean."

America.⁹¹ She presents a categorization of the doctors' theories as to the spread of puerperal fever by describing four different positions: iatrogenic contagionists, environmental contagionists, iatrogenic anticontagionists, and environmental anticontagionists.

Basically, diseases are caused iatrogenically when they are initiated by a health care worker or by some medical treatment. All nosocomial infections, or infections acquired in a hospital, are iatrogenically produced. Surgical wound infections are a recurrent example. Therefore, iatrogenic contagionists and iatrogenic anticontagionists believed that the medical practitioner was likely to blame for spreading the disease to his or her patients. Environmental contagionists and anticontagionists did not believe that the doctor or midwife spread puerperal fever, but that the environment, be it the home, hospital, or town, was to blame. Ultimately, both of these viewpoints were directly related to miasmatic theory in that the putrid effluvium which acted as the instigating agent was what caused the disease, irrespective of whether the mother had been contaminated with the effluvium by the medical staff or by the environment itself.

The distinction between contagionists and anticontagionists in the nineteenth century, according to Parsons, centers on the definition of "contagion." Today, the definitions of contagion and infection, or contagious and infectious, are much the same. But in the early nineteenth century, Parsons argues, these medical terms were separate and distinct. She quotes William Dewees, professor of midwifery at the University of Pennsylvania, as writing in 1826, "By contagion is understood effluvia (miasmata) arising directly or indirectly from the human body under particular diseases, and capable

⁹¹ Parsons, "Puerperal Fever, Anticontagionists, and Miasmatic Infection."

of exciting the same disease in other persons to whom it may be applied.”⁹² For a definition of infection, she turns to Noah Webster in his *History of Epidemic and Pestilential Diseases*, published in 1799, which reports that the term was used to indicate “that quality of a disease which may or may not excite it in a sound body within a suitable distance, or by contact; and which depends on heat, fouled air [and] an apt disposition in the receiving body.”⁹³

Therefore, the main criteria for diseases being contagious were that they regularly replicated themselves within the environment and conferred upon their victims the exact disease as that which created the transmitting agent, the putrid effluvia. The disease most exemplifying this theory was smallpox. An infection, by contrast, was a general state of unhealthy air which could render a subpopulation with certain predisposing factors susceptible to a variety of illnesses. As medical theory evolved, the definitions of contagion and infection merged to become synonymous under germ theory.

Readers of primary source documents who do not understand the evolution of these theories of disease causation often mistakenly argue that the use of these terms in the pre-bacteriological era indicates an early understanding of the role of germs in producing disease.⁹⁴ For example, contemporary medical historians of puerperal fever often fail to realize that doctors they consider “heroes,” such as Ignaz Semmelweis, would have considered themselves to have been anticontagionists alongside so-called “villains” such as Charles Delucena Meigs. Semmelweis would have fallen into the

⁹² Ibid., 431 ; William P. Dewees, *A Treatise on the Diseases of Females* (Philadelphia: Blanchard and Lea, 1854), 384n.

⁹³ Parsons, “Puerperal Fever, Anticontagionists, and Miasmatic Infection,” 433. Noah Webster, *A Brief History of Epidemic and Pestilential Diseases*, Vol 2 (Hartford: Hudson & Goodwin, 1799), 141.

⁹⁴ Vivian Nutton, “The Reception of Fracastoro’s Theory of Contagion: The Seed That Fell Among Thorns?” *Osiris* 6 (1990): 196-234.

iatrogenic anticontagionist camp while Meigs was a staunch environmental anticontagionist who resentfully condemned the branding of medical professionals, by those who argued for an iatrogenic source, as “murderers.”

Gail Pat Parsons is quick to point out that the observations of the environmental anticontagionists supported their beliefs in an environmental cause of the disease.⁹⁵ These doctors described cases that were sporadic with no obvious tie to a physician’s other patients. And while many physicians may have come across a succession of patients that became infected after he delivered them, a fact frequently cited by those arguing for an iatrogenic link in transmission, the overall landscape of puerperal fever cases were so random that an environmental cause seemed the only explanation that could account for all scenarios.⁹⁶

Environmental anticontagionists were not simply ignoring the facts or attempting to save their own reputations. Their position was simply a testament to the widespread assumption of the plausibility of a miasma. They were a significant collection of highly educated, well-respected doctors who, far from refusing to cooperate, were in fact using well-established miasmatic principles in their attempts to prevent puerperal fever. State-of-the-art hospitals in the nineteenth century were built in a locale, perhaps on a hill facing the wind, which would be less likely to imprison miasmas. Proper ventilation and

⁹⁵ Parsons, “Puerperal Fever, Anticontagionists, and Miasmatic Infection,” 440.

