Chapter 2

THE HISTORICAL IMPACT OF PREVENTIVE MEDICINE IN WAR

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INTRODUCTION

> A corps of Medical officers was not established solely for the purpose of attending the wounded and sick; the proper treatment of these sufferers is certainly a matter of very great importance, and is an imperative duty, but the labors of Medical officers cover a more extended field. The *leading idea*, which should be constantly kept in view, is to strengthen the hands of the Commanding General by keeping his army in the most vigorous health, thus rendering it, in the highest degree, efficient for enduring fatigue and privation, and for fighting.[1p100] [emphasis added]
>
> —*Jonathan Letterman, 1866*
> *Medical Director, Army of the Potomac*

Jonathan Letterman was best known for his operational and administrative contributions, such as his system of field hospitals and his reorganization of medical services.[2,3] Yet, in his mind, the *leading idea*, as he put it, was prevention.

Letterman's emphasis was (and is) appropriate on at least two counts. First, disease and nonbattle injury (DNBI) historically caused more deaths than battle injury (BI) until World War II, with few exceptions.[4,5] Table 2-1 illustrates this point using US Army war experience. Note that this relationship became less pronounced with time until, during World War II, BI deaths exceeded DNBI deaths. This crossover from DNBI to BI, resulting largely from advances in both preventive and curative medicine,[6] does not mean that DNBI has become less of a concern. When one studies morbidity (by considering hospital admissions) as opposed to mortality, it is clear that DNBI causes more combat ineffectiveness than BI, even during periods of sustained fighting (Figure 2-1). From 1941 to 1945, 95% of all Army admissions (16,941,081 of 17,664,641) were due to DNBI. During the Korean War, DNBI accounted for 82% of admissions (365,375 of 443,163, includes those hospitalized and excused from duty).[4] The second point in support of Letterman's emphasis is that DNBI is largely preventable, whereas BI is less amenable to prevention.

TABLE 2-1

US ARMY DEATHS FROM DISEASE AND NONBATTLE INJURY VS BATTLE INJURY, MEXICAN WAR THROUGH PERSIAN GULF WAR.*

War	Number Serving†	BI	DNBI	DNBI:BI
Mexican War‡§	78,718	1,733	11,550	6.7
Civil War (Union)§	2,128,948	138,154	221,374	1.6
Spanish American War§	280,564	369	2,061	5.6
World War I	4,057,101	50,510	55,868	1.1
World War II	11,260,000	234,874	83,400	0.35
Korean War	2,834,000	27,709	2,452	0.09
Vietnam War	4,368,000	30,922	7,273	0.24
Persian Gulf War¥	246,682	98	105	1.1

*Historically, DNBI has caused more deaths than BI. This relationship became less pronounced with time. Since World War II, BI deaths have generally exceeded DNBI deaths.
†Total number in Army during conflict (See notes for Mexican and Persian Gulf Wars.)
‡Predominately Army force; contains unknown number of sailors and Marines
§Based on incomplete records
¥Includes only Army troops deployed on Operation Desert Storm
BI: battle injuries
DNBI: disease and nonbattle injuries
Sources: (1) *The World Almanac 1996*. Mahwah, NJ: World Almanac Books, Funk & Wagnalls Corporation; 1996: 166. (2) Data from the Department of Defense, Directorate for Information, Operations, and Reports, Statistical Information Analysis Division, Washington, DC, July 1997. Prepared by Judith Bowles.

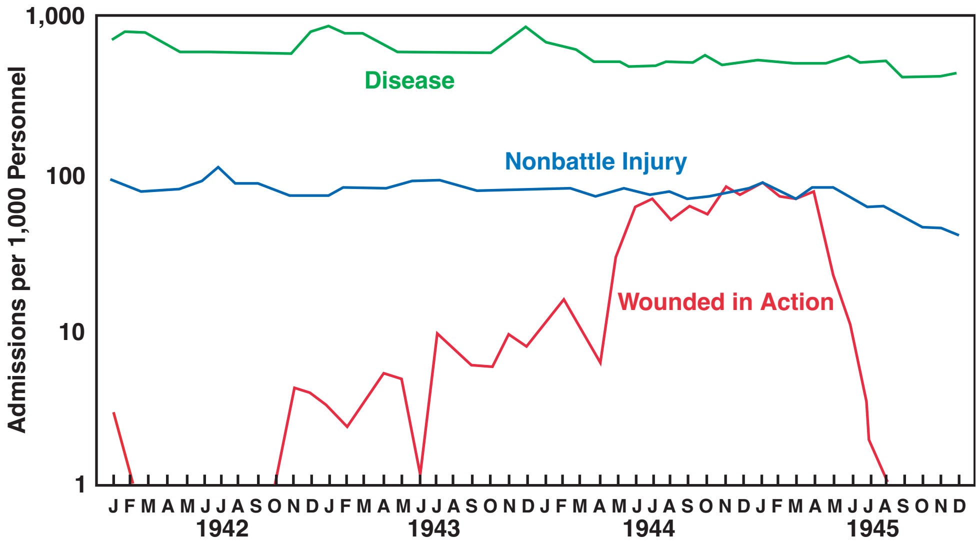

Fig. 2-1. US Army hospital admissions by type, 1942–1945. These data, which indicate *morbidity* as opposed to *mortality*, reveal that DNBI causes significant combat ineffectiveness, even during periods of sustained fighting. Footnote: Rates expressed as admissions per 1,000 average strength per year. Adapted from: Reister FA, ed. *Medical Statistics in World War II.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1975: page facing title page. Prepared by Judith Bowles.

Throughout history, some commanders have taken care to reduce DNBI while others have not. This chapter presents historical examples that illustrate the impact of preventive medicine on warfighting. The goal is to instill in medical and line officers alike an appreciation of the critical importance of preventive medicine efforts during war. These eight vignettes—four that demonstrate the problems associated with ignoring preventive medicine and four that illustrate the benefits of prevention—cover a variety of threats: environmental, communicable, and vector-borne. All are drawn from the 20th century to take advantage of its better science, data, and reporting.

THE COSTS OF IGNORING PREVENTIVE MEDICINE PRINCIPLES

Typhoid Fever in British Troops During the Second Boer War

Setting

Field Marshall Lord Frederick Roberts took command of British forces in South Africa (Figure 2-2) in January 1900 in an effort to reverse Britain's fortunes in the field and defeat the Boers.[7] To relieve the sieges of Ladysmith, Kimberley, and Mafeking and to stop rebellion in the Cape Colony, Roberts planned to seize the republican capitals of Bloemfontein, in the Orange Free State, and Praetoria, in the Transvaal.[8] Between 11 February and 13 March, Roberts' forces marched east from the Western Railway Station on the Modder River, heading for Bloemfontein. This drive, along with General Sir Redvers Bullers' push against Ladysmith in Natal, caused the Boers to retreat, giving up Ladysmith, Kimberley, and Bloemfontein. Hopes ran high that the war would end quickly.[7] Unfortunately, the British bivouacked downstream when they surrounded the typhoid-infected Boer garrison at Paardeberg. The British force's sole source of water, the Modder River, was saturated with Boer refuse.[9] When Roberts seized Bloemfontein, his main supply line was 750 miles long. His march to the capital had exhausted his supply of rations, horses, and equipment of all kinds. And while the British forces awaited supplies, typhoid fever joined their ranks.[7]

Health Issues

Field Marshall Roberts encountered four major health issues during his campaign: troop immuni-

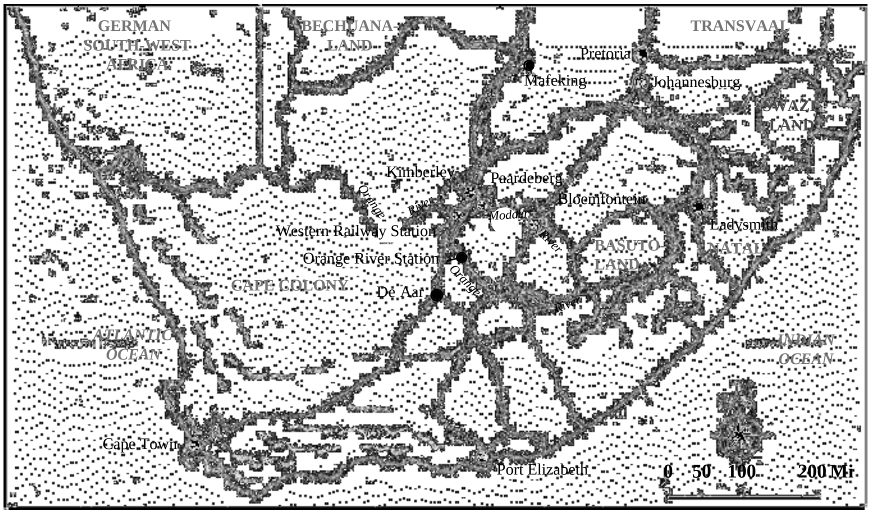

Fig. 2-2. South Africa, circa 1900. Rail lines are shown by hatched solid lines. Field Marshal Lord Roberts began his flank march to Bloemfontein from Western Railway Station on 11 February 1900 with an army corps of 40,000 men and 100 guns. By the time he reached the Free State capital, his troops were weary, poorly supplied, and becoming ill with typhoid fever. Adapted with permission from: Pagaard S. Disease and the British Army in South Africa, 1899–1900. *Mil Affairs*. 1986;Apr:71.

zation, sanitation measures, water supply and sanitation, and general medical support (Figures 2-3 and 2-4). Each was mishandled and compounded the effects of the other three.

