Clinics of Surgery

Review Article

ISSN: 2638-1451 | Volume 11

Semmelweis and Lister: Restoring Balance to Their Historiographies

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Received: 12 Aug 2024
Accepted: 20 Sep 2024
Published: 26 Sep 2024
J Short Name: COS

Citation:

Kadar N. Semmelweis and Lister: Restoring Balance to Their Historiographies. Clin Surg. 2024; 11(1): 1-14

1. Abstract

This article compares the empirical bases and reasoning by which Semmelweis and Lister reached their conclusions about the cause and prophylaxis of childbed fever (CBF) and wound infections, and the historiographies of their work. It is shown that Semmelweis's conclusions about the nature and cause of CBF were valid deductive inferences based on his own empirical observations at autopsies, whereas Lister's theory of the cause of wound infections was a mere hunch based on the work of Pasteur. Lister's method of dressing wounds relied on carbolic acid, and later on other antiseptics, to eliminate germs from contaminated wounds and to prevent germs from entering wounds while they were healing, and he opposed aseptic methods such as heat sterilization and the wearing of surgical gloves. Semmelweis's prophylaxis involved disinfection of the hands of attendants and the instruments they used with a chlorine solution. Semmelweis conducted concurrent and historical comparisons and animal experiments to prove his theory and the value of his prophylaxis, Lister did not conduct animal experiments to prove his theory and resisted calls to publish the results of his method of dressing wounds after publishing a preliminary report. Five factors are identified that help explain why Lister emjoyed such during his lifetime whereas Semmelweis's theory met with opposition.

2. Introduction

Full length statues of Ignaz Philipp Semmelweis (1818-1865) and Joseph Baron Lister (1827-1912) stand next to each other among ten others in the Hall of Immortals of Chicago's Lakefront Museum of Surgical Science commemorating the greatest physicians who have ever lived. One may, therefore, be forgiven for thinking that history has accorded in full measure the credit due each man for his contribution to medicine, but nothing could be further from the truth. Each man's theory at first met fierce opposition from their contemporaries, but over a 5-15 year period Lister's antiseptic treatment of wounds was gradually accepted-first in Germany, then the rest of Europe and the United States, and much later in Britain-and Lister acquired unprecedented fame and prestige at home and abroad during his long life, and when he died at the age of 85 Lister had had his country's highest civic honors bestowed upon him: first a knighthood, then a peerage, and finally a baronetcy. By contrast, Semmelweis died at the age of 47 beaten within an inch of his life by his attendants in the newly built Lower Austrian State insane asylum (Niederösterrichische Landesirrenanstalt) (a fact the Austrian authorities concealed for a hundred years, (Carter, Abbot & Siebach, 256), and his doctrine of antisepsis remained controversial and rejected by many leading obstetricians during his lifetime. Semmelweis was only given the universal recognition he deserved posthumously, and even then only to have the credit due to him contested in the German and Anglophone literature. Thus, what Fritsch [1-3] wrote in 1884 was apt: In the history of midwifery there is a dark page, and it is headed 'Semmelweis'. . . If the conclusions and counsels of Semmelweis had been followed. Obstetrics would have stood in the forefront of the greatest advance in Medicine which has been made since physicians and physic came into existence. This article examines the reasons for the very different reception accorded the theories of these men by their contemporaries and posthumous critiques: specifically, whether it is attributable to the correctness of their theories, the strength of the proofs they offered for them or to other factors. This examination is overdue because, as this article demonstrates, the originality of their discoveries and the rigor of their proofs are inversely proportional to the credit Semmelweis and Lister received

during their lifetimes, and in the historiographies of their discoveries. This conclusion is supported by the distinguished gynecologist, Alfred Hegar (35), Semmelweis's first biographer, who wrote that Lister "received the impetus and theoretical foundation of his doctrine from someone else, Pasteur, and is much less original than Semmelweis, who deduced everything himself." This conclusion is also supported by Lister's own rejection of his original theory that the source of the bacteria that cause wound infections is the air, and the abandonment of the attempt to avoid wound infection by the placement of antiseptic agents in the wound, which not only irritated the tissues but impaired the local immune response to infection. In his Presidential Address to the British Association for the Advancement of Science at Liverpool, Lister (1896, 736) told his audience, at the Berlin Congress in 1890, I was able to bring forward what was, I believe, absolute proof of the harmlessness of the atmospheric dust in surgical operations. This conclusion has been justified by subsequent experience; the irritation of the wound by antiseptic irrigation and washing may now be avoided and Nature left quite undisturbed to carry out her best methods of repair. This admission caused the distinguished surgeon and surgical historian, Owen Wangensteen, to ask, "Was this not the basic principle of Semmelweis's teaching and practices?" and to draw the "inescapable" conclusion that in his final professional years and in retirement. Lister gained an acquaintance with the teachings and practices of Semmelweis that he had neglected at the inception of his own life's mission. How different Lister's antiseptic wound practices might have been had he become fully alert at the outset of his important work to the significance of Semmelweis's ideas of the prevention of wound infection. (Wangensteen & Wangensteen, 1974, 124.). Perusal of the philosophy of science literature provides a further clue to the rigor of the reasoning these men used to arrive at and prove their theories. Philosophers of science have relied extensively on case histories to develop theories of scientific methodology and explanation, and to try to understand how science in the past worked [4,5]. They have used Semmelweis's work so frequently "to illustrate, appraise and compare methodological proposals" that Scholl concluded that it was "no exaggeration to say that the Semmelweis case has become a paradigm of scientific discovery and confirmation within the philosophy of science." (Scholl 2013, 67). By contrast, one would be hard pressed to find any reference at all to Lister and his work in the philosophy of science literature. This is hardly surprising for as this essay will show Lister did not believe in statistics and offered little if any proof of the superiority of his method of dressing wounds to other methods, never conducted animal experiments to prove his theory, and never identified any of the bacteria that he contended originated in the air and caused wound infections.

3. Summary and Critique of the Historiographies of Lister and Semmelweis

The historiography of Lister and his work consists of unapologetic hagiographies that describe Lister not only as the founder of modern surgery and the 'antiseptic principle', but as one of the greatest geniuses and benefactor of mankind who has ever lived. "No surgeon in history," Wangensteen & Wangensteen (101) observed "has won so many plaudits of acclamation and hosannas of praise." For example, Lister's last assistant described Lister as "an almost unearthly superhuman being", and wrote that when Lister died, "it was universally acknowledged that the King had lost his most distinguished subject and the world its greatest inhabitant [6]. The hagiographies began a year after Lister's death when the first of the many biographies about his life and work was published. (Wrench (1913). The extravagance of the panegyrics to Lister's genius, especially in the early biographies, is impossible to capture in a single essay, but reached their absurd apogee with Wrench's comments about Lister's physique. Wrench (35) claimed that the fishermen who had seen Lister swim "were wont to speak in terms of admiration of his excellently proportioned and graceful form," which caused Wrench to remark: "To me it seems that such perfection of physique is rarely, if ever, absent from great constructive genius." This historiography credited Lister with inventing modern surgery by "discovering" the cause of wound infections. Lister was, indeed, a prodigious and able experimenter, and when he formulated his 'antiseptic principle' and devised the first of the many iterations of his method of dressing wounds, he had conducted experiments on inflammation, coagulation of blood, and the muscles of the iris, and had been Regius Professor of Surgery at the University of Glasgow for five years. Nevertheless, he formulated his theory of the cause of wound infections only after his attention was drawn to Pasteur's work and not based on any original observations or experiments of his own. On learning of Pasteur's work, Lister jumped to the conclusion that 'germs' in the air were responsible for wound infections, as exposure to air was believed to be a sine qua non of wound infections (see below). This was, and could only have been, a mere "hunch" as it was based on "very little scientific evidence." (Poynter, 412). Germ theory as such—the idea that human diseases are caused by specific microorganisms-had not been shown to be true of a single human disease, much less wound infections, and Lister's hunch was incorrect in that bacteria in the air are mostly saprophytic and rarely cause wound infections, as Lister himself recognized twenty-five years later. (See Introduction) Moreover, Lister had an entirely erroneous concept of what bacteria were and believed that they developed from fungi until Pasteur corrected him in 1876 in the first correspondence between the two men. (Walker, 119). Wangensteen seems to have been alone in recognizing that Lister had in fact discovered nothing new: It has been said that Lister discovered antisepsis [and] that he introduced a new principle; Lister called it the antiseptic principle. Of course, Lister did neither. Pringle (1750) coined the word "antiseptic". Semmelweis (1947) recognized the mode of transmission of contagion and employed