⁹⁶ Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*, states on page 118, “This explains why the conflict over whether childbed fever is or is not contagious could never be conclusively resolved. Those who believe in contagion cite cases in which childbed fever had undeniably spread from an ill patient to a healthy one. Their opponents cite cases in which the disease did not spread as it would have done if it had been contagious. Childbed fever is not a contagious disease, but it can be conveyed from diseased to healthy patients by decaying animal-organic matter...” Kneeland, “On the Contagiousness of Puerperal Fever,” states on page 49, “For those who consider as epidemic disease those propagated by a morbid atmospheric principle, it will always be easy to make objections to the contagionist, unless the proofs are so convincing as to silence all opposition; which is rarely the case.”

treatment of bedding would ensure that puerperal fever victims' putrid effluvia did not contact healthy patients. Evacuation from the vicinity might be ordered as a last resort in an epidemic while the hospital was scrubbed and whitewashed in an attempt to rid the environment of the infectious miasmas. These last precautions often lowered the frequency of puerperal fever for a time, a fact that reinforced the belief that the environment was to blame.⁹⁷

Other important observations about puerperal fever were used to argue against its contagiousness. In fact, the link made by doctors such as Alexander Gordon between erysipelas and puerperal fever, which is lauded by many contemporary historians as an early clue to the contagiousness of puerperal fever, actually led doctors to conclude that puerperal fever was a miasmatic infection rather than a contagion. Under the definition of the term, as described by Parsons, a contagious disease could only have been caused by another incidence of that same disease. Therefore, those doctors who claimed that cases of puerperal fever followed cases of erysipelas were forced to conclude that puerperal fever was not a contagious disease but a byproduct of an infected environment that was causing both diseases.⁹⁸

Loudon's *The Tragedy of Childbed Fever* casually mentions Parsons and her contention that these terms were separate and distinct. But Loudon takes the position that she has overstated the degree to which these doctors employed such distinctions. He states, "Certainly both words were used, but careful distinctions between infections and

⁹⁷ Ibid., 429.

⁹⁸ Ibid.

contagions were, I believe, rarely important.”⁹⁹ However, based on a careful reading of the primary sources, his dismissal seems premature.

It is true that, no matter how hard one tries, the primary sources do not fit neatly into the four-category system that Parsons proposes. However, she introduces a distinction in terms that is by no means “rarely important.” The ambiguities and frequent outright confusions over these important key terms contained in the primary sources often escapes notice. Without such careful distinctions in the analysis of terminology, the various positions attributed to the physician’s attempts to understand puerperal fever can become hopelessly muddled.

For example, there are those, such as Alexander Gordon and Oliver Wendell Holmes, who write of puerperal fever using the terms “contagion” and “infection” as almost synonymous, much as we do today. Gordon states, “That the cause of this disease was a specific contagion, or infection, I have unquestionable proof.”¹⁰⁰ One may deduce that Gordon is simply unsure of the specific nature of the disease being spread, particularly since he argues that puerperal fever and erysipelas were concomitant epidemics, a fact that would place the disease in Parsons’ category of infectious and therefore not contagious. However, he then proclaims, “In short, I had evident proofs of its infectious nature, and that the infection was as readily communicated as that of the smallpox, or measles...”¹⁰¹ According to Parsons, smallpox was the contagious disease *par excellence*, a standard against which all doctor’s judged the contagious quality of other diseases. Gordon’s use of the metaphor of smallpox to argue for the infectious

⁹⁹ Loudon, *The Tragedy of Childbed Fever*, 79-80.

¹⁰⁰ Gordon, *A Treatise on the Epidemic Puerperal Fever of Aberdeen*, 62.