Typhoid fever was a recognized threat to soldiers in the tropics before the South African deployment.[9] Sir Almroth Wright's anti–typhoid fever inoculation appeared promising from results obtained from troops in India and Egypt,[10,11] but vaccine side effects and uncertain efficacy precluded compulsory inoculation of the army.[11] Voluntary inoculation was permitted by the War Office in the fall of 1897.[10] Inoculation was offered to troops en route to South Africa, but less than 5% of the total force received the vaccine.[7] Using 5% as an estimate, only 1,700 of Roberts' 34,000 soldiers were inoculated.

Field sanitation had been dismissed as a "fad" and sanitary officers as "useless"[12p529] by the Commander-in-Chief, Lord Wolseley, in 1886. Those holding this viewpoint overruled a suggestion by a member of Parliament in October 1899 to create a special sanitary commission for the South African campaign. Roberts' only guidance in field sanitation came from the meager training his medical officers had received.[9]

Roberts was handicapped from the beginning of the campaign by his inability to supply his army with enough clean drinking water. There were insufficient numbers of the standard wooden barrel water carts, and their design kept them from following troops over rough terrain. In addition, the insides of these carts and individual canteens were squalid and quickly covered with mold. As one colonel reported later: "Probably nothing harbours germs and disease more than our present type of wooden barrel water cart."[7p242]

Fig. 2-3. British soldiers receiving first aid, South Africa, circa 1900. Command-directed water rationing proved intolerable to soldiers in the South African heat. Many soldiers filled their water bottles directly from the typhoid-laced Modder River and ignored orders to boil water before drinking. Reproduced with permission from: Pakenham T. *The Boer War*. New York: Random House, 1979: between pages 234–235. Published in Great Britain by Wiedenfeld and Nicolson.

Fig. 2-4. British dressing station on the Modder River, South Africa, circa 1900. Filthy, inadequate treatment facilities such as this were the only accommodations available for many sick troops. Reproduced with permission from: Pakenham T. *The Boer War*. New York: Random House, 1979: between pages 234–235. Published in Great Britain by Wiedenfeld and Nicolson.

From the beginning of the war, the Royal Army Medical Corps had been inadequate for the needs of the large force deployed. In March 1900, there were only 800 physicians to care for the 207,000 British troops in South Africa. Roberts began his march with 10 small hospital units and ten bearer companies. These medical assets could only accommodate 4% of his army, or 1,360 patients, simultaneously.[7] After 4 weeks of campaigning and combat, medical provisions were low and, with his long supply line, it would take time to restock them.

Roberts' Actions

Roberts recognized his water problems early. He rationed his soldiers to half a water bottle per day and ordered from India 2,000 water carriers with goat and ox skin waterbags.[7,13] Standing orders to boil water existed but were often ignored because of lack of fuel and the soldiers' dislike of the "insipid taste."[7p242]

As early as February 1900, Roberts was aware of the typhoid fever problem in hospitals along the Modder River. He visited hospitals at De Aar and Orange River Station, finding them "as bad as he feared."[13p404] There were not nearly enough orderlies or nurses to deal with the situation. He responded by sending for 20 additional nurses but later changed this figure to 50 for all of South Africa. Roberts had no confidence in his Surgeon-General, W. D. Wilson, and commented that Wilson "does not seem to have any idea of what is required"[13p404]; however, Roberts did not relieve him.

Roberts' medical problems were compounded by "Boer daring" and "British military negligence"[9p74] when the enemy seized the waterworks at Sanna's Post on 1 April. Bloemfontein wells, of doubtful cleanliness at any time, were the British Army's only source of water until Sanna's Post was retaken 3 weeks later. This situation set the stage for a second typhoid fever epidemic, which erupted during the second week of May.[9]

Roberts failed to act promptly during the early stages of the initial epidemic. It was not until late April 1900 that he requested 300 orderlies and 30 doctors from England.[13]

Results

Roberts' unrealistic water ration for soldiers who required at least a quart and a half per hour, compounded by his inability to provide clean water in sufficient quantities, led to thirsty soldiers obtaining water from any source available and drinking it directly. On 23 March, 10 days after the British entered Bloemfontein, the first typhoid fever epidemic had 1,000 soldiers in the hospital. By 1 June, the second epidemic had increased this number almost 4-fold. Roberts' medical staff, supplies, and facilities were overwhelmed. Two general military hospitals, Numbers 8 and 9, and three private hospitals were not enough to treat all the cases. Public buildings, schools, and convents received the overflow.[7,14]

Doctors, nurses, and supplies were rushed to South Africa but not before word of the epidemic and its mismanagement had caught the attention of the British public and Parliament. Despite Roberts' attempt to explain the situation by stating that "…a certain amount of suffering is inseparable from the rapid advance of a large army in the enemy's country, when railway communication has been destroyed…,"[7p246] a royal commission was convened to investigate. While patients and civilian physicians proclaimed universal medical mismanagement, the officers and nurses of the Royal Army Medical Corps closed ranks, declaring that things were really not that bad. The royal commission sided with those in uniform, saying that overall the campaign

> '…has not been one where it can properly be said that the medical and hospital arrangements have broken down…. [There was] no general or widespread neglect of patients, or indifference to suffering.'[7p248] The commission's report gave only passing notice to sanitation and made no mention of immunization, concluding 'we do not consider that the great outbreak of enteric fever, or any considerable part of it, was due to preventable causes.'[9p75]

Impact

Roberts lost his momentum at Bloemfontein. Hamstrung by a long logistics train, his drive to Praetoria was delayed while he waited on supplies, horses, and men. Cape Town was reinforcing his army from one end while typhoid fever was depleting it from the other. He could not even be sure his reinforcements were healthy as they traveled the same road to Bloemfontein that he had weeks earlier.[7] When Roberts marched out of the city on 3 May bound for Praetoria, he found an enemy rejuvenated by a new leader, Christian deWet, and ready to continue the war.[7,13]

The typhoid epidemic also caused the medical world to reevaluate the efficacy of anti–typhoid fever inoculation. From June to November 1900 and February to March 1901, Dr. Dodgson compared the incidence of typhoid fever among the uninoculated and inoculated noncommissioned officers and men at General Hospital Number 8. In the 110 uninoculated, he found an incidence rate of 40/100. In the 21 inoculated, it was 24/100. Although his numbers were small and his data collected as the epidemic was burning out, Dodgson was struck by the magnitude of disease among the inoculated. He concluded that "Unless some system of prevention can be devised which will give better results than this, it is difficult to see that it can be of very great use."[15p251] Dodgson also reviewed a number of military hospitals across South Africa and determined that "there is little to distinguish the inoculated from the uninoculated cases, as regards death-rate, severity of disease, the incidence and severity of complications, the differences being so small as to easily come within the errors of observation."[15p254]

Anti–typhoid fever inoculation was discontinued after the war. It was not resumed until 1904, again on a voluntary basis, after a reevaluation of inoculation data from British forces in South Africa and repeated immunogenicity studies proved its value.[10,11]

Conclusion

Due to a lack of command emphasis on known preventive measures, the British army in South Africa suffered a medical disaster. Inadequate planning, along with inappropriate immunization and sanitation procedures, fostered two typhoid fever epidemics. The epidemics delayed the British drive to Praetoria, caused needless suffering for British soldiers, and produced a public scandal for the Royal Army Medical Corps.