a solution of chlorinated lime to prevent puerperal fever, disinfecting all material likely to come in contact with the parturient's vaginal tract. Auguste Nélaton (1852-1864) had used alcohol in major elective surgery with considerable success. (Wangensteen, 581) . Antiseptic management of the wound has disappeared. Of all the antiseptic agents the hypochlorites employed by Semmelweis survived the longest. (Ibid.591). Fisher (146), one of Lister's biographers, came to a similar conclusion: "Lister was a surgeon, not an inventor," he wrote. Semmelweis's antisepsis also anticipated Lister because it was not limited to obstetrics but applied with success to surgery (and gynecology) almost two decades before Lister developed his method of dressing wounds. Commenting on the evidence Semmelweis had offered to prove his theory of the cause of childbed fever (CBF), the internist, Carl Haller, who was acting head of the Vienna General Hospital (Allgemeines Krankenhaus –AKH) at the time, immediately recognized the potential importance of Semmelweis's prophylaxis to surgery, and wrote in his 1849 annual report on the AKH: And what is imperatively impressed upon the unprejudiced examiner of these figures, [referring to the reduction in mortality from childbed fever following the implementation of chlorine hand-disinfection at the end of May, 1847] has been established beyond all doubt by direct experiments on animals (injections of pus and ichor into the vaginas of newly delivered rabbits), which were done by Drs. Semmelweis and Lautner recently, and after complete analysis, will be published.

The significance of this practical knowledge for obstetrical institutions, for hospitals in general and especially for surgical wards is so immeasurable that it appears worthy of the most earnest consideration of all men of science, and is certain of suitable acknowledgement by the high state-government. (von György, 269, italics added).

Erna Lesky, the preeminent historian of the Vienna Medical School of the 19th century, also tells us that Heinrich Baron Dumreicher von Österreicher (1815-1880) adopted Semmelweis's principles of antisepsis on becoming head of AKH's first surgical clinic in 1849:

Dumreicher leached dressings in chlorine lime solution for days, and wound care on his clinic was so meticulously clean that in thirty years his clinic was visited only twice by the bugaboo of all surgical wards: hospital infections, a telling contrast to other surgical clinics where up to 80% of amputees died of hospital infections in the pre-antiseptic period. When in 1861, Semmelweis explained in his main work that "my doctrine is not forgotten at the school from where it originated", this was true, above all, of Dumreicher, who had applied it on the first surgical clinic with success. (Lesky, 1964, 86-87).

Thus it is understandable that Dumreicher did not condescend to apply Lister's process but in 1877 opposed it with his own occlusive method. He used a solution of zinc chloride 2%-8% to moisten the surface of the wound and then applied elastic compression in order to insure its proper effect. (Lesky, 1976, 175).

A year later (1850), Sir James Simpson, Professor of Midwifery at the University of Edinburgh, where Lister would be appointed Professor of Surgery in 1869, published an article with the title, "Some Notes on the Analogy between Puerperal Fever and Surgical Fever," in which he argued that "the combined febrile and inflammatory morbid state" from which surgical patients died was "generically, if not specifically, the same as puerperal fever in the childbed mother." (Simpson, 415) In that article, Simpson acknowledged Semmelweis's work and mentioned Semmelweis by name:

The mortality altered and diminished immensely and immediately from the time (May 1847) that the assistant-physician, Dr. Semmelweiss (sic), prevented students from touching parts of the autopsies, and directed all of them to wash their hands in a solution of chorine before and after every vaginal examination. (ibid., 429).

The changes in the management of wounds that Lister brought about was certainly a major landmark on the path to modern aseptic surgery, even if "Lister's work came as the culmination of several decades of hypothesis and experiment which were tending in the direction which he followed." (Poynter, 410) But Lister's method of dressing wounds did not make modern surgery possible. Modern surgery would not be possible without anesthesia, which Morton discovered when Lister was still a medical student. Nor would anyone perform surgery today as described in this account:

Lister took off his coat, rolled up his sleeves, and pinned an ordinary clean, unsterilized towel around himself. Then he dipped his hands in a basin filled with either a one-in-twenty solution of carbolic acid or else a mixture of carbolic acid and a one-in-five-hundred corrosive, sublimate solution. Towels wrung out in the one-in-twenty carbolic acid lotion were placed around the area to be operated upon. The instruments and sponges to be used in the operation had meanwhile been soaking in the same antiseptic . . . (Walker, 185)

Surgery would only be performed today using the aseptic techniques—sterile gowns, gloves and drapes, masks and caps—that Lister steadfastly opposed.

"Asepsis in this imperfect work is not to be trusted," Lister wrote, because "human carelessness and fallibility are common; it is safer to have an antiseptic." (Walker, 187). For example, surgical rubber gloves were patented in 1878, and first used in surgery by Halstead in 1890, but Lister disapproved of their use and wrote to his former dresser and house surgeon, Sir William Watson Cheyne, to reprove him for adopting sterile gloves for operations:

You must forgive me if I express my regret at what you say about the use of gloves because it may convey to some minds the idea that you distrust carbolic lotion for the disinfection of hands" (Fisher, 301)

Lister also expressed regret to Sir Hector Cameron, another erstwhile house surgeon and close friend, that Cheyne "advises sterilizing instruments by heat (boiling) without any reference to 1 to 20 (carbolic) solution." (Fisher, 301). However, as Koch pointed out, carbolic oil is "absolutely inert" and

when it is sought to disinfect dry objects, such as instruments, silk, catgut, etc., by means of carbolic oil, there is absolutely no effect, even upon the least resistant microorganism, beyond that due to the oil itself. (Cheyne, 1886, 504).

Indeed, Lister's neglect of sterilization by heat was one reason why surgeons in Britain did not follow Listerism. (Cope, 170-171: "Lister relied upon antiseptics alone for protection against the organisms; he neglected the other equally important method of killing microbes, that of heat".

Lister's opposition to aseptic surgery is difficult to reconcile with his realization that atmospheric dust was harmless to operations, but it was endorsed by his early biographers who derided aseptic surgery as a futile attempt by ordinary men to improve on the work of a genius:

It [aseptic surgery] is said to be an improvement and a step in progress, but when more ordinary men claim to improve upon the system and practice of the greatest genius surgery has ever known, one is warranted, perhaps, in being suspicious . . . I must confess that when progress follows upon the work of one of the world's great men, my anticipation is that it will be progress downhill. In this particular instance there can be little doubt that the progress in the treatment of wounds since the perfection of the antiseptic system, in elaboration, complexity, expense, and decrease safety, has been of a downward character. (Wrench, 313, italics added).

The practice of asepsis itself was at the same time satirized by Lister's first biographer as an attempt to render germ-free "all the little world in which the patient, surgeon, and dresser have their being":

After scrubbing his hands and arms with soap and water and a boiled brush, [the surgeon] is robed in boiled garments, pulled out of a boiled case by a nurse who is wearing boiled gloves. She also fits a boiled cap over his head, a boiled mask over his face, and a boiled bag over his mouth and chin. (Wrench, 316-317).

But it was the attempt to keep germs our of wounds and to render contaminated wounds germ-free with antiseptics that was futile. Micrococci were frequently found under Lister's dressings, and attempting to sterilize contaminated wound with antiseptics did more harm than good, as Lister (1896, 736) himself eventually realized. Wright and Fleming proved that carbolic acid was more likely to do more harm than good when applied to wounds in which infection had become established, (Fisher, 302), and also showed that most antiseptics lose their effect in the presence of blood and serum. (Cope, 171) Koch also concluded from his detailed study of antiseptics that carbolic acid could have no appreciable effect on spores "in the brief time occupied by a surgical operation", which caused him to comment that

It can no longer be a matter of surprise that in spite of the most scrupulous antiseptic precautions, bacteria are so often found under Listerian dressings. (Pennington, 38).

Thus, the idea that contaminated wounds could be rendered germ-free by the local application of antiseptics proved especially fatal, as it was responsible for many unnecessary deaths among British soldiers who had sustained gunshot wounds in the First World War. Cheyne's advocacy of this practice engendered what Cope (172) described as one of the most devastating critiques—by the distinguished bacteriologist, Sir Almroth Edward Wright ever to appear in print.

