The Obscure Object of Knowledge: German Military Medicine Confronts Gas Gangrene during World War I

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Among the results of the recently revived interest in the medical aspects of World War I have been reexaminations and reassessments of claims about the contributions of war-related research and experiences to medical progress.[1] The official medical histories trumpeted the accomplishments of scientific medicine and argued that the war had significantly advanced medical knowledge. Thus, for example, the official German history contrasted Germany's defeat in the war with the triumphant medical achievements in the field that vindicated the international reputation of German physicians and medical researchers.[2] More recent accounts have been divided over this issue, with some medical historians contending that the war contributed little of lasting value in terms of

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1. General issues of war and medical progress have been raised most critically in Roger Cooter, "Medicine and the Goodness of War," *Can. Bull. Hist. Med.*, 1990, *7:* 147–59; idem, "War and Modern Medicine," in *Companion Encyclopedia of the History of Medicine*, 2 vols., ed. W. F. Bynum and Roy Porter (London: Routledge, 1993), 2: 1536–73; and Roger Cooter and Steve Sturdy, "Of War, Medicine and Modernity: Introduction," in *War, Medicine and Modernity*, ed. Roger Cooter, Mark Harrison, and Steve Sturdy (Thrupp: Sutton, 1998), pp. 6–8. Cooter has been especially critical of treating wartime medicine in isolation from broader social, medical, and peacetime contexts.

2. Wilhelm Hoffmann, "Vorwort," in *Hygiene*, ed. Wilhelm Hoffmann, vol. 7 of *Handbuch der ärztlichen Erfahrungen im Weltkriege 1914/18*, ed. Otto von Schjerning (Leipzig: Johann Ambrosius Barth, 1922), p. v; Otto von Schjerning, "Einleitung," in *Chirurgie*, ed. Erwin Payr and Carl August Franz, vol. 1, bk. 1 of Hoffmann, *Handbuch* (n. 2), pp. v–xx. A translation of Otto von Schjerning's introduction appeared as "The Activities and Achievements of

either organization or specific knowledge that was carried over into peacetime, whereas others have reaffirmed contemporary assertions that the war constituted a kind of gigantic in vivo experiment that led to significant breakthroughs.[3] Given the variety of wounds and the range of infectious diseases treated during the war, it is doubtful that this issue can be decided globally. Rather, it will have to be evaluated in terms of specific fields of research and medical specializations.

In this article I will examine the German medical corps' difficulties with diagnosing and treating gas gangrene, as a case study of the problematic relation between medical research conducted during World War I and medical progress. There are several reasons for doing so. First, immediately following the war some medical writers proclaimed gas gangrene research, especially research on etiology and antitoxin sera, to be an example of the achievements of scientific medicine—a claim that has recently been echoed by a leading German medical historian.[4] Second, the two central medical concerns of the war, the treatment of wounds and the control of infectious disease, intersected in cases of gas gangrene, as did two leading medical specializations, bacteriology and surgery—thus making gas infections a major focus of military medical research activity. Third, in the early twentieth century, German medical research in both of these specializations was arguably preeminent and hence set the international standards.

Without completely discounting the achievements of German bacteriology and scientific medicine, I will argue that gas gangrene research and treatment were far from being unequivocal successes. Instead, because of a well-established horizon of expectations, the German medical corps entered the war ill prepared to cope with gas gangrene. Wartime condi-

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Medical Officers of the German Army during the War: Introduction to the 'Handbook of Medical Experiences in the World War,'" trans. F. H. Garrison, *Milit. Surg.*, 1920, *46:* 430–55.

3. Roger Cooter, *Surgery and Society in Peace and War: Orthopaedics and the Organization of Modern Medicine, 1880–1948* (Houndsmills: Macmillan, 1993), pp. 105–36; J. D. C. Bennett, "Medical Advances Consequent to the Great War 1914–18," *J. Roy. Soc. Med.*, 1990, *83:* 738–42; Robert Hawk, "Military Medicine Comes of Age: The First World War," *J. Florida Med. Assoc.*, 1992, *79:* 309–13; Wolfgang U. Eckart, "'Der grösste Versuch, den die Einbildungskraft ersinnen kann'—Der Krieg als hygienisch-bakteriologisches Laboratorium und Erfahrungsfeld," in *Die Medizin und der ersten Weltkrieg,* ed. Wolfgang U. Eckart and Christoph Gradmann (Pfaffenweiler: Centaurus-Verlagsgesellschaft, 1996), pp. 299–319. Eckart supports the notion of the war as an in vivo experiment and affirms the success of pragmatically oriented wartime research, including research on gas gangrene.

4. Franz Klose, "Gasödem," in Hoffmann, *Hygiene* (n. 2), pp. 547–73. See also Eckart, "'Der grösste Versuch'" (n. 3), pp. 311–12, on the ostensible success of gas edema antitoxin serum; and Schjerning, "Activities and Achievements" (n. 2), pp. 437–38.

tions, although offering a vast amount of experience with gas infections, posed insuperable impediments to effective medical research on these disturbingly intractable and highly lethal wound complications. Throughout the war, often-vituperative polemical debates abounded among bacteriologists and surgeons over the etiology, diagnosis, clinical picture, and therapy of gas gangrene—debates that in many instances continued unabated and unresolved. Moreover, physicians often had to resurrect procedures previously regarded as superseded, thus undermining prevailing notions of linear medical progress. Finally, as in many areas of military medicine, limitations on resources and personnel militated against the widespread implementation of expensive and labor-intensive but promising therapies.

Earlier Experience and Research

When war broke out in 1914, strong reasons could be adduced for believing that the risk of fatal wound infections was minimal, especially from anaerobic bacteria. The first reason was the apparent success attained by introducing aseptic wound management and therapy, as championed by the eminent surgeon Ernst von Bergmann since the 1880s and confirmed by subsequent experiments, clinical experience, and extensive evidence accumulated in small-scale colonial wars. Bergmann, a Baltic German, publicly renowned as Kaiser Friederich III's surgeon during the emperor's terminal throat cancer in 1888, had served as a military surgeon during the Austro-Prussian War of 1866, the Franco-Prussian War of 1871, and the Russo-Turkish War of 1877–78.[5] Although an early follower of Joseph Lister's antiseptic principles, during the latter war he had come to favor immediately covering fresh wounds with sterile occlusive bandages and leaving them to heal with as little intervention as possible, since operating antiseptically under field conditions was impossible. Although his precise intellectual itinerary cannot be fully reconstructed, during the 1880s when he served as director of the Royal Surgical Clinic of the University of Berlin and professor of surgery at the Medical Surgical Academy for the Military (later the Kaiser Wilhelm Academy), Bergmann gradually abandoned Lister's antiseptic surgery

5. Still standard is the monumental heroic biography by his student Arend Buchholtz, *Ernst von Bergmann*, 2d ed. (Leipzig: F. C. W. Vogel, 1911), which reprints Bergmann's extant war letters. It contains a wealth of information on surgery in late-nineteenth-century Germany. There is also an account of Bergmann, especially on Friedrich III's cancer operation, in Owen H. Wangensteen and Sarah D. Wangensteen, *The Rise of Surgery: From Empiric Craft to Scientific Discipline* (Minneapolis: University of Minnesota Press, 1978), pp. 8–9, 181–84.

and began developing aseptic surgery and wound treatment into a comprehensive system. After performing experiments with different disinfectants on bandages, Bergmann concluded that these solutions had little effect on the outcome of wound treatment.[6] In contrast to Lister's emphasis on sterilizing already-infected wounds with antiseptics such as carbolic acid solution, Bergmann began stressing the prevention of festering by disinfecting the skin near wounds and forestalling bacterial entry by covering them with sterile bandages. Transposing these principles to the operating room and hospital ward, Bergmann pioneered the sterilizing of the gowns of surgeons and nurses, as well as of the instruments and bandages that might come into contact with the wound.[7]

Subsequent bacteriologic research and the evidence from colonial wars underscored the validity of applying Bergmann's aseptic procedures to gunshot wounds. In principle, primary infection could arise either directly from bacteria on a bullet or indirectly when bacteria on clothing or skin penetrated the wound canal. However, experiments performed in the 1890s suggested that infections originating from any of these sources, although certainly theoretically possible, were highly improbable.[8] First, tests carried out during the 1890s showed that when small-caliber bullets were fired into gelatin, bacteria colonies failed to form. The heat generated by bullets was insufficient to sterilize their surfaces, and bullets artificially coated with pathogenic bacilli could infect the shot canal; but such a mode of infection appeared to be a distinctly remote possibility. From a practical medical perspective, simple gunshot wounds could be regarded as sterile. Second, Eduard Pfuhl, a military hygienist and Robert Koch's son-in-law, designed a series of experiments in which fragments of cloth from soldiers' dirty uniforms were inserted under the skin, in the pleura or peritoneum of laboratory animals, without infections occurring—thus ostensibly proving that clothing driven into wounds was unlikely to be a source of infection (a lesson that would have to be unlearned during the world war). Finally, both bacteriologic experiments and daily clinical experience showed that most bacteria carried on human skin were harmless. Such results from laboratory experiments were reinforced by field observations and statistics obtained from recent wars. Because of the less-destructive nature of modern

6. Buchholtz, *Ernst von Bergmann* (n. 5), pp. 435–36.

7. The first book that laid down the principles of aseptic surgery and wound treatment was written by Bergmann's assistant: Curt Schimmelbusch, *Anleitung zur aseptischen Wundbehandlung* (Berlin: August Hirschwald, 1892); Bergmann wrote the introduction. See esp. pp. 175–79 for wound treatment.