British Experience with Malaria in Salonika, Greece, During World War I

Setting

Early in the 20th century, expanding colonialism and nationalism lead to tension across Europe. Alliances that were established to regain some sense of security only served to divide Europe into two armed camps: the Triple Alliance of Germany, Austria, and Italy and the Triple Entente of France, Russia, and Britain. The brittle and unstable peace was shattered with the assassination of Austro-Hungarian Archduke Ferdinand on 28 June 1914. World War I began on 4 August.[16]

By the beginning of 1915, the war had come to an operational stalemate. Opposing forces on the

Western Front had settled into fixed fortifications, linked by a maze of trenches and separated by an expanse of desolate land. The tedium of trench warfare, highlighted by artillery bombardment and the occasional battle, became the order of the day.[16] Independently, British and French strategists determined that a thrust through the Balkans, an area undefended by the Triple Alliance, would be the key to breaking the stalemate. British supporters of the plan, led by Lloyd George, Chancellor of the Exchequer, proposed an attempt to unite the Balkans against Turkey and to relieve pressure on the Russians, while at the same time aiding the Serbs struggling with Austria. A complicated series of events involving British and French military dissent, chaotic Greek politics, and Winston Churchill's plea for naval intervention in the Dardanelles delayed a decision on the Balkans and produced the debacle at Gallipoli. Finally, the Secretary of State for War, Field Marshal Lord Kitchener, and the French government reluctantly agreed to deploy troops. On 5 October 1915, elements of the British 10th Division and the French 156th Division disembarked at Salonika (now Thessaloniki), Greece.[16,17]

Health Issues

The malaria threat in the Balkans was known to the British. Arriving after the malarial season, British medical authorities had time to reconnoiter the terrain and plan an anti-malaria campaign for 1916.

The British originally camped in an area that was "a continuous series of hills and valleys"[18p227] south of Lake Langaza, east of the Galiko River, and west of Salonika along the Monastir Road. The malaria vectors *Anopheles superpictus* and *A maculipennis* held the high and low ground, respectively. After a wet winter and spring, large numbers of these mosquitoes, well fed on the malarious population of the region, would be ready to infect nonimmune British troops with *Plasmodium vivax* and *P falciparum*.[18]

Preventive measures in the British armamentarium consisted of prophylactic medication, insect repellents, mosquito destruction, and other personal protective measures. Quinine prophylaxis was not a panacea (Figure 2-5). Insect repellent development was in its infancy; the repellents on hand were ineffective and were considered a waste of money. Therefore, the plan to reduce the mosquito threat and thereby the incidence of malaria was based on mosquito destruction and personal protective measures.[19]

In the spring of 1916, mosquito destruction was implemented by anti-malaria squads organic to corps and divisional units. The squads destroyed adult mosquitoes; destroyed ova, larvae, and pupae using disinfectants; applied oil to standing water; and drained breeding sites (Figure 2-6). Collective and personal protective measures included appropriate camp placement, screening and bed netting, and clothing (eg, head nets, gauntlet gloves, flapped shorts [Figure 2-7]).[19]

Military Actions, 1916

In late May 1916, Bulgarian infantry invaded Greece and took up positions in the Rupel Pass northeast of Lake Butkova. From this vantage point, the Bulgarians threatened the Struma Valley. French General and theater commander Maurice Sarrail

Fig. 2-5. "Quinine Parade." British soldiers in Macedonia taking quinine—under supervision—as prophylaxis against malaria, circa 1916. The use of quinine prophylaxis had been debated before the war, but no consensus on its value had been reached. Official histories state that quinine was "extensively used," but of 129 medical officers surveyed during the war, only 11% thought it had value while 75% reported it had very little or no value.
Source: McPherson WG, Horrocks WH, Beveridge WWO, eds. *Medical Services: Hygiene of the War*. Vol 2. In: *History of the Great War*. London: His Majesty's Stationery Office; 1923: 212–219. The quotation is from page 216. Photograph reproduced from: McPherson WG, Mitchell TJ, eds. *Medical Services: General History*. Vol 4. In: *History of the Great War*. London: His Majesty's Stationery Office; 1924: 106.

Fig. 2-6. A British work party improving drainage ditches in Macedonia, north of Salonika, during World War I in the effort to eliminate mosquito breeding sites. This was part of a larger plan to prevent malaria.
Reproduced from: McPherson WG, Horrocks WH, Beveridge WWO, eds. *Medical Services: Hygiene of the War*. Vol 2. In: *History of the Great War*. London: His Majesty's Stationery Office; 1923: 231.

directed British General George F. Milne to move his forces forward to occupy the entire Struma Valley and a line from Lake Butkova to Lake Doiran and then west to the Vardar River north of Smol.[17,18]

Sarrail was anxious for an offensive but met continued resistance to the idea from the British. In July, Sarrail informed Milne that he would attack the Bulgarians with or without British support. Milne cleared his troops from Sarrail's line of advance, moving them into the southern Struma River valley.[17]

These unanticipated moves, coming at the beginning of the malaria season, put British forces in a highly malarious area with no antimosquito preparation.[18] Working parties began mosquito destruction activities in this new area, but they "were never very satisfactory, owing to the vast amount of water and the changing courses of the streams, which constantly formed new pools."[20p288] In addition, bed nets, although recognized by General Milne to be "as important as the rifle,"[20p7] were constantly in short supply.[19] Likewise, screens for huts and tents

Fig. 2-7. This British soldier is wearing the complete personal protective kit issued by his command, including head net, gauntlets, and flapped shorts. The flaps folded down to cover the knees and then were inserted into puttees. Reproduced from: McPherson WG, Horrocks WH, Beveridge WWO, eds. *Medical Services: Hygiene of the War*. Vol 2. In: *History of the Great War*. London: His Majesty's Stationery Office; 1923: 220.

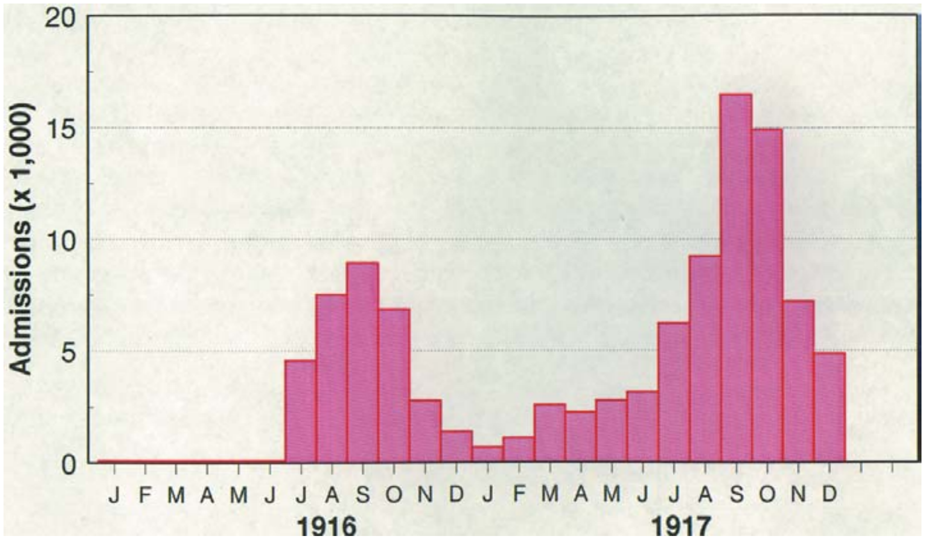

Fig. 2-8. Malaria incidence by month in the British Salonika force, 1916–1917. The rapid increase in malaria cases in 1916 came after the British had moved into positions in the Struma Valley. Relapsing and recrudescing cases are noted throughout the winter and early spring of 1916-1917. These cases provided a nidus of infection for the following year and contributed to the greater incidence of malaria in the late summer of 1917. Adapted from data on pages 108-109 in: McPherson WG, Mitchell TJ, eds. *Medical Services: General History*. Vol 4. In: *History of the Great War*. London: His Majesty's Stationery Office; 1924: 108-109. Prepared by Judith Bowles.

were pushed to the end of the logistics train in favor of what was felt to be more essential equipment.[20]

Results

Once in their new positions, British soldiers began to contract malaria at an alarming rate, despite regular dosing with quinine. By the end of July, the 10th Division was suffering 100 to 150 malaria casualties per day. A number of units with critically low troop strengths were combined to maintain combat effectiveness.[17,20] From 1 May to 31 October 1916, the malaria hospital admission rate was 237/1,000 strength, but many more soldiers were treated at field ambulances and in their unit areas. A total of 30,000 cases were recorded for 1916 (Figure 2-8). While most cases were caused by *P vivax*, enough were caused by *P falciparum* to generate a mortality rate of 1%.[18]

Blood smear examinations, performed during the winter of 1916–1917 on soldiers not then manifesting malaria, demonstrated that asymptomatic cases existed. From November 1916 to April 1917, admissions from relapsing (*P vivax*) and recrudescent (*P falciparum*) disease occurred at a rate of 57/1,000 strength. This reservoir of infected soldiers, along with the native population, would provide the nidus for the next malaria season.[18]

Military Actions, 1917

In April and May of 1917, Allied forces launched a poorly coordinated offensive against the Bulgarian Army. Casualties were high and little was gained. To suppress Allied operations in Macedonia, the Germans increased submarine activity in the Mediterranean, thus slowing the flow of supplies and halting hospital ship operations completely.[20,21] Demoralized, the Allies in Salonika resumed a defensive posture. Milne, fully aware the malaria season was now upon him, withdrew his troops from the Struma valley, continued the anti-mosquito campaign, and waited for bed nets.[17,21]