¹⁰¹ *Ibid.*, 63.

nature of the disease seems to contradict her argument. However, in a following statement he states that, "...every person, who had been with a patient in the Puerperal Fever, became charged with an atmosphere of infection, which was communicated to every pregnant woman, who happened to come within its sphere."¹⁰² This phrase, "atmosphere of infection," is commonly found in the arguments of those who do fit clearly into the iatrogenic anticontagionist category and so we are again left wondering as to his meaning of the term "infection".

He further states,

These facts fully prove, that the cause of the puerperal fever, of which I treat, was a special contagion or infection, altogether unconnected with a noxious constitution of the atmosphere... That the infection, which produces the puerperal fever is not a specific contagion, but of the same nature with synochus or typhus, has been asserted by a late writer on the Puerperal Fever. This author says, "that the disorder is not one, *sui generis*, confined to in-lying women, but merely an unusual form of a very common disease, and is in reality no other than the common infectious fever, complicated with a more or less extensive inflammation of the peritoneum." ... The cause of both is undoubtedly infection, but the two infections are of a very different nature. For the circumstance, which excites the infection of the puerperal fever, seems to prevent typhus.¹⁰³

Again, this statement seems to place him firmly in the anticontagionist camp. Yet he continues to use the terms "contagion" and "infection" in such ways that cause confusion if the terms are to have different meanings. He states, "I have had an opportunity of attending a great number of cases of puerperal fever, arising from various causes besides contagion; for I have seen it produced by cold, by fear, by errors in diet, by too early fatigue, and premature endeavours to appear well, by the application of putrid matter to the uterus, &c."¹⁰⁴ And so the reader is left ultimately wondering if

¹⁰² Ibid., 64.

¹⁰³ Ibid., 67-68.

¹⁰⁴ Ibid., 118-119.

Gordon espouses a view of contagion and infection being fundamentally different, if he is simply unsure as to which term is best appropriate in describing this disease, or if he considers the disease to be caused by a multitude of factors ultimately encompassing both terms, whether distinctly defined or not.

Meigs, admittedly a much clearer case, is a fierce supporter of environmental causation who argues against contagion and who rarely uses the term “infection” in his writings on puerperal fever, states that,

As to the contagiousness of small pox, I cannot deny it, *since I cannot deny the contagiousness of any inoculable malady*. But the contagious nature of puerperal fever, though asserted by so many of the brethren,...,I cannot for a moment admit. Its epidemic power is for me a sufficient explanation of all the asserted examples of its communication by direct contagion...*If a disease be contagious, it must be so by virtue of a material, or essence produced in and evolved from the person of an individual;*...¹⁰⁵ (italics added for emphasis)

And so he agrees that smallpox is the standard under which contagion can be understood, however he does so because he can readily observe the “inoculable” matter which is transmitted that causes the contagion to be passed from one person to another. He not only argues that puerperal fever has no contagious essence that he can readily imagine, but he concludes that the very definition of the disease prohibits one of thinking of contagion. He states,

A woman who is to be attacked with puerperal fever is a woman in health,...though she may have a proneness to fall ill with what you call child-bed fever. If you fire a bullet through her womb, or tear it, or contuse it with a pair of forceps or a sharp crochet, and she die of the inflammation resulting from the accident, she will die of puerperal fever...if, from whatsoever cause, the blood vessel system of the uterus becomes the seat of a local inflammation, that inflammation will determine in her the onset of child-bed fever...¹⁰⁶

¹⁰⁵ Meigs, *Woman; Her Diseases and Remedies: A Series of Letters to His Class* (Philadelphia: Lea and Blanchard, 1851), 604.

¹⁰⁶ *Ibid.*, 603.

And so, if one agreed that puerperal fever was a type of peritoneal or uterine inflammation, then a discussion of puerperal fever being contagious was not only inappropriate, but irrelevant. Puerperal fever, for Meigs, was defined by that inflammation, no matter what conditions helped to produce it. Inflammation, as a bodily process, could not be contagious.