8. Dr. A. Hildebrandt, *Die Verwundungen durch die modernen Kriegsfeuerwaffen, ihre Prognose und Therapie im Felde*, 2 vols. (Berlin: August Hirschwald, 1905), 1: 125–26.

bullets, and improved therapies based on Bergmann's principles, the prognoses for bullet wounds of soft tissues were two to three times better in the wars between Japan and China and the Spanish-American War than during the Franco-Prussian War.[9]

Bergmann, who maintained close ties to the Prussian Sanitary Corps, regularly trained army surgeons assigned to his clinic; hence, many of the more than two hundred Sanitary Corps officers who treated over a thousand gunshot wounds during Germany's brutal war against the Herero in Southwest Africa were trained by Bergmann and proceeded in accordance with his precepts.[10] With the exception of a handful of marginalized dissenters, by 1914 a general consensus reigned in German sanitary formations about the efficacy of Bergmann's aseptic wound management.[11] As Dr.Winkelmann, a surgeon and medical director of a field hospital, testified in 1916 about the understanding of wound treatment and supplies at the outset of the war,

> We wound doctors entered the war armed with the experience of our peacetime activity and that which German and foreign physicians had gathered and written from distant theaters of war during the last decades. Both taught that rifle shots were germ free, germ poor, or not poisonously unclean, and they usually healed with certain surety if later uncleanliness was prevented. The dirtier artillery-shell wounds healed less well, but still, after sufficient exposure and care, the tendency to self-cleansing and healing showed. Furthermore, cleansing the wound with antiseptics or other measures was impossible, even injurious. With us went a satisfactory quantity of unobjectionably germ-free bandages and material for casts, as well as equipment for eliminating germs by boiling and medicaments for cleansing the hands and objects that could not be boiled.[12]

If the triumph of aseptic surgery was the main reason why military physicians expected few difficulties from wound infections, a second reason also left them completely unprepared to deal with gas gangrene—namely, that in the prewar period it had been a rare and exotic complication familiar to only a few bacteriologists. My search of the medical literature on war-wound infections between the Franco-Prussian War and

9. Ibid., pp. 132–34.

10. Buchholtz, *Ernst von Bergmann* (n. 5), pp. 552–59, esp. pp. 556–57.

11. For some dissent from Bergmann's principles see Hildebrandt, *Die Verwundungen* (n. 8), pp. 136–39; Georg Schöne, "Behandlung frischer Kriegswunden und Verhütung des Ausbruches der Wundinfektion," in Payr and Franz, *Chirurgie* (n. 2), p. 152.

12. Dr. Winkelmann, "Erfarhungen über Behandlung der Kriegswunden mittels Dakin'scher Lösung," *Bruns' Beiträge zur klinischen Chirurgie* (herafter *Bruns' Beiträge*), 1916, *101* (4): 436–54, quotation on p. 436.

the Balkan Wars of 1912–13 turned up only a handful of references. Thus, for example, a survey of recent articles on gas gangrene published in 1917 cited six papers from the Balkan Wars, each of which traced the clinical course of several cases.[13] Certainly there had been research on pathogenic anaerobic bacteria before the war:[14] Robert Koch had conducted research on anaerobic wound infection, which he called gas edema, and had discovered *B. oedematis maligni*, a motile, rod-shaped anaerobe. In 1892 both the American pathologist William Welch and the German bacteriologist Eugen Fraenkel isolated the immotile *B. welchii* (*welchis perfringens*), usually designated the Welch–Fraenkel bacillus in Germany, which was rightly regarded as the primary infective agent in most cases of gas gangrene. The following year Fraenkel wrote the first monograph on anaerobic wound infection, a monograph that, along with the one on pathogenic anaerobes by Emanuel von Hibler written in 1908, was still consulted as standard during the world war. Other infective anaerobes, including the Ghon–Sachs edema bacillus, had also been identified and classified. Nonetheless, there can be little doubt that, as Dr. Carl Franz (a student of Bergmann's who served in Southwest Africa and was then appointed professor of war surgery at the Kaiser Wilhelm Academy) asserted in 1917, "this wound infection disease was something entirely new and unknown to the overwhelming majority of physicians at the outset of the war. Only a few surgeons knew it from peacetime, because like tetanus, it seldom appeared."[15]

The New Conditions of World War I

When confronted with the grim realities of the western front, sanguine expectations about the minimal risk of life-threatening wound infections rapidly disappeared. Soon the teachings of Bergmann were being treated as artifacts of specific historical conditions. In a lecture to surgeons in March 1916, Dr. Georg Marwedel of Aachen retraced the rapid disillusionment experienced by field doctors:

13. Dr. Leo Zindel, "Die neueren Arbeiten über Gasphlegmone," *Bruns' Beiträge*, 1917, *105* (2): 257–92, see especially pp. 263–64.

14. For some of the earlier research on anaerobic bacteria see Klose, "Gasödem" (n. 4), pp. 547–54; Otto Jüngling, "Histologische und klinische Beiträge zur anäroben Wundinfektion," *Bruns' Beiträge*, 1917, *107* (3): 443. See also the monograph by Dr. Emanuel von Hibler, *Untersuchungen über die pathogenen Anäroben* (Jena: Gustav Fischer, 1908).

15. Dr. Franz, "Über die Gasentzündung," *Bruns' Beiträge*, 1917, *106* (4): 443–84, quotation on p. 443.

> At the beginning of the war, we harbored the belief that the new fundamental propositions that in accordance with the teachings of v. Bergmann on shot wounds had become the common property of physicians, would easily put us in position in large part to hold surgical wound infections at bay and to combat them effectively. . . . Then, however, came blow-by-blow disillusionment, and at present we cannot conceal that the number of wound infections has forfeited absolutely no meaningful losses in comparison with earlier times, and when one considers the number of wounded in today's million-man armies, the number of wound inflammations must necessarily increase.[16]

At the Second War Surgery Conference, held in Berlin in April 1916, naval physician General August Bier defended Bergmann's doctrines as schematic, but basically appropriate for earlier wars:

> It is . . . unjust to accuse von Bergmann of having presented a false doctrine on the treatment of war wounds. His doctrine is certainly one-sided; it has all the weaknesses and gaps, in short all the defects that go together with almost any schema, but in general it applies to the wars in which he participated and in which he gathered his experiences. There it essentially involved infantry shots from afar and fewer soldiers soiled by earth. Von Bergmann could not know that war would assume such entirely different forms.[17]

As this quote suggests, several unexpected characteristics of war weaponry and the resulting wounds compelled physicians to retreat from the firmly established prewar doctrine. According to the standard prewar German text on gunshot wounds, "Injuries produced by the shot of artillery have previously been less thoroughly and deeply handled from a war surgical perspective, than the injuries of handheld weapons. The essential ground for this is that in all wars the number of artillery injuries is far fewer than the number of shot wounds produced by rifles."[18] The official postwar German report on surgery estimated, however, that during the world war 75 percent of wounds could be ascribed to artillery shells or shrapnel. Moreover, in contrast to the often smooth, neat wounds from small-caliber bullets, fragments from high-explosive artillery shells, mines, or shrapnel that penetrated the body with "devitalizing and even pulpifying force" carved out jagged, irregular gashes that

16. Georg Marwedel, "Einige Betrachtungen über die Wundinfektion des jetzigen Krieges," *Feldärztliche Beilage zur Münchener medizinische Wochenschrift* (hereafter *Feldärztliche Beilage*), 4 July 1916, p. 982.