As summer gave way to autumn in 1917, it became apparent to the British high command that operations on the Balkan front were having no impact on the outcome of the war. Furthermore, British operations in Palestine needed immediate reinforcement. Units were transferred to Palestine and Egypt, and, in 1918, to the Western Front.[17,21]

Results

Although the British evacuation probably reduced primary malaria cases, the rate of admissions between May and October 1917 was 278/1,000 strength, exceeding that of the previous year. By the end of 1917, another 70,000 admissions had been recorded (see Figure 2-8). Due to improved treatment and ground evacuation, the mortality rate dropped to 0.37%.[18]

With the termination of hospital ship evacuation, a population of some 15,000 chronic malaria cases was created,[21] which ambulated from hospitals to convalescent stations "with an occasional day or two of light duty."[21p58] Sir Ronald Ross was dispatched to the area in December, and through his recommendations, a troop evacuation and exchange program was initiated.[21] Large-scale evacuation of sick troops to England and France began in January 1918.[19]

Impact on Operations

Malaria incapacitated entire battalions of the British Expeditionary Force from the late spring of 1916 to the close of the war.[17,19,20] By the end of 1916, 17% of British troops had been infected, and by the end of 1917 this figure had risen to 39%.[18] Allied disunity underlay the failed 1917 spring offensive; however, malaria contributed through its effects on the endurance and efficiency of the soldiers involved.[18,20]

As they retired from the Balkan front en route to France, some units left a trail of debilitated soldiers along the way. Other units made it to the Western Front, only to have recurrent disease keep them from entering the line. These troops required special treatment and rest, burdening medical and supply operations, but they finally did return to duty.[21]

Conclusion

On their arrival in Salonika, the British used their experience and knowledge to counter the malaria threat. Their failure in this endeavor resulted from operational, medical, and supply decisions that, to some extent, were beyond the control of the local commander, medical staff, and quartermaster.

First, Milne was directed by Sarrail to reposition British forces in a highly malarious area before anti-mosquito measures could be implemented. Shortly thereafter, Milne was forced, by pressure from London to keep Britain in a defensive posture, to shift his forces again in the same malarious region. These unexpected moves during the height of mosquito season kept Milne's anti-mosquito squads from establishing effective vector control, resulting in intense exposure of long duration.

Second, the prophylactic use of quinine was not the standard of care. Historical accounts state that quinine was used extensively; however, it is difficult to believe that many prophylactic doses were given, with the majority of medical officers perceiving it to have no preventive efficacy, the ever-increasing numbers requiring treatment, and the slow rate of resupply.[19] Additionally, quinine has no effect on the exoerythrocytic stages of malaria. *P vivax*, which accounted for the vast majority of cases, has an obligatory exoerythrocytic stage during its life-cycle and thus can produce relapse if treatment consists only of a blood, not a tissue, schizonticide.

Third, without effective insect repellents, personal protective measures consisted of mechanical barriers. The most effective of these, the bed net, was continually in short supply because of enemy submarine interference with shipping operations, and, more importantly, because of its low priority with commanders and logisticians at all levels. If bed nets had had higher priority and had enough of them accompanied the initial deployment, then resupply difficulties would have had less impact.

Historian Cyril Falls concluded:

> Had it from the first been possible to decide that in Macedonia protection from malaria was, after food and ammunition, the very first necessity, it is reasonable to suppose that the Salonika Army might have been kept at a higher standard of strength and efficiency, that a certain number of lives might have been saved, and that many thousands of men might have been spared ill health after the war.[20p288]

Cold Injury in the US Army during World War II

Setting

In heavy fog on 11 May 1943, the US 7th Infantry Division made an amphibious assault on Attu Island, Alaska, (Figure 2-9), westernmost island in the Aleutian chain.[22,23] Landing at Holtz Bay in the north and Massacre Bay in the south, the Americans met light resistance until they pushed inland.[22,24] The 2,500 Japanese defenders had strongly fortified the mountainous terrain. Heavy artillery and automatic weapon fire, along with semi-frozen mud, rain, and fog, stopped the US advance.[23,24] The "bewildered 7th, ill-led, badly trained, and having its first experience in combat, went to pieces."[23p500] The Division commander was relieved of command, and 4,300 more troops landed to bolster the 11,000 already engaged. The Japanese, their food and ammunition dwindling, launched suicide attacks on 29 and 30 May, concluding the debacle and ending their occupation of North America.[22,24]

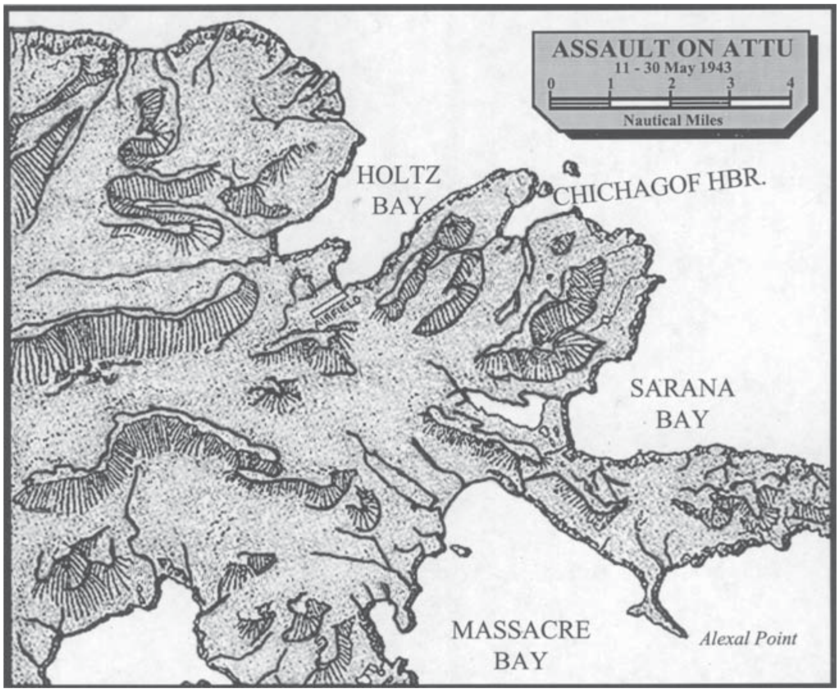

Fig. 2-9. Attu Island, Alaska, 1943. Adapted from: Morrison SE. Aleutians, Gilberts, and Marshalls, June 1942-April 1944. In: *History of United States Naval Operations in World War II*. Vol 7. Boston: Little, Brown & Co; 1951: 45.

Health Issues

At the end of the First World War, the US Army had considerable knowledge and experience in cold weather campaigning on the Great Plains during the Indian Wars and in Europe during World War I.[22,25–29] Between the world wars, the army forgot about cold injuries and their impact on troops in the field. Colonel George Dunham's *Military Preventive Medicine,* published in 1940, did not have a word on cold injuries. The *Medical Department Soldier's Handbook* (TM 8-220), 1941, failed to include trench foot, and *The Guide to Therapy for Medical Officers* (TM 8-210), 1942, not only excluded trench foot but also advised troops to lace shoes snugly, thereby decreasing circulation. American forces went to war poorly indoctrinated on cold injuries and inadequately clothed to operate in such environments.[25] The hierarchy in line, medical, and logistics commands failed to learn the lessons of history, lessons replete with cold disasters and valid preventive measures.

Contributory Actions and Inactions

The reasons for this environmentally induced disaster can be found at major and local command levels. Before the war, the army failed to develop functional cold weather protective clothing for general troop issue.[22,25,30] During the planning phase of the Attu Campaign, the Quartermaster Corps recommended the use of special clothing and footgear, but commanders did not heed this advice.[22] Troops went ashore on Attu without properly insulated and windproofed and waterproofed clothing. The more comfortable but less protective leather boot was employed instead of the shoepac, a warmer, commercially made boot with a moccasin-style rubber foot and a leather top.[22,25,30,31] Commanders and soldiers were poorly trained and ill equipped to live and fight in cold, wet climates. Foot hygiene was not enforced. Soldiers did not change wet boots and socks for days and frequently discarded wet clothing rather than dry it out. In addition, the logistics system failed in that sleeping bags were not issued for the first 4 to 5 nights after landing.[22,25,30]

Impact on Army Operations

In 22 days of combat, the 7th Infantry Division sustained 3,829 casualties of which 1,200 (31%) were due to the cold.[22] Cold injury and wounded-in-action rates (Table 2-2) were virtually the same. Although this initial experience with cold injury in combat demonstrated the necessity of proper equipment and clothing, the importance of soldier training regarding foot care, and the importance of command responsibility in enforcing foot care discipline, it did not generate rapid corrective procedures.[25] During the Italian Campaign in the winter of 1943–1944, the 5th US Army sustained 5,752 cases of trench foot, with an admission rate of 54/1,000 average strength. The ratio of trench foot to nonfatal battle

TABLE 2-2

BATTLE CASUALTIES AND ADMISSIONS FOR DISEASE AND NONBATTLE INJURY, ALLIED TROOPS, ATTU CAMPAIGN, 11 MAY THROUGH 1 JUNE 1943.