The historiography of Semmelweis and his work took a very different trajectory. The first biography of Semmelweis in English was published in 1909 by Sir William Sinclair, Professor of Obstetrics and Gynaecology at the University of Manchester. Sinclair was fluent in German and obtained his information about Semmelweis's life and work from the collected works of Semmelweis published by Tibor von György in 1905 in German, as well as from earlier biographies in German by Alfred Hegar (1882), Jacob Bruck (1887), Adolf Kussmaul (1899), and Fritz Schuller von Waldheim (1905). Sinclair's was a sympathetic, evidence-based biography in which he likened the beneficial effects of Semmelweis's discovery to Edward Jenner's discovery of the smallpox vaccine, as had Hebra, in his 1847 editorial:

In the whole history of medicine, we find a clear record of only two discoveries of the highest importance in producing direct and immediate blessings to the human race by the saving of life and the prevention of suffering. These were the discoveries of Edward Jenner and Ignaz Philipp Semmelweis . . . The discovery of Semmelweis was possible only for a man who had undergone prolonged and laborious preparation, who had directly observed, and had reflected without preconceptions, whose intellect was kept rather alert and keen because of the warmth of his human sympathy. (Sinclair, 1-2).

This conclusion was echoed by Wangesteen (1970, 356), who characterized Semmelweis's conclusions about the nature and cause of CBF as "a unique example of penetrating insight without parallel in the history of medicine". Hegar (30) was of a like mind and wrote of Semmelweis's theory of CBF: "Hardly ever has such a strangely composed and complex general idea been broken down more clearly and sharply into its constituent parts in medicine." However, after about a forty-year hiatus, the historiography of Semmelweis's work took a very different turn, first in the German and then in the English literature.

The German historiography was largely concerned with appropriating much of the credit for Semmelweis's discovery to Joseph Skoda and the Vienna Medical School, not with diminishing Semmelweis per se. It started in 1947, when the Hungarian-born phi-

losopher, Eric Podach (1894-1967), contended that the account of Semmelweis's discovery in Semmelweis's book (published in October, 1860, but for some reason the year is given as 1861) had to be an after-the-fact reconstruction because Semmelweis did not have sufficient autopsy experience to have made the anatomico-pathologic connections when he said he made them—a claim flatly contradicted by the available historical evidence (see below)—and that the true account of how Semmelweis made his discovery was provided by Skoda during his October 18, 1849 lecture to the Imperial Academy of Sciences on Semmelweis's work.

Building on Podach's work, Lesky contended that Semmelweis discovery was the intellectual property of the Vienna Medical School because Semmelweis derived "all the methodological means that were a prerequisite for his discovery," from that school, especially the 'method of exclusion' that Semmelweis learned from Skoda. Lesky contended that Semmelweis made his discovery using Skoda's method of exclusion notwithstanding that after excluding all the putative causes of childbed fever Semmelweis was able to conclude only what did not cause CBF and nothing at all about its actual cause (see below). But Lesky went further, and expressly declared Skoda to be the intellectual coauthor of Semmelweis's discovery:

Skoda, with the inner evidence of intellectual co-authorship [geistiger Miturheberschaft], immediately recognized in this new, genuine product of those methods of investigation and thought that Rokitansky and he had so distinctly imprinted on their school… [and] felt, to the highest degree, responsible to science and humanity, to ensure that this intellectual property of the Viennese school took its place in science and obstetrical practice."

The most influential accounts of Semmelweis's work in English were written by Irvine Loudon in Britain and Sherwin Nuland in the United States, but theirs are ersatz histories replete with factual errors and hermeneutic claims that have little or no support in historical evidence or are flatly contradicted by that evidence. Their objective seems to have been to diminish Semmelweis and the significance of his work by attacking Semmelweis's character and dismissing his influence on the practice of obstetrics.

Loudon (101) dismissed Sinclair's meticulously researched biography as "hero worship", accorded Semmelweis's work no historical significance whatsoever, and dismissed "most of the claims made about [Semmelweis] . . . as sheer nonsense." (Loudon, 2015, 462) With almost missionary zeal, Loudon spread the idea that Semmelweis had no influence on obstetrics, that his basic ideas were anticipated by Alexander Gordon and Oliver Wendell Holmes, and that antisepsis in obstetrics was based on the work of Lister, not Semmelweis:

During his lifetime and for many years after his death, Semmelweis had few supporters, and his work, which had very little effect on obstetric practice, was almost totally forgotten . . .antisepsis in obstetrics came not from the work of Semmelweis, but from the transfer of Lister's methods in surgery to the lying-in (maternity) hospitals. (Loudon, 2015, 462)

Loudon (2000, 145) made the same claim in his book on childbed fever: "Semmelweis had little influence either in his lifetime or for some twenty or more years after his death in 1865," he wrote. Loudon even accused Semmelweis of dishonesty in claiming that his views about the nature CBF differed from those of the British contagionists.

The available historical evidence, meticulously researched by the philosopher, Codell Carter, who is also fluent in German, flatly contradicts Loudon's hermeneutic claim, as does the first biography of Semmelweis's work by Hegar. Carter has shown that Semmelweis's theory of the cause and prophylaxis of CBF, although controversial, was widely discussed, and had a very favorable reception in Germany, the country in which Lister's antiseptic principle was first accepted and applied. The following are but some examples cited by Carter (1995, 90-91):

In 1861, shortly after Semmelweis's book was published in the fall of 1860, Wilhelm Lange, professor of obstetrics in Heidelberg, declared at a meeting of German physicians and scientists that his own experiences had persuaded him that Semmelweis's theory was correct.

In 1864, Joseph Späth, who was professor on the midwives' division of the AKH and had opposed Semmelweis's views, published an article in which he conceded, based on his own research, that "whatever anyone might say, every obstetrician now believed that Semmelweis was correct."

In 1868, Rudof H. Ferber wrote that Semmelweis had initiated a revolution in the understanding of childbed fever, and pointed out that "with only a few exceptions, the Semmelweis theory is now universally recognized in Germany."

Also in 1868, the German professor Max Boehr noted that Semmelweis's theory of the infectious origin of childbed fever

has the characteristics of every good pathological and physiological theory: it provides a unified, clear, and entirely intelligible meaning for a whole series of anatomical and clinical facts . . . None of the earlier or alternative theories or hypotheses regarding the occurrence of childbed fever has this characteristic to the same degree.

In 1876, Joseph Amann, professor of obstetrics in Munich observed that Semmelweis's theory had become shared property of the entire German medical profession.

In 1878, another German physician observed that thirty-one years had passed "since Semmelweis first spoke the truth that every case of childbed fever comes about through the resorption of decaying animal-organic matter."

Also in 1878, Otto Spiegelberg, professor of medicine in Breslau,

wrote that "Semmelweis deserves credit for placing the understanding of puerperal disease on the new and proper path," and that Semmelweis "explained that every case of puerperal fever is resorption fever arising from the reception of decaying animal matter . . . These claims hold today. In general they contain everything there is to be said about puerperal fever."

To this may be added that Hegar (1882, 35) wrote in his biography of Semmelweis that "the correctness of Semmelweis's theories [had been] completely established", and despite the fact that Semmelweis had received little recognition from his colleagues, his doctrine and teachings influenced obstetrical practice thanks to the efforts of Hirsch, Veit and Winckel:

It happened here just as it has happens in many other places. A discovery is belittled, and the discoverer himself mocked but his achievement is exploited, even by those who have belittled the discovery.

As for Nuland's derogatory accounts of Semmelweis and his work, they not only border on historical fiction but are shot through with a palpable antipathy to Semmelweis as evidenced by the following critique in which Nuland (123) excoriated Semmelweis for not conducting 'proper' animal experiments, and for not using the microscope to examine the lochia of post-partum women in attempting to prove his theory:

Bad enough that Semmelweis did not do proper substantiating experiments; bad enough that he never availed himself of the microscope's help that might have validated his theory in the minds of potential critics . . . Had Ignác Semmelweis so much as once asked the microscopist Joseph Hyrtl to study a drop of pus from one of the dead mothers, he would have found it to be teeming with the same kinds of organisms that Lister later found in his infected wound.