17. Dr. Bier, "Anärobe Wundinfektion (abgesehen von Wundstarrkrampf)," in *Verhandlungen der zweiten Kriegschirurgentagung, Berlin, 26. und 27. April 1916,* published in *Bruns' Beiträge*, 1916, *101* (3): 271–335, quotation on p. 272.

18. Quoted in Franz Bruening, "Die Kampfmittel im Weltkriege und ihre Wirkungen auf den Körper," in Payr and Franz, *Chirurgie* (n. 2), p. 20. See also pp. 20–24 for the effects of artillery shells on soldiers' bodies.

transformed soldiers' bodies into replicas of the "Trichtergelaende," or "cratered land"—as Germans called the lifeless, charred, bombarded wasteland that separated the opposing trenches.[19] These powerful shell and mine explosions not only deadened tissue and shattered bone, they also left behind fragments of shells or shrapnel and forcefully drove large pieces of uniforms, earth, splinters, skin, and other foreign bodies deep into the wound. Since much of the fought-over land in northern France had been assiduously cultivated and dunged for centuries, and since soldiers spent more than a week at a stretch in the trenches, during which time their skins and uniforms became encrusted with mud, such foreign bodies inevitably introduced pathogenic bacteria into wounds. Furthermore, and quite contrary to prewar expectations, the depth of the wounds and the surrounding necrotic tissue constituted an ideal oxygen-free environment in which anaerobic bacteria and their spores, present in large quantities in the fertile soil, multiplied and throve. Although reliable statistical data are lacking, substantial impressionistic evidence indicates that anaerobic infections, although less frequent than aerobic infections, became by far the most serious wound complications.

During the early months of the war, tetanus emerged as the major wound complication on the western front, with a mortality rate of at least 75%.[20] Cases of tetanus declined precipitously, however, with the introduction of widespread prophylactic injection of wounded soldiers with Emil von Behring's tetanus antitoxin serum, a preventive measure introduced incrementally in late 1914 and early 1915 as manufacture and imports began to match demand.[21] As the importance of tetanus waned, gas gangrene thrust to the fore as the most troubling and lethal wound infection. Since military physicians were never required to report gas gangrene, however (except late in the war, when antitoxin serum was being tested), and since diagnosis was often exceedingly difficult, statistical material for the German armies is fragmentary and unreliable. Prof. Dr. Marwedel and Dr. Wehrsig wrote that among wounded soldiers

19. W. W. Keen, *The Treatment of War Wounds*, 2d ed. (Philadelphia: Saunders, 1918), pp. 62–67; the phrase "devitalizing and pulpifying force" appears on p. 65. A more recent discussion of wound ballistics during World War I is included in Ronald F. Bellamy and Russ Zajtchuk, eds., *Conventional Warfare: Ballistic, Blast, and Burn Injuries. Textbook of Military Medicine*, Pt. 1, vol. 5 (Washington, D.C.: Office of the Surgeon General, 1990), pp. 90–91.

20. For the impact of tetanus early in the war see Fr. Völker, "Zur Behandlung des Tetanus," *Feldärztliche Beilage*, 27 October 1914, p. 2146; Dr. Kreuter, "Bericht über 31 Tetanusfälle nach Kriegsverletzungen einheitlich intraspinal and intravenös mit Serum behandelt," ibid., 17 November 1914, p. 2255; and the official report, *Sanitätsbericht über das Deutsche Heer im Weltkriege 1914/18*, vol. 3, *Die Krankenbewegung bei dem Deutschen Feld und Besatzungsheer* (Berlin: Mittler, 1934), pp. 77–82.

21. *Sanitätsbericht* (n. 20), p. 84.

treated in Aachen in the fall of 1914, they had seen as many cases of gas gangrene as of tetanus, over three hundred altogether.[22] Early in the war Carl Franz estimated that 2% of all wounded soldiers had gas infections—a figure he later revised downward to 0.3%, since he had omitted the lightly wounded.[23] In early 1917 Otto von Schjerning, the Director of Field Sanitary Services, commissioned a study of the incidence of gas infections on the western front. This study found that a total of 516 wounded soldiers contracted gas infections during the first three months of the year, or 0.6% of those wounded.[24] Whether this figure can be accepted as representative is an open question, since apart from the difficulties and confusions about diagnosis (to be explored below), figures fluctuated seasonally and among the various sectors of the front.

Incidence of infection also depended on the rapidity with which the wounded were collected and treated. One French study claimed that 13% of the wounded contracted gas gangrene, whereas a probably more accurate British study carried out during the intense German offensive of spring 1918 placed the figure at slightly above 1%.[25] Whatever the actual numbers and percentages, the enormous literature and sharp debates about gas infections testify to the considerable anxiety that these lethal complications evoked within the German Sanitary Corps.

The Enigmas of Gas Gangrene

Debates abounded—over the name of the disease, the problem of diagnosis, the clinical picture, the efficacy of various therapies—and regularly spilled over into both general medical journals and specialist surgical periodicals throughout the war. As to the name of the infection, various physicians called it "gas phlegmon" (*Gasphlegmone*), "gas edema" (*Gasödem*), "gas gangrene" (*Gasgangrän*), "gas burn" (*Gasbrand*), "malignant emphysema" (emphysema malignum), "gas inflammation" (*Gasentzündung*), or "gas infection" (*Gasinfektion*), depending on the clinical symptoms or stages of infection they highlighted.[26] The debate

22. Dr. Marwedel and Dr. Wehrsig, "Über Gasbrand durch anärobe Streptokokken," *Feldärztliche Beilage*, 27 July 1915, p. 1023.

23. See the comments during the discussion at the Second War Surgical Congress in *Verhandlungen* (n. 17), pp. 331–32. Franz later revised the figure up to 0.5%: Franz, "Über die Gasentzündung" (n. 15), pp. 445–46.

24. *Sanitätsbericht* (n. 20), p. 84.

25. Cuthbert Wallace, "Gas Gangrene," in *History of the Great War Based on Official Documents: Medical Services: Surgery of the War*, ed. W. G. MacPherson, vol. 1 (London: H.M. Stationery Office, 1922), pp. 135–36, 148.

26. Dr. Fessler, "Die Gasphlegmone (Gasödem, Gasgangrän, Gasbrand oder Emphy-

over nomenclature was itself a sign of the difficulty in arriving at a consensus on a differential diagnosis or a unified clinical picture. Thus, for example, Carl Franz argued against "gas phlegmon" on the ground that it implied progressive suppuration, whereas in most instances of "gas inflammation" (the term he preferred) no pus was formed.[27] Dr. August Bier, by contrast, defended "gas phlegmon," since despite the absence of pus, some unified concept was necessary and "phlegmon" had the advantage of calling attention to the rapid progress of the infection.[28]

Even more heated (and ultimately more important) than debates over nomenclature were the clashes among bacteriologists over which bacteria caused gas gangrene, and whether or not each infective agent gave rise to different symptom complexes. As mentioned above, before the war a number of anaerobes were known to be agents of gas infections, the most important of which was the one discovered by Fraenkel and Welch in 1892. Since pathologists could not invariably find Fraenkel bacilli in the tissues of autopsied soldiers who died of gas gangrene, a number of bacteriologists searched for other etiologic agents. In a series of articles in *Münchener medizinische Wochenschrift* in 1916 two bacteriologists, Heinrich Conradi and Richard Bieling, who worked in an army infectious disease laboratory, asserted that the etiology of gas gangrene was poorly understood.[29] On the basis of their bacteriologic investigations of every case of gas infection in one German army over a short period—a total of fifty-three cases—they attributed gas gangrene to a highly motile Gram-positive rod-shaped bacillus, probably *Clostridium sacrophysematodes*. Morphologically, it closely resembled the Rauschbrand (symptomatic anthrax) bacillus, a bacterium quite distinct from the Fraenkel bacillus. Conradi also opined that there might be just one infective bacterium of which the Fraenkel bacillus was a variant, since the agent underwent radical morphological changes—sometimes assuming the form of an immotile rod, as described by Fraenkel, but sometimes transforming into a motile, flagellated bacillus in living tissue.[30]

In a sharp rebuttal, Fraenkel replied that his own bacteriologic and histologic investigations, as well as the observations of countless other

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sema malignum)," *Feldärztliche Beilage*, 6 March 1917, p. 331; Zindel, "Die neueren Arbeiten über Gasphlegmone" (n. 13), pp. 281–82.