Cause	Number	Rate*
Battle casualties		
Killed in action	549	1.6
Wounded in action	1,148	3.4
Nonbattle admissions		
Disease	614	1.8
Nonbattle injury	1,518	4.5
Cold injury	(1,200)[1]	(3.6)†
Other	(318)[1]	(0.9)†
All causes	3,829	11.4

*Rate expressed as number per day per 1,000 average strength
†Figures in parentheses are subtotals
Source: Whayne TF, DeBakey ME. *Ground Injury, Cold Type*. Washington, DC: Office of The Surgeon General, Department of the Army; 1958: 85.

injury was 1:5 (Table 2-3).[22] Additionally, clothing was still inadequate and in short supply, troops lived on cold rations, and few had received cold weather training before deployment.[25,30]

Cold weather indoctrination and training improved during the summer of 1944. Field commands were apprised of training materials and directed to provide appropriate training and enforce foot hygiene. During the Italian Campaign of 1944–1945, woolen socks and hot rations were supplied and foot hygiene was enforced. Improved shoepacs reached the front by October 1944; however, there was resistance among soldiers to using them because of sizing difficulties and lack of training.[25,30] When the 5th Army Quartermaster realized that 80% of cold casualties in Italy were not wearing shoepacs, he developed and implemented training on the supply and use of winter clothing.[25] The trench foot admission rate dropped to 20/1,000 average strength during the winter of 1944–1945, demonstrating the success of preventive efforts.[22]

The US experience with cold environments in the Aleutians and Italy did not translate into adequate prevention in the European Theater during the war's last winter. The European Command expanded rapidly after June 1944 and planned for a quick victory as the 3rd Army drove through France. The Theater Quartermaster General downgraded the priority of winter clothing, ignoring the Italy experience and preventive medicine advice to the contrary. Poor planning and delayed requisitions resulted in soldiers fighting through the severe winter of 1944–1945 inadequately clothed and shod.[22,25,30] The Theater Adjutant General disapproved a preventive medicine publication to edu

TABLE 2-3

CASES OF TRENCH FOOT (HOSPITALIZED AND GIVEN QUARTERS) AND OF BATTLE INJURIES AND WOUNDS, BY MONTH, FIFTH US ARMY, NOVEMBER 1943 THROUGH APRIL 1944.

	Trench foot		Battle injuries and wounds		
Month and year	Cases	Rate*	Cases	Rate*	Ratio of trench foot to battle injuries and wounds
1943					
November	371	24	3,897	249	1:11
December	1,265	69	5,020	274	1:4
1944					
January	1,490	96	4,496	289	1:3
February	1,805	108	8,378	500	1:5
March	779	35	3,685	167	1:5
April	42	2	2,126	121	1:51
Total	5,752	54	27,602	261	1:5

*Rate expressed as number per annum per 1,000 average strength
Adapted from Whayne TF, DeBakey ME. *Ground Injury, Cold Type*. Washington, DC: Office of The Surgeon General, Department of the Army; 1958: 103.

Fig. 2-10. Infantrymen from the 9th Regiment, 2d Division, 1st US Army take cover from German artillery during the Battle of the Bulge. An unusually heavy snowfall and intense cold accompanied the German offensive in December 1944. These conditions made combat operations difficult, strained an already overworked logistics system, and induced an epidemic of cold injuries among US troops.Reproduced from: Whayne TF, DeBakey ME. *Ground Injury, Cold Type*. Washington, DC: Office of The Surgeon General, Department of the Army; 1958: 137.

cate commanders on general foot care because the subject was covered in existing manuals. Not until late November did the Command Surgeon provide official guidance on this issue. By that time, the 3rd Army, advancing on Metz, had sustained over 6,200 cold injuries. Not uncommonly, units lost 10% to 15% of their strength. In the first 4 days of the Lorraine Campaign in November 1944, the 328th Infantry Regiment evacuated 500 cold casualties and was rendered combat ineffective.[22,25] In mid-December, the Germans launched a counteroffensive, the Battle of the Bulge (Figure 2-10). Heavy fighting in extreme cold generated a second epidemic of cold injuries in US forces.[22]

There were 46,107 hospital admissions for cold injuries in the European Theater between October 1944 and April 1945.[25] (The figures for the 5th US Army are in Table 2-4.) Fifty percent of these cold casualties, roughly equivalent to one and a half infantry divisions, occurred during November and December 1944.[22] The magnitude of these casualties stimulated action at the general-staff level. Cold

TABLE 2-4

CASES OF TRENCH FOOT (HOSPITALIZED AND GIVEN QUARTERS) AND OF BATTLE INJURIES AND WOUNDS, BY MONTH, 5TH US ARMY, OCTOBER 1944 THROUGH MARCH 1945.

	Trench foot		Battle injuries and wounds		Ratio of trench foot to
Month and year	Cases	Rate*	Cases	Rate*	battle injuries and wounds
1944					
October	258	24	8,404	783	1:33
November	274	25	2,046	188	1:7
December	305	22	1,274	90	1:4
1945					
January	309	26	561	48	1:2
February	324	25	1,966	154	1:6
March	102	6	1,613	97	1:16
Total	1,572	20	15,864	206	1:10

*Rate expressed as number per annum per 1,000 average strength
Adapted from Whayne TF, DeBakey ME. *Ground Injury, Cold Type*. Washington, DC: Office of The Surgeon General, Department of the Army; 1958: 1034.

injury prevention received a high priority during planning for the invasion of Japan. Medical personnel taught soldiers the importance of foot hygiene, proper footgear, and nutrition. The Quartermaster Corps geared up to provision the invasion force for cold weather operations. And most importantly, commanders were made responsible for cold injuries among their troops.[25]

Conclusion

The Aleutian Island campaign clearly demonstrated the US Army's unpreparedness for cold weather operations. The European campaigns illustrate how difficult it can be to correct such deficiencies in a large field army. During World War II, the Army only gradually relearned the value of planning, training, logistics, and awareness (both command and medical) in cold weather operations.

Fig. 2-11. The principal transportation routes and terminals of the Persian corridor, World War II. The red lines are roads and the hatched solid lines are railroads. The US Persian Gulf Command operated truck and rail routes between Persian Gulf ports and northern Iranian terminals, with most of the supplies going to the Soviet Union. Reproduced from Motter THV. *The Persian Corridor and Aid to Russia. United States Army in World War II, The Middle East Theater.* Washington, DC: Office of The Chief of Military History; 1952: inside back cover.

Heat Injury in the US Persian Gulf Command During World War II

Setting

Before the US entered World War II, American leaders realized that helping enemies of the Axis powers was, in effect, self defense. This notion led to the Lend–Lease Act of 1941. Ultimately, the US and its allies established five major transportation lines that by war's end had funneled 17.5 million tons of mostly American and British supplies to the Soviet Union.[32] Through most of the war, the US allocated 20% of its production to Lend–Lease, with substantial shipments occurring during 1943 and 1944.[33]

The Persian corridor, one of the five major Lend–Lease routes to the Soviet Union, extended from the headwaters of the Persian Gulf through northern Iran. Development of this primarily British–US effort began in 1941. Allied logistics efforts here were critical in thwarting Axis designs on the Suez Canal and the oil fields of Iran, Iraq, and the Caucasus.[32,34]

Supply was the main business of the US Persian Gulf Command, which came to number 30,000 soldiers.[32] The command primarily operated truck and rail routes between the Persian Gulf ports of Basra, Khorramshahr, and Bandar Shahpur and the northern Iranian terminals of Kazvin and Tehran (Figures 2-11 and 2-12).[32–34] From 1941 to 1945, the Persian Gulf Command handled 3.9 million tons of supplies—enough to sustain 60 combat divisions—with 90% going to the Soviet Union.[32]

Fig. 2-12. Liberty ship with supplies bound for the Soviet Union at the port of Khorramshahr, Iran, WW II. The Persian corridor began at the ports of Basra, Khorramshahr, and Bandar Shahpur in the headwaters of the Persian Gulf, and the coastal port of Bushire. Here, supplies were downloaded from ships, such as the one pictured, for the journey north via truck, rail, or barge. Reproduced from: Coakley RW, Leighton RM. Global logistics and strategy, 1943–1945.*United States Army in World War II, The War Department.* Washington, DC: Office of the Chief of Military History; 1968: 677.