In an opposite vein, Semmelweis argued for the non-contagious character of the disease *because* there was inoculable material that caused puerperal fever. Semmelweis states,

Childbed fever is not a contagious disease. *A contagious disease is one that produces the contagion by which the disease is spread.* This contagion brings about only the same disease in other individuals. Smallpox is a contagious disease because smallpox generates the contagion that causes smallpox in others. Smallpox causes only smallpox and no other disease...Childbed fever is different. This fever can be caused in healthy patients through other diseases...*However, childbed fever cannot be transmitted to a healthy maternity patient unless decaying animal-organic matter is conveyed...* For example, suppose a patient is seriously ill with a form of childbed fever in which no decaying matter is produced. Then the disease cannot be transmitted to healthy patients. On the other hand, if the patient with childbed fever has septic endometritis or discharging metastases, then her disease can be conveyed to healthy patients.¹⁰⁷ (italics added for emphasis)

Here, Semmelweis is arguing that puerperal fever is not contagious because the term “contagion” for him means the same that it does for Parsons: that a contagious disease creates effluvium that can only cause that particular disease in others. Because puerperal fever, in Semmelweis’ opinion, is caused by any decaying animal-organic matter, such as that introduced into the vagina on the hands of a doctor after he assisted in a postmortem dissection, that matter constitutes an inoculable poison. And because many

¹⁰⁷ Ignaz Semmelweis, *The Etiology, Concept, and Prophylaxis of Childbed Fever*, 117.

diseases or injuries result in decaying matter, a number of diseases can cause puerperal fever.

This leads the reader to further question sources that describe the link between puerperal fever and erysipelas because Semmelweis argues that,

Childbed fever bears the same relation to erysipelas and its sequelae that it does to every other disease that generates decaying matter...In recognizing only erysipelas and its sequelae, beyond puerperal fever itself, as sources of childbed fever, English physicians draw their boundaries much too narrowly...Thus childbed fever is the same disease that occurs among surgeons and anatomists, and following surgical operations, it is the same disease whether decaying matter is brought into the circulation system of males or of females.¹⁰⁸

Therefore, Semmelweis cites cases of inoculation with the decaying matter of erysipelatos abscesses as causing puerperal fever. He does so while arguing that puerperal fever is not contagious. However, Storrs cites examples of erysipelas leading to cases of puerperal fever, and vice versa, as evidence that puerperal fever *is* contagious, a fact that further discredits Parson's view that links between cases of erysipelas and puerperal fever necessarily argue for its infectiousness.

And so it goes for the vast majority of primary sources on puerperal fever. Some sources set out a clear distinction between the terms "contagion" and "infection." Some do not and seem to use these terms interchangeably. Some argue that the causes of the disease are unknown and that a discussion of contagiousness is premature. And some deal with the apparent contradiction in data by separating cases of the disease into different forms of puerperal fever: some contagious, some infectious, and some produced by direct inoculation.

¹⁰⁸ Ibid.

Therefore, while Parsons' categories are not as useful as one might hope for sifting through the apparent contradictions between and within the treatises on puerperal fever, Loudon's assertion that the distinction between terms was "rarely important" is a gross underestimation of the ambiguity with which these terms were used.

On the contrary, such differences in definitions are seldom trivial. The definitions of the central terms in any advanced explanation are always theory-laden, in that they make implicit claims about the natural world. Such theoretical definitions contain within them the assumptions of the theory. How one defines the key features of a given disease impacts basic assumptions about that disease and diseases in general. No two physicians could accurately communicate, much less successfully craft an explanation of a disease, if their definitions of the key features of that disease contained significant discrepancies. If one physician defined a miasma as an unobservable noxious spirit while another claimed that it was composed of fine, wind-blown particles, clearly their "agreement" that the disease was caused by a miasma does not mean they endorse the same theory.

Worse, we find these physicians, sometimes very good physicians, being rather cavalier about consistently using the same meanings for key terms in their own writings. This inconsistency appears both in labeling the causative agents of the disease and in the symptoms that they believed indicated the presence of the disease. Either way, such internal inconsistencies crippled any attempt at forming a coherent explanation of the disease. In part, this inconsistency has been a product of their refusal to drop bleeding as a treatment. Small *ad hoc* adjustments to a theory may have seemed trivial or gone unnoticed for a long time, but hundreds of these small adjustments over decades or

centuries created a theory that looked nothing like its predecessor. In this way, a theory can become so convoluted, so unable to sustain itself, that it becomes unworkable.