27. Franz, "Über die Gasentzündung" (n. 15), p. 484.

28. Bier, "Anärobe Wundinfektion" (n. 17), pp. 277–78.

29. H. Conradi and R. Bieling, "Zur Ätiologie und Pathogenese des Gasbrands," *Feldärztliche Beilage*, 25 January 1916, pp. 133–35; 1 February, pp. 178–82; 11 July, pp. 1023–25; 31 October, pp. 1561–64; 7 November, p. 1608.

30. See Conradi's comments at the Second War Surgical Conference in *Verhandlungen* (n. 17), pp. 333–34.

doctors in the field, demonstrated that the Fraenkel bacillus was present in most, if not all, cases of gas gangrene.[31] Moreover, these bacilli appeared precisely as he had limned them in the 1890s and did not undergo the remarkable metamorphoses that Conradi ascribed to them. When they were cultivated in pure colonies and injected into guinea pigs in accordance with Koch's postulates, the animals were struck rapidly by gas infections. Fraenkel noted that similar infections were caused by other anaerobic bacteria, such as Koch's edema bacillus, but such cases were rarer. He suspected that Conradi and Bieling had isolated a human symptomatic anthrax bacillus, which was not the etiologic agent of true gas gangrene. Fraenkel was not in a position to determine whether these different pathogenic anaerobic bacilli could be correlated with distinct clinical symptoms, but since they were distinct infective agents and had distinct microscopic effects on tissues, he had no hesitancy in maintaining that they gave rise to quite distinct diseases.

Further research during the war largely confirmed Fraenkel's contentions, though the issues still were not entirely resolved by the end of the war.[32] Nor did this research contribute to improving diagnosis. Since most wounds nourished richly diverse mixtures of anaerobic bacteria (and often aerobic bacteria as well, with complex interactions), and since anaerobic microbes were elusive and difficult to cultivate even by experienced technicians in the best-equipped laboratories, bacteriologic diagnosis in military hospital laboratories was practically precluded.[33] Bacteriologists were largely confined to analyzing tissues from autopsies. Moreover, despite the paucity of prewar work and the volume of wartime writing, no major contributions were made toward understanding infective anaerobes.

31. Eugen Fraenkel, "Kritisches über Gasgangrän," *Feldärztliche Beilage*, 29 March 1916, pp. 476–80.

32. See, e.g., R. Pfeiffer and G. Bessau, "Über bakteriologische Befunde bei den Gasphlegmonen Kriegsverletzter I," *Deutsche medizinische Wochenschrift*, 27 September 1917, pp. 1217–20; Klose, "Gasödem" (n. 4), pp. 547–49. For his comments on morphology see Eugen Fraenkel, "Über Gasbrand," *Deutsche medizinische Wochenschrift*, 14 December 1916, pp. 1533–35. On the unresolved character of this debate see Martin Kirschner, "Wundinfektionskrankheiten," in *Die Deutsche Chirurgie im Weltkrieg 1914 bis 1918*, ed. August Borchard and Victor Schmieden (Leipzig: Johann Ambrosius Barth, 1920), pp. 182–83.

33. Some of the difficulties in isolating and cultivating pure cultures of anaerobic bacteria, and problems with bacteriologic diagnosis, are indicated in Klose, "Gasödem" (n. 4), pp. 547–48, 562. See also Dr. Wilhelm Kolle and Dr. Heinrich Hetsch, *Die experimentelle Bakteriologie und die Infektionskrankheiten*, 6th ed., 2 vols. (Berlin: Urban & Schwarzenberg, 1923), 1: 582–89, for some of the difficulties of culturing anaerobes as well as a list of literature on these problems, much of it from the war years.

While bacteriologic diagnosis of gas gangrene was excluded in most military hospitals, clinical diagnosis often proved difficult and unreliable as well, especially in the infection's early stages. Certainly there were specific types of wounds that immediately aroused suspicions of gas infections, since they offered ideal conditions. These included extensive and jagged wounds with small entries and irregular pockets—caused by high-explosive artillery shells or shrapnel—into which pieces of clothing, bone fragments, earth, and foreign bodies had been driven and in which muscle tissue was lacerated and prolapsed.[34] The wound had usually bled extensively; the blood vessels suffered severe damage, and hematomas were often present. The more extensive the muscle trauma and the greater the damage to the surrounding circulatory system, the higher the probability that gas infections would develop. Necrotized muscle tissue formed the necessary medium for gas infection, and the soft tissues of the extremities—thighs, calves, upper arms, or buttocks—were the most common sites. Under favorable circumstances, the bacteria, which were toxigenic, hemolytic, and saccharolytic, began generating the telltale gas that was a harmless metabolic by-product, and toxic bacteria-laden edema began spreading to previously healthy muscle tissue, which rapidly lost its ability to contract and changed color to brownish red.[35] Fulminant gas gangrene proceeded with alarming rapidity. In rare instances, local infections could metastasize and the bacilli infect distant organs. In most cases, bacterial toxins spread, and if the infection was not arrested death followed from paralysis of the basal nerve centers.

Although cases of gas gangrene could almost invariably be diagnosed in advanced stages and confirmed by autopsies, the difficulty that field doctors confronted was the detection of cases in their early stages when the patient's life might still be saved. In ascending order of reliability, field doctors based their diagnoses on general constitutional changes, the appearance of the wound, the nature of the edema, and signs of gas formation—none of which was entirely foolproof. In mild cases the appearance of the wounded and their subjective feelings were indistinguishable from those of patients with pyogenic wound infections:

34. Bier, "Anärobe Wundinfektion" (n. 17), pp. 272–74; Franz, "Über die Gasentzündung" (n. 15), pp. 446–50. For an excellent English-language source from the war see Ellsworth Eliot, "Gas Gangrene," in *The Medical Department of the United States Army in the World War,* vol. 11, *Surgery,* ed. Merritte Weber Ireland (Washington, D.C.: Government Printing Office, 1927), pp. 267–68.

35. Bier, "Anärobe Wundinfektion" (n. 17), pp. 280–84; Kirschner, "Wundinfektionskrankheiten" (n. 32), pp. 183–84; Eliot, "Gas Gangrene" (n. 34), pp. 269–71.

> The temperature remains within moderate bounds, the pulse shows a middle value corresponding to the fever; the sick persons make a good general impression and retain their appetite. Only the considerable pain that usually begins suddenly points to a special complication of an otherwise indicated light or moderately serious disease picture of suppurating infection.[36]

In more serious cases, constitutional changes were more pronounced. The pulse was light and rapid, often over 140—although the temperature remained moderate around 39 %C (101–103 %F), and the tongue, despite fever, moist. The face was pallid with hints of jaundice, and in the severest cases patients gasped for air, since the oxygen content of their blood was diminished. The wounded sometimes vomited and had diarrhea, and they often experienced restlessness, though seldom delirium.

In contrast to aerobic wounds, tumor and dolor were present, but not calor and rubor.[37] Because of the damaged circulatory system and the absence of leukocytes, pus was also absent. Especially in cases of deep-lying anaerobic infections, no typical change in the wound appearance occurred, but in most cases the surrounding skin was discolored, turning orange-brown or blue. Vesicles, which often followed the superficial veins, proliferated in the discolored area. The swelling, due to edema and gas emission, was often substantial, and the wound site was sometimes described as resembling an air pillow. Most infected wounds emitted a yellowish, malodorous, serous edema that subsequently turned a dirty brownish color. Depending on the infective agent, some gas gangrene wounds generated vast amounts of edema, which oozed from the wound and spread into lymphatic spaces and subcutaneous tissues, whereas others produced relatively little.

The most reliable method for determining whether a gas gangrene infection was present was to search for signs of gas formation.[38] However, no test or tool yielded a certain diagnosis. Hence there was considerable discussion about preferred diagnostic procedures to detect gas production. Some cases of gas gangrene gave off no notable gas until the final stages. Moreover, according to some German surgeons it was necessary to bear in mind that:

36. Kirschner, "Wundinfektionskrankheiten" (n. 32), p. 186. See also ibid., pp. 186–87, for constitutional changes accompanying more serious cases; Eliot, "Gas Gangrene" (n. 34), p. 276.