Health Issues

While the Persian Gulf Command did not engage in combat, the environment was enemy enough, as the region is one of the hottest in the world. Gulf coastal areas are warm and moist, with outside daytime temperatures in the summer averaging 32°C to 46°C (90°F–115°F) in the shade and humidity reaching 90%. Inland is the Iranian desert, where the climate is hotter and very dry, with temperatures ranging from 38°C to 54°C (100°F–130°F). Temperatures climb even higher inside vehicles, buildings, rail cars, and tents. The mountainous areas of Iran, such as in the vicinity of Tehran, have a more pleasant climate (Figure 2-13).[35,36]

The common scheme for classifying heat injuries, which includes heat stroke, heat exhaustion, and heat cramps, was used during World War II. Additionally, miliaria (heat rash) caused extensive morbidity and sometimes resulted in hospitalization.

During World War I, the incidence of heat injury admissions in the US Army was 1.0/1,000 soldiers per year.[35] During 1940, heat injury admissions occurred at the rate of 0.5/1,000 per year.[36] For the war years (1942–1945), there were 35,398 heat injury admissions, each lasting an average of 5.3 days, producing a rate of 1.38/1,000 per year. There were 238 deaths. Overall, however, heat injury was a relatively minor problem in World War II, except in hot regions.[35]

Fig. 2-13. Tehran, Iran, 1943. This calm street scene belies the activity of the US Persian Gulf Command. With its network of roads and rail lines, Tehran figured prominently in the command's mission and served as the location for its headquarters. Impressive amounts of supplies were shipped from the ports north through Tehran before moving east, west, and north, en route to the Soviet Union. Reproduced from: Sams CF. The Middle East countries. In: Hoff EC, ed. *Civil Affairs/Military Government Public Health Activities*. Vol 8. In: *Preventive Medicine in World War II*. Washington, DC: Office of The Surgeon General, US Department of the Army; 1976: 226.

Preventive Actions Taken and Missed

When the US entered World War II, it had not fought a major war in hot climates in nearly 40 years, and many experienced officers had left the army since World War I. Furthermore, basic science knowledge of heat stress physiology was lacking. There were misconceptions concerning hot environments. The need for exogenous salt was misapplied and overemphasized. Many believed that drinking water while working in the heat was harmful. And there was the concept of "water discipline," which held that men could be "trained" to require less water while working in the heat. While these misconceptions played some role in heat casualties, the main problem was that many known and practical principles of heat injury management were simply not followed early in the war.[35–37]

When the Middle East Theater was formed and planning begun in late 1941, there was a paucity of medical intelligence available to the command surgeon. Medical planning occurred but focused more on hospitalization and communicable disease than on heat injury prevention.[34]

Thus, when the Persian Gulf Command became operational, several problems existed—in addition to its location—that predisposed its soldiers to heat injury. Officers were inexperienced, and training for operations in hot environments was lacking. Medical planning was inadequate, as were knowledge and equipment for treating heat casualties. Preventive medicine concerns were often subordinated to logistical considerations or discarded. More specifically, operations, including desert convoys, were routinely conducted in the heat of the day. No respite was available as quarters lacked fans, evaporative coolers, or air conditioners. Night crews especially got little rest in the relentless heat of the day.[35]

Results

Heat injury was a serious problem for the Persian Gulf Command during the summers of 1942 and 1943. Indeed, this command suffered more heat-related morbidity than any other during the war. What makes this remarkable is the fact that the command *never engaged in combat operations*.[34–36]

During the first 7 months of operations, from June to December 1942, heat trauma was the second leading cause of admission (behind enteritis) to the Army hospital at Ahwaz, and heat stroke accounted for 11.7% of all admissions. The problem peaked in June 1943, when heat trauma incidence from all causes, including those hospitalized and treated as outpatients, reached 296/1,000 per year command-wide, with 8 heat-related deaths.[35] Medical treatment of heat casualties sometimes added to the problem, as initially there were no cool areas for recovering patients. Mild cases, when sent to hot wards without observation, sometimes became severe casualties within hours.[35]

Little was written specifically addressing the impact of heat trauma on the Persian Gulf Command's operations, but it was significant. No unit can suffer morbidity like that described without experiencing a decrement in effectiveness.

Conclusion

This story illustrates the advantage of operational experience in an environment, and the absolute necessity of proper planning and enforcement of preventive medicine measures. Heat injury took an unnecessarily large toll on the Persian Gulf Command during 1942 and 1943. The command, however, did eventually take specific actions to counter the heat problem: it controlled work schedules, curtailed routine operations between 1200 and 1700 hours, upgraded and cooled living quarters, and set up air-conditioned heat-casualty treatment centers at strategically located hospitals.[35,36] As a result of these measures and the knowledge gained by experience, heat-related morbidity declined sharply in 1944. The incidence of heat trauma fell from a high of 296/1,000 per year in June 1943 to 49/1,000 per year in July 1944, and then to 41/1,000 per year in August 1945. This is remarkable when one considers that a record tonnage of supplies was moved in 1944 and that the summer was just as hot as that of 1943.[35]

Because of anticipated and actual operations in desert and tropical environments, World War II spurred much hot-environment research. A team led by Dr. E. F. Adolph, working under contract for the Office of Scientific Research and Development, conducted research in various US deserts from 1942 through 1945, significantly increasing our understanding of human physiology in hot environments.[37] Applied studies were conducted at the Armored Medical Research Laboratory, Fort Knox, Ky.[35] The Army Medical Department remains actively involved in hot environment research at the US Army Research Institute of Environmental Medicine at Natick, Mass.[38,39]

Some 50 years later, from 1990 to 1991, the US military again deployed to the Persian Gulf region but in even larger numbers (696,000 personnel).[40] The Persian Gulf War involved combat as well. This time, however, command awareness was high, and preventive medicine measures were published and enforced. As a result, US forces suffered minimally from the effects of heat. While the majority of forces were deployed during the cooler months, it is still remarkable that no deaths were attributed to heat injury.[41]

The more one knows about an adversary, the less threatening it becomes, hence the importance of medical intelligence. Adolph makes this point poetically in the preface to his classic text.

> Once the desert environment is understood, it loses its mystery. The great, open desert soon grows to be a friendly place with an ever-changing beauty of shifting color and shadow…Especially at night is the desert serene and friendly; the stars stud the sky, or the landscape is flooded with moonlight.[37p vii]

THE BENEFITS OF PREVENTION

The US Army Yellow Fever Commission in Cuba, 1900–1901

Setting

The issue of Cuban independence from Spain and the US role in negotiating Spanish withdrawal from Cuba had been a thorny problem since 1895. By late April 1898, tensions had increased to a fever pitch, and President McKinley requested a declaration of war against Spain. A hastily mobilized army, commanded by Major General William R. Shafter, landed at Daiquiri, Cuba, on 22 June. Shafter realized that he had to subdue the Spaniards before yellow fever and malaria incapacitated his forces. He drove obliquely from Daiquiri to Santiago de

Cuba, forcing the capitulation of the Spanish garrison on 16 July. US forces accomplished their mission but not before tropical diseases began to decimate their ranks. With a US military government and army of occupation established on Cuba, commanders engaged a more dreaded enemy.[42]

Health Issues

During the war, US troops suffered "severely from yellow fever and other tropical diseases."[43p4] One priority of the US military government was to eradicate yellow fever from Havana. This would help maintain the health of the occupying force and, the authorities hoped, preclude the introduction of yellow fever epidemics into the US.[43]

Medical authorities agreed that because yellow fever was a filth disease, appropriate sanitation in cities such as Havana would eliminate the problem. With this in mind, Major William C. Gorgas, Chief Surgeon for the city of Havana, began an intense cleaning program for Havana and other Cuban cities.[43,44] These efforts reduced the cases of typhoid fever and dysentery, and the general death rate declined, but yellow fever remained unabated.[43–45] Epidemics broke out in Havana and other Cuban cities in 1899 and 1900, fueled by nonimmune from the United States and a growing number of Spanish immigrants attracted by the economic opportunities of the newly stable political situation.[42,43,46] Frustration mounted, along with yellow fever cases, as the year 1900 progressed. Major General Leonard Wood, the new governor-general and a physician himself, appointed Gorgas Chief Sanitary Officer of Havana, continued to fund the cleaning efforts, and prodded Army Surgeon General George Sternberg to appoint a special medical commission to investigate the etiology of yellow fever.[47] Whether in reaction to Wood's urging, to discussions with Major Walter Reed, or to criticism of his management of the typhoid fever epidemic in the mobilization camps, Sternberg ordered the creation of a medical board, The Second Havana Yellow Fever Commission, to convene at Columbia Barracks, Quemados, Cuba. This board was to investigate "acute infectious diseases prevalent on the island of Cuba" and "give special attention to questions relating to the etiology and prevention of yellow fever."[48]