None of these criticisms is justified, and none has any support in historical evidence. For one thing, Semmelweis conducted two sets of animal experiments to validate his theory, the first of which showed that the injection of material from cadavers dying of a variety of disease into the uteruses of post-partum rabbits induced, with few exceptions, the same pathological changes as are observed in mothers dying of CBF. (Kadar, 2021) Lister, by contrast, conducted no animal experiments on wound infections at all, and it fell to Koch to conduct animal experiments in 1876 to prove that bacteria could cause wound infections.

For another thing, although Lister grew up, as it were, with the microscope, as his father had developed the achromatic lens,

Lister never tested the rationale of his theory experimentally. He never, of his own volition, examined pus under the microscope, although he used the microscope and was the son of a distinguished microscopist. (Lyell, 306)

Alexander Ogston did, however, test the theoretical basis of antiseptic surgery experimentally.

Ogston compared pus from acute abscesses with that from "cold" (i.e. tuberculous) abscesses. Pus from acute abscesses always contained micrococci . . . Pus from acute abscesses injected into guinea pigs and mice provoked pus formation; pus from "cold abscesses did not. Both heating and carbolic acid abolished the activity of pus from acute abscesses . . . Pure cultures of micrococci produced the same sequence of disease in guinea pigs and mice as "acute' pus had done. (Lyell, 308-309)

Although Lister mentioned Ogdon's work on the part played by micrococci in acute inflammation, he agreed with Cheyne that pus was merely a "congenial habitat for micrococci" and contended that micrococci are "a mere accident of these acute abscesses." (See below)

Moreover, had Semmelweis examined lochia microscopically, he would have discerned nothing at all as the microscope available to him on the obstetric clinic was not powerful enough to make organism clearly visible. This is evident from the fact that when Mayrhofer became assistant in Division I in 1960, and examined lochia of post-partum women microscopically more than a decade after Semmelweis left Vienna, he at first found nothing at all, and his professor had to purchase a more powerful microscope out of his own private funds before Mayrhofer could detect any bacteria. (Carter, 84-85)

III. Semmelweis and Lister: their theories, reasoning and proofs

The theories of Semmelweis and Lister concerned the cause and prevention of a disease—CBF in the case of Semmelweis, and wound infection in the case of Lister. Lister's theory was a hunch based on the empirical observations of others, and his reasoning consisted of analogical inferences. Semmelweis, by contrast, based his theory on his own empirical observations and deductive inferences that were valid by the tenets of formal logic. Moreover, whereas Lister's theory of the cause of wound infections was a mere extension of the extant view that the cause of wound infections was somehow related to air, Semmelweis's theory of the cause of CBF was a radical departure from the extant views about the causes of CBF.

A. Semmelweis

Semmelweis defined CBF, which was the leading cause of maternal mortality in his day, as a resorption fever caused by the absorption of 'decomposed animal-organic matter' from the lower genital tract into the blood stream of parturient women—"no single case excepted". (Semmelweis, 429) Carter (2003) has shown convincingly that Semmelweis was the first person to define a disease in terms of its universal necessary cause instead of clinical signs and symptoms and was the progenitor of the causal concepts of disease. Based on an extensive review of the literature Carter (1985, 45-53) also concluded that "investigations of childbed fever were much more prominent in the development of germ theory than one

might think, and that "Semmelweis's work contributed importantly to the theoretical development of germ theory."

The general view in Semmelweis's day was that CBF was a disease peculiar to pregnancy, especially the puerperium, caused by noxious properties of the air caused by atmospheric-cosmic-telluric influences and predisposed to by a number of factors such as overcrowding, the seasons, psychological factors, etc. But Semmelweis showed that the disease was spread not by the air but by the hands of attendants and the instruments they used and could be prevented by disinfecting the hands of attendants and their instruments with a chlorine solution. In most cases, the sources of the decomposed animal organic matter were external but in up to1% of cases the source was internal (called "auto-infections" by Semmelweis) derived from retention of blood clots, the placenta or placental and membranous remnants, in the uterine cavity, bruising of the genital organs from prolonged second state labor or as a result of necrosing perineal lacerations after operations. (Semmelweis, 552). Prophylaxis in these cases consisted of avoidance of prolonged labor and rotating and pendular motions during forceps deliveries, removal of placental remnants and blood clots. (Ibid., 557). How did Semmelweis reach these conclusions?

Semmelweis had "belonged to a group of students who gathered around Rokitansky and his assistant, the professor of forensic pathology, Joseph Kolletschka, in a particularly intimate circle." (Lesky, 1976, 141). Rokitansky had taught that the gross pathological changes observed at autopsies of individuals dying from various diseases were produced by disease processes and that an exact knowledge of these pathological changes was "indispensable for acquiring an insight into the nature of these processes." (Klemperer, 277) Semmelweis took this principle to heart, and after he was accepted as a trainee in obstetrics, spent most of the next two years performing autopsies in Rokitansky's morgue on all mothers who had died of CBF and on their babies, if they too had died, which was often the case, in order to gain insight into the nature of the process causing CBF. (Lesky, 1976, 185)

Semmelweis noted that the pathological changes at autopsy in newborns who died after birth, and who had been born to mothers who had died of CBF, were the same as those in their mothers, except for the internal genitalia, (Semmelweis, 392), and he reasoned that if the pathological findings were the same the diseases producing those pathological changes must also have been the same, otherwise it would render anatomic pathology meaningless. (Semmelweis, 381: "To recognize the changes in the bodies of the puerperae and not to recognize the identical results in the bodies of the newborn, invalidates pathological anatomy.").

Semmelweis (381) also assumed that if the diseases were the same their causes must also be the same, although at that time he had no idea what that cause was. This second axiom was a radical departure from the majoritarian view of his day when diseases were thought to have multiple causes, (Carter, 1981, 58-59) and was redolent of Hume's rule, "the same cause always produces the same effect, and the same effect never arises but from the same cause . . ." (Hume, 223-224).

These two generalizations or axioms—(1) that if the pathological changes at autopsy were the same, the diseases producing those changes must also have been the same, and (2) that if the diseases are the same their causes must also be the same were the major premises of Semmelweis's inferences about the nature and cause of CBF.

When Semmelweis was appointed First Assistant (the equivalent of chief resident) in the first maternity division (Division I) of the AKH on July 1, 1846, his twenty-eighth birthday, he was faced with a problem that had troubled the authorities and alarmed the public for years. At that time, the lying-in section of the AKH had two maternity division: medical students were taught only in Division I and student midwives only in the second maternity division (Division II). Women were admitted from the same pool of non-fee paying patients to the two divisions on essentially alternate days—on Mondays, Wednesdays and Fridays to Division II, and on Tuesdays, Thursdays and the weekend to Division I (Semmelweis 355-356), yet the maternal mortality rate (MMR) from CBF in Division I was three or more times higher than in Division II. In some years more than 500 women died of CBF in Division I, which caused so much public alarm that pregnant women pleaded not to be admitted to Division I or contrived to deliver outside the hospital (the so-called 'street births'). (Semmelweis, 374) Several investigating committees appointed by the Ministry of Education had been unable to determine the cause of this disparity in MMR from CBF between Divisions I and II, but Semmelweis solved the problem in less than a year after his appointment even though he had to step down as First Assistant for five months as his predecessor, Franz Breit, had been offered an extension of his assistantship.

From the fact that women were admitted to the two divisions on essentially alternate days Semmelweis concluded that the cause of the disproportionately high MMR from CBF in Division I had to be endemic, and not epidemic as generally believed, "otherwise one will be forced to the absurd assumption that lethal epidemic influences must be subject to twenty-four-hour remissions and exacerbations in their pernicious activity." (Semmelweis, 358) Semmelweis then sought to identify the endemic factor(s) responsible for the disproportionately high MMR from CBF in Division I by examining whether the accepted causes of CBF could explain the difference in MMR from CBF between the two division, but found that none of the putative causes—epidemics, (ibid., 357-359); the seasons, overcrowding, fear, (ibid., 376), the position in which women delivered, (ibid., 390), and many other factors such as pregnancy itself, embarrassment at being examined by male attendants, etc. (ibid., 379-380)—could explain the difference. All this work left Semmelweis completely in the dark about the cause of the disproportionately high MMR from CBF on Division I, and,

hence, about the cause of CBF, and left him dejectedly to lament, "Everything was uncertain, everything was doubtful, everything was inexplicable, only the enormous number of deaths was an indubitable fact." (Ibid., 390). This was the situation when Semmelweis had to step down as First Assistant on October 20, 1846.