37. Bier, "Anärobe Wundinfektion" (n. 17), pp. 274–75; Franz, "Über die Gasentzündung" (n. 15), pp. 450–53; Kirschner, "Wundinfektionskrankheiten" (n. 32), pp. 187–88; Eliot, "Gas Gangrene" (n. 34), pp. 272–74.

38. Bier, "Anärobe Wundinfektion" (n. 17), pp. 294–96; Franz, "Über die Gasentzündung" (n. 15), p. 462; Kirschner, "Wundinfektionskrankheiten" (n. 32), pp. 187–88.

> not every collection of gas encountered in the bodily tissues must be caused by a gas infection. Collections of gas in tissues also occur as a result of injuries of air-carrying organs (larynx, trachea, lungs, cellulae ethmoidales, colon, duodenum), of air carried along by voluminous bullets that hit the skin at sharp angles, of the entry of explosive gases from close shots, and of incorrect injections.[39]

Gas bubbling from an open wound, however, was an almost certain sign of a gas infection. In more superficial wounds, physicians could feel crackling gas pockets beneath the skin. Where such gas pockets were not close to the surface, another common and fairly dependable way to detect gas was to tap lightly at the site of suspected infection with a pencil or flat object: if gas was present, the physician could hear a tympani-like percussion roll. But neither of these methods was infallible: in many cases the infection simply lay too deep to discover crackling pockets of gas and, moreover, wound canals and bones near the surface often gave off tympanitic sounds when tapped. Surer by far were X rays: when these revealed striations of gas between and in the muscle tissues, gas gangrene could be diagnosed with virtual certainty. Especially when gas was subcutaneous, however, some plates were difficult to interpret, and in any event many field hospitals lacked Roentgen machines. Hence belated and mistaken diagnoses remained common.

A major debate that suggested the problematic nature of diagnosis and pointed to the amorphous and poorly defined nature of the disease entity was the one that divided German military surgeons into two camps. The first believed that there were two forms of gas gangrene: one fulminant with a practically hopeless prognosis, the other mild with a relatively good prognosis; and the second held that this distinction simply confused real gas gangrene with more-localized gas abscesses or other sorts of infections. In early 1915 the eminent professor of surgery at the University of Leipzig and consulting army surgeon Medical Privy Councilor Dr. Erwin Payr published a short but highly influential and much cited article in the *Münchener medizinische Wochenschrift* that distinguished between two forms of gas phlegmon.[40] Payr considered the first, which he called subcutaneous or epifascial, as largely benign. It was characterized by swelling under the skin, a pale edema, which preceded a change in the color of the skin surrounding the wound to a lemon yellow or orange, with some hemolytic copper-red spots or stripes that often followed the

39. Kirschner, "Wundinfektionskrankheiten" (n. 32), p. 188.

40. E. Payr, "Über Gasphlegmon im Kriege," *Feldärztliche Beilage*, 12 January 1915, pp. 57–58. Payr's epifascial form may have been anaerobic cellulitis or necrotizing fascitis. See Bellamy and Zajtchuk, *Conventional Warfare* (n. 19), pp. 210–11.

course of the veins. The infection spread rapidly, but could usually be halted by making numerous 2–3 cm incisions in the fascia. By contrast, the second form, which he called subfascial, was malign and spread with uncanny rapidity through the muscle tissue, transforming it into chocolate-colored wet pap. Cutting deeper incisions or rinsing the wound with hydrogen peroxide might stop the infection if it was recognized early, but once it had started to spread only amputation could arrest its fateful progress—and even this radical procedure frequently failed.

A number of Payr's colleagues dissented. Bier suspected that Payr's subfascial form usually indicated relatively benign localized gas abscesses that might develop into serious gas gangrene under certain circumstances, but that should not be conflated with the disease itself.[41] Even more critically, Carl Franz argued that Payr's distinctions introduced a series of confusions. Many gas abscesses and wounds were largely infected with aerobic bacteria (although anaerobic bacteria and their spores might also be present) due to pockets of exogenous air driven in by explosion from close shots. Most cases of so-called epifascial gas phlegmon were thus not real gas infections at all. Payr had probably diagnosed correctly some real cases of gas phlegmon, but rather than being "epifascial," the anaerobes were infecting necrotic muscle tissue near the surface of the skin; Payr's incisions then killed these bacteria by admitting air to the infected site.[42] Franz's case against "epifascial gas phlegmon" was affirmed by one field doctor in 1918 who noted that among the hundreds of cases of gas phlegmon he had encountered in the field, he had yet to observe a single case of Payr's epifascial form; instead, in even the most superficial wounds with anaerobic infections, serious muscle damage could be shown.[43]

A variant of Payr's distinction was revived and elaborated in 1917–18 by Professor Anton Thies of the University of Giessen, who also served as a staff physician in the field. He too believed that there were two distinct forms of gas infection, the second and rarer of which coincided in most respects with the subfascial form described by Payr. He suspected that it was a distinct disease restricted to certain sectors of the western front, perhaps caused by the Ghon–Sachs bacillus, although the bacteriologic evidence was too equivocal to allow him to do more than speculate about this.[44] But whether these were two forms or distinct diseases, they mani-

41. Bier, "Anärobe Wundinfektion" (n. 17), pp. 275–77.

42. Franz, "Über die Gasentzündung" (n. 15), pp. 282–84. For "exogenous gas" see the quote associated with n. 39 supra.

43. Julius Fleissig, "Feldspitalchirurgie im Stellungskrieg 1915–17," *Bruns' Beiträge*, 1918, *109* (5): 663.

44. A. Thies, "Die Behandlung der Gasphlegmone mit der rhythmischen Stauung,"

fested clinically distinct symptoms. The first, less serious form resulted in brown skin color, the emergence of subepidermal pockets with clear yellow serum, and the formation of yellow-green edema in the connective tissue—among other distinguishing features. The more hopeless, fulminant form displayed itself by a rapidly spreading reddish blue skin color.

Franz contested this version as well. According to him, the "blue" gas infection was "the typical picture of those gas inflammations that begin deep in the musculature of a limb or body part, and the projection of which to the external surface requires a longer time until it is perceptible to sight and feeling."[45] Calling the other form "brown" was unfortunate as well, since brown skin was less the expression of gas infection per se than the absence of blood in the skin and underlying tissue.

As part of his determined search for a more accurate and easier clinical diagnosis, Otto von Schjerning charged the pathologist General Staff Doctor Ludwig Aschoff with investigating whether Thies's distinction could be validated. In August 1918, after Aschoff had determined that surgeons perceived a distinction between brown and blue forms on both the eastern and western fronts, Schjerning requested that military surgeons report on whether such distinctive forms were almost invariably found, what sorts of exceptions they encountered, and how cases of each of these forms turned out, independently of whether or not gas edema serum had been injected.[46] These reports were supposed to be submitted by 1 December 1918, but the war's end meant that they were never completed, collated, or evaluated. Hence the issue of distinct forms was never settled satisfactorily.

This disagreement not only exacerbated difficulties in diagnosing and treating gas infections, but also largely accounts for the extraordinary discrepancies in mortality rates that various surgeons estimated. Surgeons who lumped all cases together tended to report mortality rates as low as 12%, whereas Dr. Hermann Kümmell at the first War Surgery Conference in Brussels placed mortality at 33% for gas gangrene cases in which appropriate surgical intervention had taken place.[47] At the second War Surgery Conference in Berlin, August Bier demurred, claiming that

Bruns' Beiträge, 1917, *105* (5): 595–636, see especially pp. 610–14; idem, "Über zwei Hauptformen der Gasinfektion," ibid., 1918, *109* (2): 157–93.

45. Dr. Franz, "Kritisches zur Diagnose der Gasentzündung," *Deutsche medizinsiche Wochenschrift*, 27 September 1917, pp. 1220–21, quotation on p. 1220.

46. HSTAB (KA) AOK 6 Armee Arzt Hygiene Shriftenverkehr 1914–18 Bd. 250: Chef des Feldsanitaetswesens v. Schjerning, Gr. Hauptquartier den 21.8.1918 an alle Herren Armeeaerzte des Westens.