Actions of the Yellow Fever Commission

Reed presided over the board, which consisted of himself and Drs. James Carroll, Jesse Lazear, and Aristides Agramonte (Figure 2-14).[48,49] Reed and Carroll were intimately familiar with the prevalent theory that the *Bacillus icteroides* of Dr. Giuseppe Sanarelli was the yellow fever agent. They had done much to disprove it, although had not conclusively done so.[50] In late June 1900, the Board initiated bacteriological studies using blood from yellow fever patients and blood and organs from deceased victims, in an effort to isolate *B icteroides*. These studies yielded negative results.[48]

When the Board arrived in Cuba, a yellow fever epidemic was in progress at Quemados, making it a compelling and convenient subject for immediate investigation.[48] During the last week of July 1900, a yellow fever epidemic occurred in the military barracks at Pinar del Rio. This epidemic was misdiagnosed as pernicious malarial fever and no disinfection of fomites occurred. The Board noted that this omission of fomite disinfection did not increase the number of cases, nor did any of the nonimmunes who slept in beds of the sick or handled contaminated clothes become ill. This agreed with earlier observations that nonimmunes who cared for yellow fever patients or came in contact with them during early convalescence did not contract the disease. Reed knew of Dr. Carlos Finlay's old and unproven theory that yellow fever was transmitted by the *Aedes aegypti* mosquito and Dr. Henry R. Carter's recent studies concerning the time interval between primary and secondary infection. In addition, he observed the seasonal nature of both yellow fever and malaria and felt it reasonable that yellow fever might require a special agent for its transmission.[46] Reed may not have regarded Finlay's theory any more highly than Sternberg or Gorgas did. But when considered in conjunction with Carter's observations, seasonal variations, and the noncontagious nature of the epidemic at Pinar del Rio, he considered the idea of an intermediate host as a possibility. Reed obtained mosquito eggs from Finlay for cultivation and redirected the focus of the Board toward disease transmission.[48]

In early August, Reed was temporarily recalled to Washington. He directed the Board to begin experiments on human subjects. Lazear and several others failed to become ill after being bitten in mid-August.[44,48] On 27 August, Carroll allowed himself to be bitten by a mosquito that had fed on a yellow fever patient 12 days before. Three days later he was gravely ill. Carroll admitted to being in infected areas just before being bitten, however. To definitely prove what they now suspected, Lazear and Agramonte needed a nonimmune volunteer who

Key: (1) Dr. Carlos Finlay, Cuban physician who originated the theory of mosquito transmission of yellow fever; (2) Major Walter Reed, President of the Yellow Fever Board; (3) Dr. Jesse W. Lazear, Yellow Fever Board member; (4) Dr. James Carroll, Yellow Fever Board member; (5) Dr. Aristides Agramonte, Yellow Fever Board member; (6) Major General Leonard Wood, Governor-General of Cuba; (7) Major Jefferson R. Kean, Chief Surgeon, Dept. of Western Cuba; (8) Lieutenant Albert E. Truby, Commander, Columbia Barracks Post Hospital; (9) Dr. Roger P. Ames, clinician; (10) Dr. Robert P. Cooke, Contract Surgeon and experimental volunteer; (11) Private John R. Kissinger, Hospital Corps, experimental volunteer; (12) John J. Moran, Acting Steward, Hospital Corps, experimental volunteer; (13) Private Warren G. Jernegan, Hospital Corps, experimental volunteer; (14) an American, representative of eleven additional volunteers; (15) a Spanish immigrant, representative of four additional volunteers. Data source: *Conquerors of Yellow Fever*, American Medical Association, Copyright 1941.

Fig. 2-14. "Conquerors of Yellow Fever," by Dean Cornwell, 1941. With Major Walter Reed and Dr. Carlos Finlay among the spectators, Dr. Lazear inoculates Dr. Carroll with an infected mosquito on August 27, 1900. This experiment indicated that the mosquito was the carrier of yellow fever. The painting includes portraits of many of the men whose combined efforts made this great achievement possible. Reproduced with permission of Wyeth-Ayerst Laboratories.

had not been out of camp. On 31 August 1900, Private William H. Dean of B Troop, 7th Cavalry, stated that he met the requirements and offered his arm. Five days later Dean became the first proven case of yellow fever experimentally produced by infected mosquitoes.[48,49] Convinced that mosquitoes transmitted yellow fever, the Board ceased further human experimentation.[49] Carroll and Dean survived, but, regrettably, Lazear was accidentally bitten in mid-September and died on the 25th.[44]

Reed returned to Cuba the first week in October to find a devastated Board and a depressed medical staff at Columbia Barracks.[44] Studying Lazear's notes, he became convinced that Finlay had been correct but felt the theory must be proven beyond a shadow of a doubt. He produced a preliminary report of the Board's results and persuaded Wood to fund an "experimental sanitary station"[46p204] where controlled experiments could be conducted to exclude any other sources of infection.[44,46,51]

Reed designed and oversaw construction of the experimental station, composed of tents and frame buildings, which was named Camp Lazear (Figure 2-15). The "Infected Clothing Building" was sealed to prevent ventilation, while the "Infected Mosquito Building" was constructed for ventilation and divided into two living areas by a wire screen partition.[44]

Fig. 2-15. Camp Lazear. The experimental sanitary station near Quemados, Cuba, 1900. At this unimposing camp, the Reed Board, assisted by the staff of the Columbia Barracks Post Hospital, conducted the experiments that established the *Aedes aegypti* mosquito as a vector of yellow fever. Major William Gorgas implemented practical anti-mosquito measures in Havana that virtually eliminated yellow fever from the Cuban capital, thereby confirming the Board's results. Reproduced courtesy of the Historical Collections, Health Sciences Library, University of Virginia.

The Board would now attempt to infect nonimmunes by three methods: (1) bites from infected mosquitoes, (2) exposure to fomites contaminated with discharges from yellow fever patients, and (3) injection of blood from confirmed yellow fever cases. Between 8 December 1900 and 10 February 1901, the Board fed mosquitoes, infected 10 to 57 days previously, on 13 nonimmune volunteers. This produced 10 yellow fever cases. Five nonimmunes were injected with blood from these experimentally induced cases, producing 4 additional cases. In the "Infected Mosquito Building," those in contact with infected mosquitoes contracted the disease while controls on the opposite side of the screen remained healthy. No cases resulted from intimate contact with clothing, towels, and bed linens soiled by yellow fever patients.[46]

Results

The Board concluded that the yellow fever agent was transmitted by the *Stegomyia fasciata* (*A aegypti*) mosquito, that the agent required approximately 12 days incubation in the intermediate host, that the mosquito remained infectious for at least 57 days, and that the theory of propagation by filth was a myth.[46]

Gorgas acted on the Board's conclusions immediately. In Havana, yellow fever patients were quarantined, and, through the use of wire screening, uninfected mosquitoes were kept at bay. "Stegomyia Squads" controlled the mosquito population by screening or oiling cisterns and water barrels and fumigating patients and nearby houses with sulfur or pyrethrum. These control measures dramatically reduced yellow fever cases and deaths (Figure 2-16).[43,52]

Impact on Operations

The operational importance of the Board's work was 2-fold. First, field commanders no longer needed to continuously shift bivouac sites in an effort to avoid yellow fever. Rather, they and their surgeons implemented vector control procedures in camp.[53] Second, it provided medical researchers with a starting point for isolating the yellow fever agent, ultimately leading to vaccine development. This vaccine was first administered to US Army soldiers in 1941.[54]

Conclusion

The outcomes of the Spanish-American War and the Philippine Insurrection rapidday to 1/1,000 per day. Not only did the incidence of malaria, the major contributor to DNBI, decrease, but with forward treatment, time lost per case dropped from 5 months to 3 weeks.[55]