Semmelweis resumed his position as First Assistant on March 20, 1847, after Breit was offered a professorship in Tübingen, but before doing so visited Venice with two friends "to refresh [his] mind and spirits…among the Venetian art treasures." (Semmelweis, 391.) It was on his return from Venice that Semmelweis learned of the pivotal event that would enable him to deduce the nature and cause of CBF: Jacob Kolletschka had died after a student had accidently cut Kolletschka's finger during an autopsy. (Ibid.) Semmelweis examined Kolletschka's autopsy report and noted that the pathological changes, except for the internal genitalia, were essentially the same as those he had repeatedly observe in women who had died of CBF, and from which he educed that Kolletshcka had died of the same disease as had killed so many parturient women, namely, CBF:

From the identity of the pathological findings in the cadavers of the newborns with the pathological findings in the women who died of childbed fever, we had concluded earlier, and we think rightly, that the newborns also died of childbed fever, or in other words, the newborns died of the same disease as did the puerperae. Since we came upon the identical results in the pathological findings in Kolletschka as in the puerperae, the conclusions that Kolletschka died of the same disease, from which I had seen so many hundred puerperae die, likewise was justified. (Semmelweis, 392, italics added).

This was a deductive (modus ponens) inference the form of which is: If P, then Q; P; therefore, Q. In Semmelweis's inference the minor premise (shown in italics) was suppressed: if the pathological changes at autopsy are the same, the diseases producing those changes must also have been the same; the pathological changes at autopsy are the same; therefore, the diseases are the same.

Having deduced that CBF was the same disease as the disease that had killed Kolletschka, Semmelweis relied on his second axiom to conclude that the cause of CBF had to be the same as the cause of Kolletshcka's illness, which was known from the circumstances of Kolletschka's death:

The exciting cause of Professor Kolletschka's illness was known, that is to say, the wound produced by the autopsy knife was contaminated at the same time by cadaveric material. Not the wound, but the contamination of the wound by cadaveric material was the cause of death. Kolletschka was not the first do die in this fashion. I must acknowledge, if Kolletschka's disease and the disease from which I saw so many puerperae die are identical, then in the puerperae it must be produced by the self-same engendering cause, which produced it in Kolletschka. In Kolletschka the specific agent was cadaveric particles, which were introduced into the vascular system. I must ask myself the question: Did the cadaveric particles make their way into the vascular systems of the individuals whom I had seen die of an identical disease? This question I answered in the affirmative. (Semmelweis 1941, 392).

This is again a modus ponens inference with a suppressed minor premise (shown in italics): if the diseases are the same, their causes must be the same; the diseases are the same; therefore, their causes are the same.

Semmelweis concluded that the nature of the disease from which Kolletschka had died was pyemia because the causative agent was introduced directly into Kolletschka's bloodstream by the cut to his finger, and, therefore, the locations at which pathological changes were observed at autopsy could not have been where the disease started, but were, rather, the consequences of the disease process. Since the disease producing the pathological changes in Kolletschka had to be the same as the disease that had produced identical pathological changes in mothers dying of childbed fever, Semmelweis concluded that CBF was also a form of pyemia. (Semmelweis, 558)

Semmelweis had also noted from the many autopsies he had conducted that "no infant ha[d] ever died from CBF while the mother remained healthy," from which Semmelweis concluded that babies contracted CBF not after birth, but in utero from their mothers' blood through the placenta. (Semmelweis, 402) Semmelweis now concluded that the causative agent could similarly gain access into the blood stream of mothers dying of CBF through the raw surface of the placental bed—where the placenta had been attached to the uterus during pregnancy:

In gravidae, parturients or puerperae, there is a place in the body, which has no epidermis or epithelium, and that is the internal surface of the uterus; starting from the internal os upwards, this is the absorption place for the decomposed matter which causes puerperal fever. If wounds are caused by labour, then every place on the genitals, indeed any wound on the body, can be the site of absorption. (Semmelweis, 504).

The vast literature on Semmelweis is entirely silent on a crucial question about his theory: on what basis, if any, did Semmelweis conclude that material from cadavers could, if introduced into the blood stream, cause pyaemia? In his biography of Lister, the surgeon Kenneth Walker wrote that Semmelweis's attribution of the cause of CBF to "decomposed organic matter . . . was an extraordinarily fine guess, as near to the mark as it would be possible for anyone to get at that time." Walker was surely right in concluding that Semmelweis came as close "to the mark as possible at the time", but Semmelweis's conclusion was not a guess; it was almost certainly based on experiments that Gaspard had reported in 1822 and 1824 showing that the injection of putrid matter into animals intravenously caused fever and multiple abscesses, i.e.

pyaemia, (Bullock, 129-131), for Semmelweis wrote in his book, "the fact was known to me that decaying organic matter brought into contact with living organisms produced in them a putrefactive process. (Semmelweis, 393, italics added). Semmelweis must have been aware of Gaspard's work for he also wrote in his book:

Fergusson says that Gaspard and Cruvelhier have injected decomposed matter into the vascular system, and thereby these same inflammatory reactions were produced in animals as we find in puerperae. (Semmelweis, 690)

It was only after Semmelweis had deduced the nature and cause of CBF that the characteristic odor that was well known to linger on the hands of those engaged in autopsies for varying periods of time after they had washed their hands in soap and water entered into Semmelweis's chain of inferences that led to Semmelweis's hypothesis about the cause of the disproportionately high MMR from CBF in Division I:

That the cadaveric particles clinging to the hands are not entirely removed by the ordinary method of washing the hands with soap and water, is shown by the cadaveric odour, which the hands retain for a longer or shorter time. During the examination of gravidae, parturients, and puerperae, the hand contaminated with cadaveric particles is brought into contact with the genitals of these individuals, and hence the possibility of absorption, and by means of absorption, introduction of cadaveric particles into the vascular system of these individuals is postulated, and by this means the same disease is produced in these puerperae, which we saw in Kolletschka. (Semmelweis, 393).

This was only a hypothesis and had to be proved, which Semmelweis sought to do by destroying the cause chemically to determine if this would prevent the disease:

If the hypothesis is correct . . . this disease can be prevented to the extent that it is dependent upon the effect of cadaveric particles carried by the examining finger . . . In order to destroy the cadaveric particles adhering to the hand . . .I began to use "Chlorina liquida" . . .[but] after some time . . . changed to the considerably cheaper chlorinated lime. (Semmelweis, 393, italics added).

Semmelweis's proof was based on the modus tollens inference (the form of which is: If P then Q; not-Q; therefore, not-P), and implemented a principle that Vonka and Gillies have shown dates back to at least the 13th century, and was expressed by Thomas Aquinas (1225-1274) in the maxim sublata causa, tolitur effectus ("if the cause is removed, the effect is taken away"). (Vonka 2000; Gillies 2019, 26, n.3). In 1848, the first full year in which CH-D was practiced, the MMR from CBF in Division I was reduced to 1.27%, a rate no different from the rate in Division II (1.33%). (Semmelweis, 394)

After his assistantship ended on March 20, 1849, Semmelweis conducted nine experiments in domestic rabbits and demonstrated that pathological changes indistinguishable from those found at autopsy of women who had died of CBF could be induced in rabbits by injecting material from cadavers into their lower genital tracts. (Kadar, 390) Semmelweis's inductive proof that CBF was caused by the introduction of the causative agent into the genital tracts of parturient women on the hands of their attendants continued after Semmelweis left Vienna in October, 1850 by the demonstration that CH-D significantly reduced the MMR from CBF at two different hospitals in Pest—to 0.85% at St. Rókus Hospital, (Semmelweis, 415) and to 0.39% during the 1855-56 academic year at the University of Pest Hospital. (Ibid., 424).

Semmelweis adduced additional historical evidence to show that the high rates of CBF in Division I were tied to autopsies. First, he demonstrated that there was a sharp increase in the MMR from childbed fever at the AKH after 1823, when autopsies first started to be performed routinely at the hospital. Before 1823, the MMR hovered around 1%, which Semmelweis took to be the irreducible minimum rate below which the MMR could not be reduced by CH-D, and interpreted the cause as originating from within the mother's own genital tract and called it 'autoinfection'. (See above)

Second, he demonstrated that the disparity in MMR between the two maternity clinics only started after medical students and student midwives were taught on separate divisions. This began in October 1840 pursuant an order from the Ministry of Education. (Semmelweis, 397-399) The second maternity division was created in 1833 (because additional building had been added to the hospital, including 600 maternity beds), but the MMR from childbed fever remained the same on the two maternity divisions until 1941, and only became persistently higher on the first maternity division after 1840 (ibid.)