47. Dr. Kümmell, "Wundinfektion, insbesondere Wundstarrkrampf, Gasbrand

if localized infections in which gas was present were omitted, mortality for the remaining real gas gangrene cases was substantially higher.[48]

Attempts at Prophylaxis and Treatment

The direct cause of death in cases of gas gangrene was heart or respiratory stoppage from toxemia. It is therefore not surprising that the Prussian War Ministry, in conjunction with a number of pharmaceutical firms, launched several research projects to develop an antitoxin serum that could be injected as either a prophylactic or a cure; the hope, of course, was to replicate the successes of Emil von Behring's diphtheria and tetanus sera. Another strategy was to vaccinate directly against the bacilli, and in the summer of 1916 Schjerning intensified this effort by appointing Franz Klose director of the gas edema laboratory at the Kaiser Wilhelm Academy with the aim of researching, testing, and standardizing an antitoxin or finding a vaccine.[49] While the dye firm Höchst had already been contracted to develop a serum on the basis of experiments by Aschoff and his colleagues, the Behring Works in Marburg and the Ludwig Gans Pharmaceutical Institute initiated other projects, all of which ultimately fabricated sera—antitoxins, bactericides, or inhibitors of bacterial growth. These projects enlisted such eminent bacteriologists as August von Wassermann and Wilhelm Kolle. The goal was to produce polyvalent sera that would be effective in neutralizing the toxic byproducts of, or killing, a range of anaerobic pathogens, including the Fraenkel bacillus, the symptomatic anthrax bacillus, Koch's gas edema bacillus, and the Ghon–Sachs bacillus.

Multiple difficulties, both practical and theoretical, hampered and delayed these projects.[50] It was difficult to procure horses, which were needed both for research and for producing serum, and equally difficult to procure adequate fodder for the horses that were available. Moreover, strains of the Fraenkel bacillus varied considerably in their virulence, which made it impossible to produce a satisfactorily standardized serum. Although several sera were rushed into use at the front, they were all manufactured in small quantities and at best offered limited protection for a short duration.

(Verhütung durch primäre Wundversorgung)," in the partial transcript of the "Kriegschirurgentagung in Brüssel am 7. April 1915," *Feldärztliche Beilage*, 20 April 1915, p. 571.

48. Bier, "Anärobe Wundinfektion" (n. 17), pp. 296–97.

49. Klose, "Gasödem" (n. 4), p. 563; on attempts to fabricate bactericidal as well as antitoxin sera, see pp. 563–67.

50. Ibid., pp. 564–66, 568–69.

In April 1917 the serum developed by Höchst became the first to be approved. The advisory leaflet, however, warned that

> the gas edema serum should hinder wound infection only until the usual necessary surgical interventions can be undertaken: the opening, cleansing, or debridement of the wound, or amputation of the limb. Unlike tetanus serum, it offers a relative, not an absolute, protection. *The surgical treatment of the wound must therefore be carried out as before with the same care and in the same manner.*[51]

A dose of 20 cc was to be injected in the muscles near the wound as soon as possible and, again unlike tetanus serum, injections were to be repeated within the first three days after every transport or surgical procedure. To substitute for the absence of clinical trials and to gather some credible information on the serum's efficacy, a little over a month later Schjerning required that a card recording every case of gas edema be sent to Klose at the Kaiser Wilhelm Institute, independently of whether prophylactic injections had taken place.[52] He further instructed sanitary officers and physicians to fill out more comprehensive reports on their use and doses of serum. Advisory surgeons were to follow closely the clinical course of gas edema cases in which wounded soldiers had been injected, and pathologists were to perform autopsies on all cases in which soldiers died despite having received serum. Wound material from those soldiers who contracted gas edema after injection was to be preserved and sent to the bacteriologic laboratory of the medical investigation section of the Kaiser Wilhelm Academy.[53]

A second serum, produced by the Gans Institute, was approved for testing in the field in November 1917, although only a thousand 10 cc and five hundred 20 cc doses were made available for the entire Bavarian Sixth Army.[54] Given the primitiveness and lack of homogeneity in evaluative procedures, it is not surprising that considerable doubt arose as to whether the sera worked, either as prophylactics or as cures—doubts that were pervasive in the British and French medical corps as well.[55] On the

51. HSTAB (KA) AOK 6 Armee Arzt Hygiene Schriftenverkehr 1914–18 Bd. 250: Chef des Feldsanitätswesens v. Schjerning, Gr. Hauptquartier den 22.4.1917 an den Herrn Feldsanitätschef beim Oberfehlhaber Ost und alle dem Chef des Feldsanitätswesens im Gr. H. Qu. unmittelbar unterstellten Herren Armee usw. Ärzte und Etappenärzte, and the accompanying Höchst Gebrauchsanweisung (italics in original).

52. Ibid., Chef des Feldsanitätswesens v. Schjerning, Gr. Hauptquartier den 8.6.1917.

53. Ibid., Chef des Feldsanitätswesens v. Schjerning, Gr. Hauptquartier den 23.4.1917.

54. Ibid., Chef des Feldsanitätswesens v. Schjerning, GHQ 15.11.1917 an den Herrn Armeearzt 6.

55. Eliot, "Gas Gangrene" (n. 34), pp. 279–80, included a brief account of the research on experimental polyvalent antitoxin sera by French, British, and German researchers. He

basis of the limited and admittedly defective material submitted from the field, after the war Klose reached modest conclusions about the effectiveness of these sera. Because of the numerous anaerobic pathogens and strains, he stated, none of the sera was sufficiently polyvalent to provide absolute protection:

> Despite this, however, serum treatment in conjunction with surgical measures is suitable to impede successfully the mortality of this frightful war epidemic, since 1,314 gas edema cases, some of which were treated with serum, some of which were not, showed that the mortality of those treated with serum lagged behind those not treated by 16%: 42%, compared to 58%.[56]

Yet he was unable to vouch for whether physicians had injected appropriate doses at the appropriate times, nor did he provide any information about the process by which these two groups were selected, or the relative seriousness of their cases.

At best, serum bought some time for the surgeons to carry out their "usual necessary surgical interventions" (in the words of the Höchst advisory).[57] These surgical procedures clearly constituted a complete break with the principles of conservative aseptic treatment institutionalized by Bergmann and his disciples before the war. Soon after the start of the war most surgeons were forced to conclude, however reluctantly and hesitantly, that the best way to prevent gas infections, as well as pyogenic infections—at least in extensive wounds caused by artillery shells—was by radical preventive wound treatment. Judgments continued to vary widely,

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emphasized Frances Ivens's prophylactic injections on 433 wounded soldiers from March to September 1918, many of whom had clinical signs of gas gangrene and only eight of whom died directly from this infection. From these results Ivens had concluded that serum was "of real value in preventing gas gangrene" (p. 280). Eliot found it unfortunate, however, that there had been no comparison with wounded soldiers on whom only surgical measures had been used, and he remarked that mortality rates might have been higher in other areas of the front given the variable virulence of gas gangrene infections. In the British official history, Cuthbert Wallace noted that in a comparative trial in June 1918 in which two large groups of wounded (9,294 and 13,024) were given either just tetanus serum or a combination of tetanus and anti–*B. welchii* serum, the mortality rates were the same among those contracting gas gangrene, about 22%. He allowed, however, that the dose of gas gangrene serum administered was about 1/5 of that recommended. He reported the French enthusiasm for anti–gas gangrene sera and agreed that their results were promising enough that research should be continued: Wallace, "Gas Gangrene" (n. 25), pp. 147–49. For Klose's evaluation of two French sera see Klose, "Gasödem" (n. 4), pp. 569–70. He found that they provided no protection against the Welch–Fraenkel bacillus, but in animal trials he determined that they were comparable to German sera against motile *Butyrikus* bacilli like the Ghon–Sachs edema bacillus (*Vibrion séptique*).