Finally, the unfortunately common practice of citing long strings of agents they believed produced puerperal fever, most of which were ill-defined, fatally complicated any hope of clear theory formation.

Success: Treatments Break Free from Galen

Even after the acceptance of germ theory in the lying-in hospitals of Europe in the second half of the nineteenth century, little progress was reflected in the statistics of maternal deaths in Great Britain. Despite the fact that Listerian antiseptic procedures were finally made mandatory in British hospitals, ninety-five percent of deliveries still took place at home attended by a general practitioner or midwife.¹⁰⁹ While the prevention of puerperal fever was finally vindicated as hospital cross-infection dropped close to zero, the overall death rate from puerperal fever actually rose in Great Britain. It was not until after World War II that British maternity hospitals became the site of the majority of middle-class deliveries. Helen Roberts points out that since Great Britain began keeping records of maternal deaths in 1835, these statistics remained at a constant high until 1935.¹¹⁰

It was in the mid 1930s that sulphonamide drugs were first used to treat puerperal fever. Suddenly, the outcomes of home-birth infections improved considerably to meet those established around the turn of the century in the closely monitored environment of the lying-in hospitals. Only after the introduction of this dramatically successful

¹⁰⁹ Loudon, *The Tragedy of Childbed Fever*, 159-60. Despite antiseptic procedures being introduced into the hospitals on the Continent as early as 1868, neither British nor American lying-in hospitals standardized Listerian procedures until the 1880s. These procedures included sulphurous or carbolic acid sprays used to disinfect the clothing of all birth attendants and solutions of carbolic acid or chloride of lime to wash the hands and to sterilize any metal instruments. Some hospitals went so far as to inject carbolic acid directly into the vagina of their patients and/or to cover the vulva in a carbolized oil or a mixture of salicylic acid in wheat flour. Whenever possible, the rooms were left vacant and fumigated in between patients and the bedding was burned when patients exhibited the symptoms of puerperal fever so as to lessen the chance of an epidemic spreading.

¹¹⁰ Helen Roberts, *Women, Health, and Reproduction* (London: Routledge & Kegan Paul, 1981).

treatment did the maternal death rate in Great Britain decline to meet that of other countries on the continent whose culture of maternal delivery was centered in the hospital. Listerian antiseptic procedures had proven an effective prevention against nosocomial infection, but in Great Britain, a program of prevention was not an effective treatment because the culture relied almost exclusively on home birth facilitated by general practitioners who had many objections to the use of Listerian practices. These objections ranged from the expense and the difficulty of controlling the environment in the patient's home to older, traditional beliefs in spontaneous infection and the inherent differences between cases of puerperal fever.¹¹¹

Until a more successful treatment was discovered, no change in models of disease causation would change the behavior of the majority of British physicians. While the main treatment for puerperal fever, bloodletting, remained fundamentally humoral in origin, all models for explaining and understanding the disease would be made to justify that treatment. Even after the introduction of germ theory was shown, in British lying-in hospitals and elsewhere in Europe, to significantly change the outcome of these puerperal infections, prevention and treatment for the majority of British mothers continued to be influenced by old ideology. For puerperal fever, the treatment always dictated the philosophy.

Today, puerperal fever is almost unheard of. The widespread availability of antenatal care, chemotherapy, antibiotics, and blood transfusions has made puerperal fever, and many other complications of childbirth, a thing of the past in most

¹¹¹ Loudon, *The Tragedy of Childbed Fever*, 164-5.

industrialized nations.¹¹² However, it has not been completely eradicated. In the United Kingdom, between the years 2003 and 2005, the number of maternal deaths directly attributed to genital tract sepsis was 18 in 100,000 deliveries.¹¹³ This number includes abortion with sepsis, which is the leading cause of current infections in Britain. Another significant risk factor for infection is Caesarean section.

Most women giving birth in hospitals or in midwifery birthing centers today receive regular screening for bacterial infection and the majority of hospitals give routine antibiotics for streptococcus. Most of these same women have no idea that these precautions are to guard against puerperal fever. A program of prevention has been the first line of defense against puerperal infection and strong antibiotics are given when infection is even remotely suspected. However, agencies that work to promote better outcomes of pregnancy and birth, for both mother and child, stress that medical staff must be trained to recognize the signs of puerperal and genital tract sepsis as many cases turn fatal because of misdiagnosis or an underestimation of just how serious these infections can be.