B. Lister

Wound infections were the burning surgical problem of Lister's day. Surgical procedures were followed so frequently by fatal sepsis from wound infections that far fewer operations were performed than today, and always performed as life-saving or emergency measures, seldom electively.

Although not recognized at the time, suppuration of wounds was also responsible for what called 'hospitalism', a reference to a collection of surgical infections that occurred frequently in hospitals, often in epidemic proportions: hospital gangrene, pyemia, erysipelas and tetanus. Surgical wards at the time were filthy, overcrowded places that reeked of a mawkish stench, and in which "most of the patients were visibly ill, with flushed faces, parched lips, delirium, severe pain, etc., and many of them were evidently on the verge of death." (Cheyne, 1925, 923). Simpson had conducted a survey and compared 2089 amputations performed in hospitals with 2098 amputations performed outside hospital in private homes in pro-

vincial and county practice, and found that "Out of 2089 amputations in hospital practice, 855 died; Out of 2098 amputations in country practice, 226 died; Giving an excess to hospital 'practice of 629 deaths." (Simpson, 1869, 1114-1115) He pointed out that "a man laid on the operating table of one of our surgical hospitals is exposed to more chances of death than the English soldier on the field of Waterloo." (Godlee, 136). This was true whether the patient was the victim of an accident, wounded in battle or had a simple operation like repair of a hernia .

The prevailing theory at the time was that wound infections were caused by exposure to air. This idea originated in the observation that simple fractures almost always healed without infectious complications, whereas compound fractures almost always became infected, even when the breach of the skin overlying the fracture was minimal. Lister told his students in Glasgow

Gentlemen, it is a common observation that when severe injuries are received without the skin being broken, the patients usually recover and do so without any severe illness. On the other hand, trouble—often of the gravest kind—is always apt to follow, even in trivial injuries, when a wound of the skin is present. How is this? I cannot help thinking that the man who is able to explain this problem will be the one who will gain himself undying fame. (Wrench, 83)

Many attributed the cause of wound infections to oxygen and used pressure dressings or continuous irrigation in an attempt to exclude air from wounds. However, the Scottish surgeon, John Hunter (1728-1793), whose writings Lister had studied carefully, concluded that the cause of wound infection could not be the gaseous constituents of air because surgical emphysema was not associated with infections. A rib fracture, for example, in which the end of the fractured rib pierced the pleura but not the overlying skin resulted in a pneumothorax that never became infected, whereas if the end of the fractured rib pierced the overlying skin as well as the pleura the result was an empyema. In other words, infection would not occur if the air was filtered by the lung.

Lister attributed suppuration in compound fractures to the decomposition of blood in the tissues around the fracture site. Blood exposed to air in a glass container at body temperature was known to decompose, and Lister argued that there was no "reason to suppose that the living tissues surrounding a mass of extravasated blood could prevent it from being affected in a similar manner by the atmosphere." (Lister, 1967(a), 326). In a paper read before the British Medical Association in Dublin on August 9, 1867, Lister stated that he had come to this conclusion based on experiments he had conducted on inflammation

In the course of an extended investigation into the nature of inflammation, and the healthy and morbid conditions of the blood in relation to it, I arrived several years ago at the conclusion that the essential cause of suppuration in wounds is decomposition brought about by the influence of the atmosphere upon blood or serum retained within them, and in the case of contused wounds, upon portions of the tissue destroyed by the violence of the injury. (Lister, 1867b, 246)

Lister had been professor of surgery at the University of Glasgow for four years when the professor of chemistry, Thomas Anderson, drew Lister's attention to Pasteur's work. Between 1859 and 1864, Pasteur had been engaged in a debate with the naturalist, Felix Pouchet, over the question of spontaneous generation, and had conducted several ingenious experiments to prove that micro-organisms that appeared in a variety of decomposing media—urine, blood, milk—did not arise spontaneously but originated from living germs carried on dust particles suspended in the air. (Falery & Geison, 1974.) Pasteur showed that these media could be kept sterile in a glass flask despite being exposed to the air as long as the neck of the flask was bent down in such a way that although air could enter the flask the dust particles containing the germs were trapped in its neck and could not ascend. (Bullock, 100) On reading Pasteur's papers, Lister jumped to the conclusion that the causes of wound infections were microorganisms floating on dust particles in the air, and that just as minute organisms suspended on dust particles in the air could be prevented from causing putrefiable media to putrefy, putrefaction and suppuration in wounds could be prevented by preventing these minute organisms floating in the air from gaining access to wounds:

when it had been shown by the researches of Pasteur that the septic property of the atmosphere depended, not on the oxygen or any gaseous constituent, but on minute organisms suspended in it, which owed their energy to their vitality, it occurred to me that decomposition in the injured part might be avoided without excluding the air, by applying as a dressing some material capable of destroying the life of the floating particles. (Lister, 1867a, 353)

Lister chose carbonic acid with which to destroy these microscopic organisms based on a newspaper report on "the remarkable effects produced by carbolic acid upon the sewage of the town of Carlisle", a small amount of which not only "removed all odor from the lands irrigated with the refuse material, but . . . destroy[ed] the entozoan which usually infest cattle fed upon such pastures." Lister, 1867, 327). Lister explained in a letter to the Lancet that he had decided to use carbolic acid because it was "the most powerful of known antiseptics", but he never gave his authority for this claim, nor "had there been articles in the British medical press on the use of this or any other antiseptics in wounds between 1859 and 1865." (Fisher 153).

A. "Listerism": A method of dressing wounds

Lister devised a very complicated method of dressing to keep germs out of wounds, but he repeatedly emphasized that the essence of his theory was the Antiseptic Principle on which his method of dressing wounds was based, and not on any specific feature

of his dressing. That principle was that to prevent putrefaction in wounds one had to (1) destroy any bacteria already in the wound at the time of treatment, and (2) prevent bacteria from entering the wound while it was healing. Lister continually modified his method of dressing wounds to mitigate two undesirable features of carbolic acid: it irritated tissues and was volatile so its effectiveness was vitiated by evaporation. However, he found that each modification was attended with disadvantages which he sought to eliminate by further modifications, and which led to an almost endless cycle of modifications to his dressing, which can be summarized as follows:

To destroy organisms already present in the wound, the wound and all its interstices were swabbed with a pledget of calico or lint held in a pair of forceps and saturated with undiluted crude carbolic acid. To prevent organisms from entering the wound after the operation, a dressing was devised to mimic healing by scabbing that John Hunter had described in the eighteenth century. It consisted of a double layer of lint saturated with undiluted carbolic acid placed over the wound, overlapping it in all directions for about half an inch. A piece of thin block-tin or sheet lead was placed over the double layer of lint to prevent evaporation of the carbolic acid. The metal cap was molded in a concave form to fit the dressing, and fixed in position by strips of adhesive plaster. Blood and serum were allowed to mix with the carbolic acid to form a crust or scab, which adhered tenaciously to the wound and mimicked healing by scabbing. The tin cap was removed daily, and the outer surface of this crust was painted with carbolic acid to prevent the scab from becoming septic.

The method did not work when the wound was large as the flow of blood and serum was too profuse for the antiseptic to prevent the spread of decomposition into the wound. Lister overcame this difficulty by creating a 'putty' dressing composed of carbonate of lime ("whitening") mixed with one part carbolic acid in four parts of boiled linseed oil to form a paste that served as a reservoir for the antiseptic. The putty was rolled out between two pieces of calico, placed over a rag dipped in carbolic acid on top of the wound, and covered with a sheet of block tin to prevent evaporation of the carbolic acid. The putty was raised daily without disturbing the rag covering the wound and painted with carbolic acid as long as there was some discharge. The putty was removed once the discharge ceased, and the wound allowed to heal by scabbing under the rag covering it. The putty technique was also used for dressing operative wounds, and 'cold abscesses' after drainage, as there was no pus or blood with which the carbolic acid could mix to form a scab in thee cases.

The first change Lister made to his dressing was to replace the crude, oily form of carbolic acid that was at first available to him, and referred to as German creosote, as it was "caustic, excoriated the skin, and made living tissues suppurate." (Godlee, 184) It could also cause prolonged vomiting. It was replaced with a 5% solution of water soluble carbolic acid once pure crystals of carbolic acid became available, and used to irrigate the wound.