56. Klose, "Gasödem" (n. 4), p. 573.

57. Höchst Gebrauchsanweisung (n. 51).

however, over which wounds could be handled conservatively and which required radical preventive treatment.[58] Preventive treatment entailed extracting all shell fragments, pieces of cloth, bone fragments, and other foreign bodies; excising all damaged and necrotic tissue; and splitting open all abscesses and pockets.[59] Primary excision was usually followed by drainage, open wound treatment, and a resort to various forms of antisepsis—other revived procedures that violated Bergmann's injunctions. One common antiseptic for gas gangrene was hydrogen peroxide, which was forced deep into the wound. Some surgeons pronounced it highly effective for rinsing or irrigating wounds suspected of harboring gas infections, but others hesitated after reports that patients had died of embolisms following this procedure.[60]

Radical wound excision also had its dangers and limitations: not only was it often mutilating, but this approach also quickly came up against insurmountable practical limitations.[61] First, during large-scale battles,

58. Georg Schöne agreed with his colleague Garré that the war had restored the need for careful surgical judgment based on evaluation of the specific wound rather than either stereotyped conservative treatment or invariable surgical intervention. During the war, smooth rifle shots and some shrapnel wounds were still handled conservatively, as recommended by Bergmann, whereas all artillery wounds and most shrapnel wounds were handled actively. See Schöne, "Behandlung frischer Kriegswunden" (n. 11), pp. 133–40, esp. pp. 138–39. For the development of British, French, and U.S. thinking on wound excision, and the development of the terminology, see Bellamy and Zajtchuk, *Conventional Warfare* (n. 19), pp. 91–92. The French distinguished between *débridement*, the opening of the wound by incision, and *épluchage*, the excision of devitalized and damaged tissue; whereas the Americans collapsed the two terms. Germans used excision (*excidieren*) for the latter, a usage close to the British "primary wound excision." Whatever the terminology, surgeons in all army medical corps came to regard "physical disinfection" by excision as the most effective means for preventing wound sepsis and especially anaerobic infection.

59. Schöne, "Behandlung frischer Kriegswunden" (n. 11), pp. 140–52, including an account of the debates and development of thinking on active preventive wound treatment during the war; for a general discussion of the return to antiseptics, see pp. 152–54. Further accounts include Prof. Fessler, "Weitere Erfahrungen über die Gasphlegmone," *Feldärztliche Beilage*, 16 November 1916, pp. 1581–82; and Kirschner, "Wundinfektionskrankheiten" (n. 32), pp. 148–50, 189–91. The same lessons on the need for wound excision or debridement were, of course, learned by military surgeons in the allied armies as well; see Wangensteen and Wangensteen, *The Rise of Surgery* (n. 5), pp. 161–63. For an account of the techniques of debridement and suture as practiced during the First World War with a more recent commentary and illustrations, see Bellamy and Zajtchuk, *Conventional Warfare* (n. 19), pp. 204–9.

60. Eugen Fraenkel, "Über Gasgangrän," *Feldärztliche Beilage*, 10 November 1914, p. 2218; Eduard Borchers, "Vorsicht bei der Sauerstoff Behandlung der Gasphlegmone!" ibid., 28 September 1915, pp. 1338–41; Hermann Kehl, "Klinik und Behandlung der anäroben Wundinfektion," in Payr and Franz, *Chirurgie* (n. 2), p. 247.

61. Bier, "Anärobe Wundinfektion" (n. 17), p. 298; Fleissig, "Feldspitalchirurgie" (n. 43), pp. 661–62.

the wounded were often brought to field hospitals so late that infections were already spreading. Second, when swamped by cases, surgeons simply did not have sufficient time to extract fragments from every extensive wound, especially since wounds caused by high-explosive French shells often contained hundreds of splinters. Third, in many cases of shattered bones, removing all fragments early often worsened the prognosis for healing, as Bergmann had preached. Finally, in the vicinity of major veins, arteries, and nerves, extractions were highly dangerous. Thus, except during lulls at the front, such thoroughgoing wound revision was seldom possible, and in some cases was unwarranted. Moreover, even such radical preventive measures provided no absolute guarantee against further infection.

Surgery was necessary, however, to deal with anaerobic infections once they were already under way. As could be inferred from Payr's article, the most important surgical procedures were the cutting of incisions to introduce air to kill the anaerobic bacilli, and amputation, especially in cases of fulminant gangrene. Incisions were frequently made when the surgeon believed that the infection was still in its early stages, the site was superficial, or the infection had attacked parts of the body unsuitable for amputation. In his comprehensive study of gas infection, Carl Franz reported that nine of twelve soldiers died whom he had treated only with incisions because the infections were located in the buttocks, shoulders, back, or stomach.[62] Of thirty cases of gas infection of the extremities that he had treated only with incisions, eighteen had died. When incisions had been followed by amputations, eleven of twelve had died—an appallingly high mortality rate that he attributed to his initial reluctance to amputate.[63] In one case, the patient's pain disappeared after incisions and he felt much better, perhaps because of the anesthetic. When Franz visited the patient a few hours later, the wound looked better and no gas was issuing forth. Nonetheless, he was deceiving himself: the patient's pallor and rapid pulse remained unchanged, and a few hours later he died of gas gangrene.

The lesson, of course, was that the most effective way to deal with gas gangrene was through early amputation, a lesson that stood in clear contradiction to Bergmann's conservative approach. Better to mutilate patients than to lose them. Only eleven of forty-seven patients whose limbs Franz had amputated early had died, a marked improvement over his other results. From this he adduced as a fundamental principle that "*as soon as a shot fracture of a long bone or a large joint is accompanied by a gas*

62. Franz, "Über die Gasentzündung" (n. 15), pp. 473–77.
63. Ibid., pp. 474–75.

inflammation, then an immediate amputation is necessary."[64] As revealed by the official German medical history and other postwar evaluations of gas gangrene treatment, by the end of the war this principle had won universal acceptance. Amputations were generally considered the surest means of saving the lives of soldiers with gas infections of the extremities.[65]

Two other experimental methods for treating early infections of areas such as the buttocks were cataplasm and rhythmical constriction (*rhythmische Stauung*). Both of these treatments, which were unique to German wartime medicine, sought to induce hyperemia—that is, to flood areas adjacent to necrotic tissue with oxygenated blood.[66] Cataplasm seems to have been more common. As described by Bier, an advocate of hyperemic treatment long before the war, sacks filled with cooked linseed meal and warmed with steam heat were placed around the affected areas. These poultices were as hot as the patient could endure and not only brought about hyperemia, but also blistered the skin. The application of poultices continued around the clock for several days, cooling poultices being replaced by new hot ones every hour or so. The patient sweated profusely, and the pain often disappeared rapidly. According to Bier, of the sixteen patients he had treated in this fashion, only four had died—three of gas gangrene, the fourth of an unrelated lung embolism. Moreover, many of the wounds of these sixteen patients still contained numerous fragments and none of them had previously received any sort of surgical treatment.[67] Assessments by other physicians who tried cataplasm, however, were far from unanimous about its value. Some recommended it highly; others allowed that it worked for gas gangrene of the buttocks, but nowhere else; still others avowed that outcomes were no better than with any other mode of treatment.[68]

The second method of hyperemic treatment, rhythmical constriction, was largely devised by Thies. This method required the use of a machine powered by gas or air pressure and regulated by a feedback mechanism.[69]

64. Ibid., p. 475 (italics in original). See also pp. 475–79 for the advantages of early amputation.

65. H. Cönen, "Ein Rückblick auf 20 Monate feldärztlicher Tätigkeit mit besonderer Berücksichtigung der Gasphlegmone," *Bruns' Beiträge*, 1916, *103* (1–3): 397–463, see especially pp. 426–31; Dr. Mertens, "Zur Frage der Amputation bei inficierten Kriegsschussverletzungen," ibid., *103* (4): 604–16, see especially pp. 604–6; Kirschner, "Wundinfektionskrankheiten" (n. 32), p. 189; Kehl, "Klinik und Behandlung" (n. 59), pp. 245–46.