¹¹² Roberts, *Women, Health, and Reproduction*, 21.

¹¹³ Confidential Enquiry into Maternal Death, "Saving Mothers' Lives: Reviewing Maternal Deaths to Make Motherhood Safer – 2003-2005" (Executive Summary and Key Recommendations prepared for the Confidential Enquiry into Maternal and Child Health, London, December, 2007).

Conclusion

The central puzzle of puerperal fever in Britain in the eighteenth and nineteenth centuries is thus one of reasoning and assumptions. Neither an absence of data, personal shortcomings, nor a lack of dedication account for the inability of two centuries of British doctors to find a way to prevent this killer. It was their commitment to outdated, ancient Greek assumptions about the human body, and their pre-scientific use of terminology and reasoning that blocked the way.

With so many women's lives at stake it is dismaying to see the number of modern practitioners and thinkers who resorted to subtle shifts in definitions, or appeals to near-mystical forces, or *ad hoc* explanations of their findings rather than change their clinical techniques. The temptation is great to join the many commentators who have castigated these physicians, attacking their motives, institutional politics, or personal foibles. To do this is both unfair and historically shortsighted.

The modern struggle against puerperal fever is an account of an intellectual quest wherein major concepts of the past began to crumble under the weight of new data and, faced with the impotence of their customary cures and preventions, people were challenged to look at the world in a fundamentally new way. To use Thomas Kuhn's language, the eighteenth-century explanation of disease was ripe for a paradigm shift. The battle against puerperal fever illustrates the difficulties involved in an especially complex example of one such shift.

The effort to understand nature is not just the accumulation of data nor the inventing of new explanations. In order for data and explanations to be cogent, they must fit into the intellectual fabric of the time. And the medical fabric of modern Britain was dominated by ancient concepts. To a contemporary scientist, this longevity alone would make such ideas suspect. For these physicians, unaccustomed to our frantic pace of monumental upheavals (true paradigm shifts such as natural selection, relativity, and quantum mechanics) the length of a medical doctrine's reign was an endorsement of its strength and veracity.

Whatever the need for a new paradigm, of course, physicians at the time could not see their place in history. Kuhn notes that most of what scientists do consists of gathering data and forming hypotheses that support the established paradigm. This was the role of the modern physician. The hope was always that they were engaged in an exhausting but ultimately fruitful quest for a cure. The anticipation was that a new discovery, a new technique, would perfect the device of bleeding or prevent miasmas, and puerperal fever would be conquered.

In retrospect, we see that this hope was futile. The preponderance of unobservable, at times almost ghostly, agents that were invoked as causes, the ambiguities in the definitions of key concepts, and the temptation to "fit" any novel data into the old theories, *ad hoc*, had created a complex of notions that lacked internal coherence and ultimately, could explain nothing. The classical explanations of Hippocrates and Galen were revered, and those *ad hoc* adjustments, the sometime subtle but often flagrant "repositioning" of one's explanations, ensured recent discoveries would always conform to classical explanations.

It is easy to forget that the scientific method is a relative newcomer on the stage of human knowledge. While a full paradigm shift, germ theory, was a century away from wide adoption, the central concepts that would break the hold of Hippocrates' and Galen's ideas were beginning to emerge, and several modern physicians gladly grasped these tools. Mechanism, fluid dynamics, and anatomy, were all building blocks of the new intellectual world and each would have a fundamental role in the new knowledge of medicine.

While the story of puerperal fever is essentially over, the battle against similar infectious diseases continues. Our current antibiotics are becoming progressively less effective and strains of streptococcus could undergo dramatic mutations at any time. Our challenge remains similar to that of these modern physicians. Flu pandemics, infectious prions, and ever-evolving bacteria are sure to demand greater feats of understanding if they are to be controlled. Despite decades of concentrated study we confront innumerable medical dead ends and some diseases that remain as deadly as ever. The history of the brave physicians who fought puerperal fever holds a lesson for our medical future. There is no signal that tells scientists that a new paradigm is needed. It will never be easy or obvious. But without the willingness to abandon established wisdom, scientific knowledge flounders and progress can be terribly prolonged.

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