The next modification was designed to protect the wound from the irritating effects of carbolic acid by the placement of a 'protective' of limited size immediately over the wound that was impervious to carbolic acid and not itself irrigating. Many different materials were tried before Lister settled on "oiled silk covered with copal varnish" that was then "coated . . . with a layer of dextrine and starch." (Godlee, 217).

The putty was next replaced because it was too cumbersome and heavy, first with the 'cerate dressing' and then the 'lac-plaster'. The 'cerate dressing' was made from a mixture of paraffin, wax, a little olive oil, and carbolic acid spread on calico, but it was too brittle, and replaced by a plaster made of shell-lac mixed with carbolic acid in a 4:1 ratio. This time the plaster was too sticky, and Lister tried to overcome this by coating the plaster with a thin layer of gutta- percha. This was the 'lac plaster' dressing, which Lister used until 1870, when he learned that oakum was effective as an antiseptic dressing, and based his dressing on what he called "the oakum principle". He chose resin diluted with paraffin as the vehicle for the antiseptic, and muslin gauze to hold the resin.

Lister next did an about turn and discontinued dressings that did not absorb discharges and adopted his antiseptic gauze technique. (Godlee, 222). The dressing consisted of 8 layers of gauze, with a layer of Mackintosh placed between the seventh and eight layer to prevent evaporation of the carbolic acid. The dressing was made of cheap muslin soaked in a mixture of 4 parts resin 1 part carbolic acid, and 4 parts paraffin. He placed a protective over the wound made of oiled silk covered with copal varnish powdered with dextrin. Lister tried different antiseptics until in 1881 Koch reported his detailed findings and conclusion about antiseptics that

Mercuric chloride is . . . the only known disinfectant which, without any previous moistening or other preparation of the articles to be disinfected, destroys the most resistant organisms in a few minutes by a single application of a highly dilute solution (1 to 1,000 or even 1 to 5,000). (Cheyne, 1886, 517)

Based on Koch's findings, Lister eventually replaced carbolic acid in his dressings with double cyanide and zinc in 1889. (Lister, 1884; Godlee, 297-304).

In 1870 Lister also added the "carbolic acid spray" based purely on his belief about the important role that the air played in the cause of putrefaction in wounds. Indeed, he was so convinced of this role that he believed that

"the mere removal of a drainage tube without antiseptic precautions would be likely to be followed by decomposition in the wound because the air that passed in to take its place would be almost certain to carry in some germs along with it." (Godlee, 282)

Lister used a solution of "one part of carbolic acid to forty of water" to create a vapour of carbolic acid first with a hand-held pump,

then with a foot-operated pump, and finally with a "steam engine". Until 1887 all incisions were made and dressings changed under its protection, but although Lister used the spray for 17 years he did not test, and, therefore, never established that the spray achieved its intended purpose. At a conference in Berlin in1890, an embarrassed Lister publicly acknowledged that the spray never did what it was intended to do, and, therefore, he had abandoned its use.

As regards the spray I feel ashamed that I should ever have recommended it for the purpose of destroying the microbes in the air. If we watch the formation of the spray and observe how its narrow cone expands as it advances, with fresh portions of air continually drawn into its vortex, we see that many of the microbes in it, having only just come under its influence cannot possibly have been deprived of their vitality. (Walker, 121).

At the end of 1867, after he had published his first antiseptically treated series of cases, Lister began a long series of experiments on a more lasting contribution: the development of reliable, sterile chromic catgut. (Godlee, 227-239).

B. Lister's evidence

Lister's first successful treatment of a compound fracture was on August 12, 1865, the day before Semmelweis died. Earlier in the year he had treated two cases unsuccessfully but attributed the failures to methodological errors. In 1867, he reported on the treatment of 11 compound fractures, and presented preliminary results of treating chronic abscesses using his method of dressing wounds. The report comprised of detailed, very long case histories published in five installments in the Lancet in 1867. (Lister, 1867a).

The first article (March 16) presented the reasoning that led Lister to devise his method of dressing wounds, and four cases of compound fractures. A week later another case was reported (March 23), and yet another case reported the week after that (March 30). A month later (April 27) four more cases were described, and another case was added on July 27, together with his preliminary results of treating chronic abscesses. The 11 compound fractures consisted of 7 fractures of the tibia, with fracture of the fibula in 3 cases; 2 fractures of the femur, and 2 fractures of the radius, one including fracture of the ulna. Two patients developed sepsis: one patient died from delayed hemorrhage, the other required amputation. This was a remarkable result as the mortality rate from amputations was about 45% in most centers.

Lister promoted his 'antiseptic principle' and method of dressing wounds in several similar articles published in The Lancet and the British Medical Journal between by describing the application of the antiseptic system of treatment to chronic abscesses and wounded soldiers in detailed case histories. (Lister, 1867b&c; 1868; 1869; 18713) Lister also attempted to show that hygienic measures were not necessary to prevent wound infections by reporting that during the nine month period in which his antiseptic system had been used "not a single case of pyemia, erysipelas or hospital gangrene had occurred" in his wards in Glasgow, which were very unhygienic as they were only four feet from the mass internment site used to dispose of the bodies of the victims of the Glasgow cholera epidemic of 1849. (Lister, 1870a-c) To drive home his contention that hygiene was not necessary to prevent wound infections, Lister stopped the annual cleaning of his ward, did not improve their ventilation or his patients' diet so that any improvement in outcome had to be attributed to the antiseptic treatment. In 1875, Lister published a series of articles describing the changes he had made to his system of dressing wounds. (Lister, 1875) Thereafter, his publications were essentially review articles describing the developments in bacteriology forged by the work of Pasteur and Koch. (Lister, 1880, 1881). In the second of these articles Lister also mentioned Ogston's work but dismissed his conclusion that micrococci were the cause of acute abscesses, and attributed the inflammation in the abscesses to tension, and the subsidence of the inflammation after the abscesses were drained to relief of the tension:

Surely the natural, if not the inevitable, interpretation of this course of events is that the essential cause that kept up the acute inflammation of the abscess and prevented it from subsiding, like that which maintained the chronic inflammation of the bursa patellae, was the tension of the accumulated fluid and that the presence of the micrococci was of an entirely insignificant importance. Hence I am disposed to regard the view which has been taken of this matter by Mr. Cheyne as the one most consistent with the present state of our knowledge—viz. that the micrococci are, so to speak, a mere accident of these acute abscesses, and that their introduction depends upon the system being disordered. (Lister, 1881, 697).

Commenting on Lister's article, Wilson (410) wrote

Lister's paper was an embarrassing lapse on the part of a great man.. He cited no experiments and failed entirely to discuss Ogston's experiments and observations. Lister was clearly a prisoner of his earlier belief that the pus in an unopened abscess was, as a rule, free of micro-organisms. Pus, he thought was sterile and was not subject to putrefaction until it came into contact with micro-organism in the air after an abscess was opened.

Although Wilson called this a rare lapse, on close reading of Lister's articles one can find several similar instances of pure dictum unsupported by experiments or empirical observations, his pronouncements about carbolic acid and the use of the carbolic spray being obvious examples.

C. Proof

The only comparative data Lister ever published was in 1870. His report consisted of comparing the results of the treatment of compound fractures before and during what was called 'the Antiseptic Period'. Cases treated in 1864 and 1866 made up the 'before the Antiseptic Period', and cases treated in the years 1967-1869 made up the 'during the Antiseptic Period'. Lister stated that data for 1865 were missing but did not explain why he had not included the

cases he had treated during the preceding three years, 1861-1863, following his appointment as professor in 1860.

There were 16/35 (46%) deaths before, and 6/40 (15%) during the Antiseptic Period, a statistically significant difference (X2 =8.5, p>0.01). Lister, however, commented that "[t]hese numbers are, no doubt, too small for a satisfactory statistical comparison," because he did not believe in statistics. Lister considered the individual variation between patients too great for comparative statistics to be meaningful. "The truth is that life is short," he wrote in 1879, "and when every day begins, one has to consider what is the occupation which is most likely to be valuable . . . I have felt there was something more congenial and profitable to do than to compile statistics." Therefore, Lister decline to provide further comparative data despite being expressly and repeatedly asked by the Editor of the Lancet to provide further comparative data.