66. Eliot, "Gas Gangrene" (n. 34), p. 282.

67. Bier, "Anärobe Wundinfektion" (n. 17), pp. 319–23.

68. Kehl, "Klinik und Behandlung" (n. 60), p. 250; Cönen, "Ein Rückblick" (n. 64), pp. 436–37.

69. For a description and illustration of the machine, as well as an account of the

A band was placed above the infected area (usually an arm or a leg) and gas from the machine filled the band under a pressure of .12–.15 atm for a minute; pressure was then released for approximately two minutes, and the cycle was repeated. This alternating cycle of exerting and releasing pressure was applied for a period ranging from two days to two weeks, depending on the nature of the infection and the patient's state of health; in most cases, five to ten days sufficed. According to Bier, rhythmic constriction brought about major changes in the patient's sense of well-being and in the infected limb. The process produced an enormous edema; the infected area turned a flaming red and was hot. When the area was touched, a crackling and tympanitic sound could be heard that generally indicated a gas abscess still concealing a foreign body. The sick muscle healed beyond expectation: the patient felt much better, and his pain disappeared. Initially the limb swelled, not only with edema but also with gas, but the gas was soon reabsorbed or else streamed from the wound. No gas gangrene occurred as a consequence of the limb's swelling. Bier reported that of the 106 gas phlegmon cases he had treated exclusively with this method, only 17 had died, 10 of these from unrelated complications.[70] Thies proudly claimed equally positive results. He admitted, however, that rhythmical constriction was not effective in treating cases of fulminating gas gangrene, which he called the "blue form." Of the hundred cases of mild or "brown form" handled with this procedure, only five had died. In seventeen cases an operation had been necessary subsequently, and in six cases amputation. Thies warned against applying this method schematically, however, since adjustments had to be made for individual cases. But in many cases, he avowed, rhythmical constriction was an exceptionally effective cure.[71]

Not all of Bier's and Thies's colleagues shared their enthusiasm. Since Franz questioned Thies's ability to diagnose gas gangrene accurately, he expressed considerable skepticism about the reported rate of success with rhythmical constriction. He strongly suspected that many of the cases treated by Thies and Bier were either localized gas abscesses or mixed infections containing both aerobic and anaerobic bacilli, rather than genuine instances of gas gangrene.[72] Another surgeon who experi-

technique of rhythmical constriction, see A. Thies, "Die Behandlung chirurgischer Infektionen mit rhythmischen Stauung," *Feldärztliche Beilage*, 8 August 1916, pp. 1165–69.

70. Bier, "Anärobe Wundinfektion" (n. 17), pp. 304–5, 307.

71. Dr. A. Thies, "Die Behandlung der Gasphlegmone mit der rhythmischen Stauung," *Bruns' Beiträge*, 1917, *105* (5): 595–636, see especially pp. 634–36. For comments on the limits of the technique as well as cautions against its schematic application, see ibid., pp. 596–97; Thies, "Die Behandlung chirurgischer Infektionen" (n. 68), p. 1166.

72. Franz, "Über die Gasentzündung" (n. 15), pp. 470–72.

mented with rhythmical constriction, Oskar Rumpel, concluded that it was effective only when the infected muscle lay just beneath the skin—and even then results improved when it had been preceded by thorough wound revision, including splitting open gas pockets.[73] In any event, discussions about the effectiveness of rhythmical constriction turned out to be moot, since the technique could never be introduced on a large scale. Although the machine could handle as many as sixteen patients simultaneously, even Thies complained that its exorbitant cost diminished prospects for its widespread use. Rumpel observed that not only was the procedure costly, but it could be carried out in only a handful of well-endowed war hospitals, since it was difficult to master and depended on a large staff of experienced health-care workers who could constantly monitor the patients and ensure that the apparatus was functioning properly. Hence hyperemic treatments, although much discussed, were largely restricted to small-scale trials conducted by leading surgeons associated with university clinics. Thus, no novel therapies gained widespread acceptance during the war.

Conclusion

In the final analysis there is not a shred of credible evidence that the incidence of gas gangrene decreased during the war, or that the treatment of gas gangrene improved significantly. The comprehensive final report of the chief army physician of the Bavarian Sixth Army on medical conditions during the spring offensive of 1918 noted: "The scourge of gas phlegmon has still not disappeared despite serum treatment. I would like to make the observation that in most cases gas phlegmon is recognized too late, because following a schema, and often with a certain self-satisfaction, every wounded soldier whose wound is bandaged and therefore not visible is viewed as cared for."[74] In part such late recognition was probably attributable to the specific conditions of the 1918 spring offensive, when the collection of the wounded was often delayed, front-line field hospitals were overwhelmed with wounded soldiers, transport to the rear was backed up, physicians were overworked, and supplies were short—though it may also be testimony to how many surgeons automatically reverted to Bergmann's conservative approach under conditions of extreme pressure, especially in treating gunshot and even some shrapnel

73. Kehl, "Klinik und Behandlung" (n. 60), p. 249.

74. HSTAB (KA) AOK 6 Hygiene Gesundheitssammelberichte 1914–18 Bd. 250: Armee Arzt 6 AHQu 12 August 1918 "Sanitätsbericht für die Monate April, Mai und Juni 1918," in the section of the report on Kriegslazarette.

wounds.[75] Even allowing for the near breakdown of medical services in spring 1918, the report testifies to the continued seriousness of gas gangrene to the war's end.

It is difficult to disagree with the Hermann Kehl's judicious assessment in the official medical history of the war:

> During the entire war wherever the wounded were to be treated, the question was pursued with greatest diligence, if methods other than . . . surgical prophylaxis could keep anaerobic wound infection distant from our wounded. All methods that were tried and temporarily praised as cures, have certainly sometimes essentially improved the after treatment of wounds, but they have not sufficed to make anaerobic infection disappear as a wound disease.[76]

Research on gas gangrene as well as serum prophylaxis and therapy were far from being the resounding successes that some German physicians proclaimed after the war and some medical historians have subsequently reaffirmed. Indeed, without stretching the evidence, wartime research could be described more plausibly as a failure in terms of its proponents' expectations of rapid progress in eliminating gas gangrene. There is no question that bacteriology and the German Sanitary Corps could legitimately claim some extraordinary successes. The virtual disappearance of tetanus infections after early 1915 is certainly a case in point, although one paralleled by the army medical corps of other belligerents as well—but tetanus was a well-known infection and a reliable clinically tested antitoxin serum had existed for more than twenty years before the war. In contrast, only a handful of bacteriologists and surgeons had even the most glancing acquaintance with gas gangrene and there had been no research on gas edema serum, largely because, apart from specific wartime conditions, anaerobic infections (except tetanus) were exotic rarities. Nor did wartime conditions lend themselves to the production of new bacteriologic knowledge or the accurate testing of sera or new surgical procedures.

No major advances were made in terms of understanding anaerobic infections, and surgeons who entered the war with complete confidence in Bergmann's principles of conservative aseptic wound treatment found themselves resurrecting procedures that had been relegated to the medical

75. For a rather understated, but nonetheless revealing, report about the extraordinary difficulties with transport of the wounded, placement of central bandaging stations, loss of medical personnel, shortages of materials, and staggering caseloads during the 1918 spring offensive, see the twenty-two-page report of the chief physician of the Sixth Army: HSTAB (KA) Bd. 264 Armeearzt 6 "Erfahrungen während der Apriloffensive bei d. 6 Armee."

76. Kehl, "Klinik und Behandlung" (n. 60), p. 246.

archives—including radical excision and wound antisepsis, which had been previously dismissed as dangerous and tissue-damaging.[77] Gas edema sera, which were novel, proved to have limited efficacy at best.

This case study should suggest some of the key reasons why, despite a vast amount of accumulated medical experience, attempts to improve diagnosis and treatment under wartime conditions resulted in numerous unresolved debates and dissention—rather than in effective research, as many bacteriologists and surgeons had hoped and expected. The exigencies of war basically precluded the gathering of reliable statistics; diagnosis and treatment had to take place near the front, in field hospitals under highly unfavorable circumstances, and were implemented by physicians with no previous experience in this area. Research was hampered by shortages of funding and materiel, and medical researchers were often overburdened with military-related duties—as was the case with Aschoff, Franz, Payr, and Bier, all of whom were carrying out staff and advisory duties in addition to bearing responsibility for research projects. The need to find quick solutions and rapidly introduce them in the field worked against careful and accurate clinical trials. If research on gas gangrene was representative, then speaking of the killing fields of World War I as a kind of in vivo medical experiment is misconceived, and assertions about the favorable relation between the war and advances in medical and hygienic knowledge need to be subjected to a far more critical and probing scrutiny than has hitherto often been the case.

77. For open wound treatment and wound antisepsis, both of which had been regarded as heretical before the war, see Dr. O. Braun, "Über aseptische und offene Wundbehandlung im Feldlazarett," *Bruns' Beiträge*, 1917, *105* (1): 69–91, see especially pp. 70–75; O. Hirschberg, "Zur Wundbehandlung mit Dakinscher Lösung. Physiologisches Antisepsis," *Deutsche medizinische Wochenschrift*, 21 December 1916, pp. 1581–82, in which he speaks of "the new antisepsis"; Dr. L. Kathariner, "Der verschiedenengradige Wert der Antiseptika für die Wundbehandlung," *Feldärztliche Beilage*, 2 November 1915, pp. 1522–24. There was a vast German medical literature on the use and results of the Carrel–Dakin method as well.