Lister was criticized by his contemporaries for his failure to subject his method to comparative trials. For example, James Morton a professor of surgery at the Glasgow Royal Infirmary wrote in 1870

This method has not been submitted by its proponent to any comparative trial . . . It has been accepted as a foregone conclusion, and with unwavering faith. Philosophic doubt seems to have been discarded: even Mr. Lister's medical brethren are scarcely allowed to doubt; or if they venture to express any doubt, they are very plainly told that they fail to understand it. (Morton, 188)

Morton compared Lister's method, employed by his assistants who were 'believers', with other methods to treat "wounds, compound of fractures, and some other cases" and concluded that "carbolic acid was certainly not superior." (Morton, 188) Tait also tried Lister's antiseptic method and found that it produced the worst result of all the methods he tested; he also published large case series of abdominal operations without antisepsis and lower mortality rates than obtained by Lister's adherents. (Schlich, 417). Callender also pointed out that surgeons themselves contributed to the high infection rates associated with amputations by closing wounds primarily without drainage, and by creating soft tissue flaps to close over the bone that left a large dead space in which fluid could accumulate. (Kernahan, 9).

Callender is representative of British surgeons (the so-called Sanitarians) who belonged to a 'cleanliness school' and were slow to accept Lister's methods because they obtained equally good results by avoiding overcrowding, improving ventilation and the general cleanliness of their wards, by paying close attention to the preoperative preparation of the patient (to which Lister attached little importance, (Kernahan, 21), and by surgical techniques that focused on gentle handling and careful approximation of tissues, complete hemostasis before closure, and twisting vessels for hemostasis to minimize the risk of suppuration. Callender reported no deaths among twenty-five major amputations performed between 1869 and 1872, (ibid., 18), and in 1978, his last publication, Callender reported a mortality rate of 3.4% among 2070 cases, a rate as good if not better than that achieved by Lister. (Ibid., 23)

3. Discussion

It is evident from the foregoing summaries of their work that the acclaim accorded Semmelweis and Lister during their lifetimes is incommensurate with the quality of the evidence and reasoning on which they based their conclusions about the nature and cause of CBF and wound infections, the proofs with which they supported their conclusions or the relative effectiveness of their prophylaxis.

Semmelweis's conclusions about how women contracted CBF, what the sources and vectors of the causative agent were, and how the disease could be prevented were all based on Semmelweis's own empirical observations and deductive inferences that were valid by the tenets of formal logic; his conclusions except for the proximate cause of CBF were all correct, and even his conclusion that CBF was caused by decaying animal organic matter was evidence based. Semmelweis conducted comparative trials and animal experiments to prove his theory.

By contrast, Lister's theory about the cause of wound infections was merely a hunch: he was mistaken about the sources and vectors of the causative agent, and his ideas about the nature of bacteria were incorrect until Pasteur corrected him more than 10 years after his original hunch. Lister's prophylaxis incorrectly focused on the wound, was inapplicable to abdominal and other internal operations because of toxicity, and he resisted each advance that heralded the advent of modern aseptic surgery. Most surprising of all is that Lister provided essentially no evidence to prove the superiority of his method over other methods, and never conducted animal experiments or identified any of the bacteria that caused wound infections.

Why then was the influence that Semmelweis and Lister exerted on their contemporaries so different? It is suggested that five factors account for the difference.

First, it required a paradigm shift to accept Semmelweis's theory because Semmelweis rejected the accepted theories of his day about the cause of CBF and he advanced a theory of causation that was entirely novel. No such paradigm shift was required to accept Lister's theory of the cause of wound infections as his theory was a mere extension of the widely held view that the cause was related to exposure to air. Second, and perhaps even more important, Semmelweis's theory implicated the accoucheur in the cause of CBF, whereas under Lister's theory the surgeon was blameless in the cause of wound infections.

The third factor was the relative frequency of infections after surgical operations compared with deliveries. Because wound infections were so common people expected wounds to become infected, and therefore Lister could demonstrate the effectiveness of his method in a very direct way to visitors to his department, as the

following vivid account by Cheyne illustrates:

Lister then lifted off the outer dressing, which was solemnly handed round to each distinguished foreigner to smell. Having satisfied themselves that there was no putrefaction, the deeper piece of gauze . . . was passed round to show that there was no pus . . . Lister . . . would take a pair of forceps and peel [the protective layer] off, exposing the wound . . . As a rule, this was followed by a sort of gasp of surprise by the distinguished foreigners [on seeing that there was no inflammation or pus in the wound]. . . (Cheyne, 1925, 925)

Childbed fever was much less frequent, and all that visitors to Semmelweis's department in Budapest could be shown was how disinfection was practiced.

The fourth factor were the parallel developments in bacteriology forged by Pasteur and then Koch that validated scientifically Lister's hunch that wound infections were caused by bacteria. Indeed, Lister's publications after about 1875 consisted mostly of review articles in which he described these advances as evidence supporting his original theory. Lister replicated Pasteur's experiments and showed his audiences Pasteur-like flasks containing sterile urine to support his analogical inference that if urine could be kept sterile by excluding the germs in the air, putrefaction in wounds could also be prevented by keeping out the germs in the air. No such demonstration was possible to illustrate Semmelweis's theory. Indeed, even after microorganisms were implicated in the cause of CBF, the air and not the hands of the attendants was considered the predominant vector of the disease, and improved mortality rates were attributed to improved ventilation rather than hand disinfection. (Carter, 1985, 543)

The fifth factor was sociological. Semmelweis and Lister were both born into wealthy mercantile families but there the similarity between their circumstances ended. Lister was a professor when he started his work on antisepsis, able to manage his patients as he best saw fit. He had trained under the pre-eminent surgeon in Britain, if not the entire European Continent, James Syme, married Syme's eldest daughter, and enjoyed Syme's patronage throughout his career, which helped secure Lister professorships at the Universities of Glasgow and Edinburgh after Syme's retirement as Professor of Surgery there.

When Lister was 10 years old, Queen Victoria ascended to the British throne, and during her reign Britain became the preeminent economic, military and political power in the world, so Lister was able to attract many visitors from around the world to see the results of his work in Edinburgh. In addition, the Editors of two internationally recognized and widely read medical journals, The Lancet and The British Medical Journal, offered Lister every opportunity to publish his articles and letters, often the identical articles in both journals, (Lister, 1867b and 1867b2), so Lister's ideas were widely disseminated internationally even as he was developing and modifying his ideas and practices.

The contrast with Semmelweis's circumstances could not have been greater. Semmelweis had trained under a backward thinking chief, Johann Klein, and was First Assistant dependent on Klein's goodwill when Semmelweis developed his theory of the nature and cause of CBF. In 1848, Europe was convulsed by revolutions that barely touched Britain. This engulfed Vienna in March, and progressive members of the University exploited the unrest to demand independence from the Ministry of Education under the banner, Freedom of Teaching, Freedom of Learning. (Lesky, 1976, 91) Thanks to the actions taken by the internist, Joseph Skoda, Semmelweis's discovery was caught up in this battle between the conservative and progressive wing of the university faculty that turned Klein against Semmelweis and caused Klein to deny Semmelweis's request for an extension of his assistantship. (Lesky, 11964, 71) Fisher (127) was incorrect in believing that Semmelweis left Vienna because he was suspected of participating in the revolution: Semmelweis left Vienna because the adjunct professorship (Docenture) he was offered would not have allowed him to continue his research.

When Semmelweis returned to Pest, he was returning to a defeated country under martial law, whose police had been replaced by Austrian gendarmerie that sent spies to all scientific meetings to ferret out political conspiracies and required the minutes of every meeting to be submitted to the authorities. (Kadar, 2019, 34) The Hungarian Academy of Sciences had suspended its meetings, the only Hungarian medical journal had ceased publication, and the only medical publication in Hungary was the officially censored Minutes of the Pest Medical Society. (Ibid.) Semmelweis could finally publish his theory in 1858, once martial law was lifted and the Hungarian 'Medical Weekly' had resumed publishing, but it was in a language that few spoke outside of Hungary.

Conclusion

Lister belongs in the pantheon of great physicians for the changes he wrought in the management of surgical and accidental wounds that were landmarks on the path to modern aseptic surgery. Semmelweis also belongs in that pantheon not only because of the originality and scientific rigor of his work that Lister's work lacked, but also because his pioneering work on disinfection saved the lives of countless thousands of pregnant women and rightly earned him the epithet, Savior of Mothers.