Impact on Operations

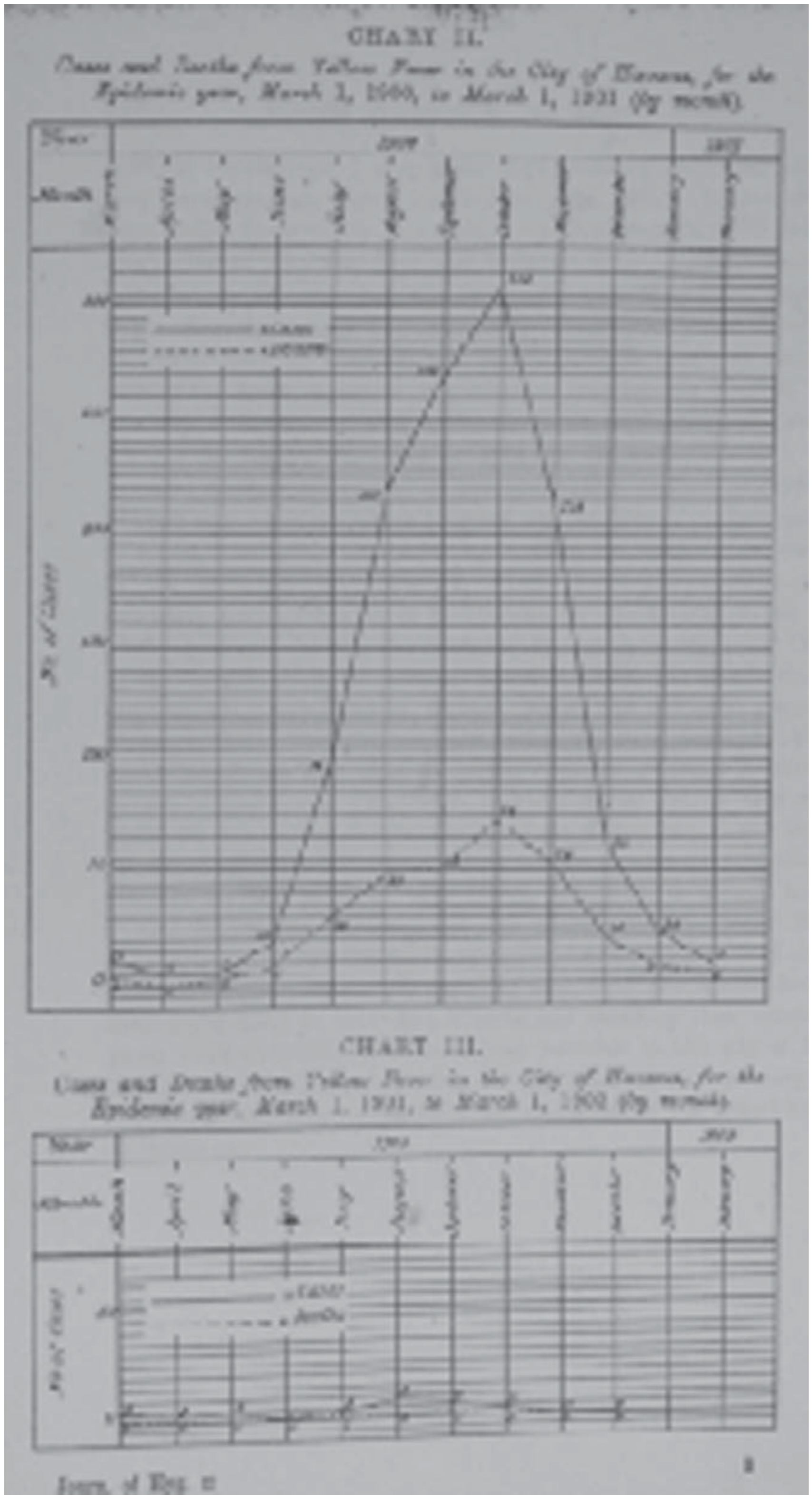

Fig. 2-16. Walter Reed's graphs of yellow fever cases and deaths in Havana for the years beginning March 1, 1900 (top) and 1901 (bottom), showing the dramatic reductions that resulted from the work of the Yellow Fever Commission. Reproduced with permission from: Reed W. Recent researches concerning the etiology, propagation, and prevention of yellow fever, by the United States Army Commission. *J Hyg*. 1902;2(2);117.

ety of diseases provided an abundance of research material. The Army Medical Department took full advantage of these opportunities. Scientific medical research in the Army, which included the Typhoid Fever Board in 1898 and the Yellow Fever Commission, expanded and developed in these tropical areas, becoming a valuable asset not only to the Army but also to medicine as a whole.

Lieutenant General William Slim in the China–Burma–India Theater, World War II

Setting

British Lieutenant General William Slim (Figure 2-17) provides one of the finest examples of the application of preventive medicine to war. In the China—Burma–India (CBI) theater of World War II, he faced enormous challenges. From January to May 1942, the Japanese 15th Army invaded Burma, driving out the combined British, Burmese, Indian, and Chinese forces. The British Army retreated north through Mandalay and west into India. Slim took over the I Burma Corps during the retreat in March 1942. In October 1943, he assumed command of the British 14th Army. Made up primarily of English and Indian forces, the army was demoralized and in poor health. His task was to retake Burma from the Japanese (Figure 2-18).[55–61]

Fig. 2-17. Lieutenant General William Slim, Commander-in-Chief, 14th Army, World War II. Slim was knighted during the war for his brilliant conduct of the China–Burma–India campaign. Reproduced courtesy of the Imperial War Museum, London, England.

Health Issues

Many diseases thrive among armies in tropical environments when the public health infrastructure is absent. The CBI theater was extremely harsh and unhealthy.[62,63] The main disease threats were malaria, dysentery, nonbloody diarrhea, various skin diseases, and scrub typhus. In the hot, humid environment of the CBI theater, skin diseases—particularly fungal infections and immersion foot—were hard to control. With no effective drug therapy, scrub typhus caused significant mortality. In 1943, the year Slim assumed command of the 14th Army, allied forces in the CBI theater were in bad shape. The force-wide incidence of malaria was 491/1,000 per year; the incidence of dysentery was 48/1,000 per year.[64] Hepatitis A incidence was 18/1,000 per year, having risen from 0.7/1,000 per year in 1938.[65]

Slim's Actions

Slim dealt deliberately with the army's poor health. He assessed the situation, developed and implemented a medical plan, and enforced it.

Shortly after Slim took over the 14th Army, he *assessed the health of his command*. He was amazed at what he found. On an annualized basis, 84% of his soldiers got malaria, and rates were even higher in forward units. His DNBI evacuation rate was 12/1,000 per day, 120 times greater than his BI evacuation rate. Slim concluded that "…in a matter of months at this rate my army would have melted away. Indeed, it was doing so under my eyes."[55p177] With the help of his surgeons, he formulated a list of medical threats and problems. After the initial assessment, he conducted ongoing medical surveillance, following key health indicators such as hospital admission rates. In this way he kept abreast of the health of the Army.[55]

Slim *developed and implemented a medical plan* that emphasized four points: research, forward treatment, air evacuation, and prevention. He assembled teams of scientists and physicians to conduct field

Fig. 2-18. British troops fighting in Burma jungle, World War II. Combat was fierce and difficult in the dense jungles of the China–Buma–India theater. Moreover, the hot, humid environment made many diseases hard to control. Reproduced courtesy of the Imperial War Museum, London, England.

research and apply this knowledge to prevention and treatment. He emphasized forward treatment instead of evacuation to India, establishing malaria forward-treatment units and forward surgical teams just miles behind the lines. He employed aircraft for medical evacuation. Air evacuation to India was faster and better tolerated by the seriously wounded, particularly given the heat, humidity, and tortuous road system of the CBI theater. His most important initiative was to raise "medical discipline," the British term for field hygiene and sanitation. Slim had concluded that "…prevention was better than cure. We had to stop men going sick, or, if they went sick, from staying sick."[55p178] Slim issued orders regarding various personal and collective preventive measures, such as not bathing after dark and taking anti-malarial medicine under supervision.[55,66]

Finally, Slim *enforced* his medical plan. He had relatively little trouble getting his research, forward treatment, and air evacuation initiatives going, but medical discipline was—and remains—a problem requiring frequent command emphasis. Slim spent most afternoons visiting his troops. While his main purpose was to encourage them by his presence and brief talks from the hood of his jeep, he paid close attention to their hygiene and sanitation, thereby forcing his leaders to give priority to preventive measures. He was tough in this regard, to the point of relieving commanders who tolerated poor medical discipline. Concerning the taking of the anti-malarial drug mepacrine (atabrine), for example, Slim wrote

> I, therefore, had surprise checks of whole units, every man being examined [by a blood test]. If the overall result was less than ninety-five per cent positive I sacked the commanding officer. I only had to sack three; by then the rest had got my meaning.[55p180]

Results

Slim observed that

> Slowly, but with increasing rapidity, as all of us, commanders, doctors, regimental officers, staff officers, and N.C.O.s, united in the drive against sickness, results began to appear.[55p180]

The results were dramatic, comparing 1943 to 1945. The DNBI evacuation rate dropped from 12/1,000 per day to 1/1,000 per day. Not only did the incidence of malaria, the major contributor to DNBI, decrease, but with forward treatment, time lost per case dropped from 5 months to 3 weeks.[55]

Impact on Operations

Confident in the health of his army, Slim was able to pursue combat operations during the monsoon season, when the disease threat peaked. Though Slim denied this, some thought he purposefully chose disease-ridden areas to engage the Japanese to take tactical advantage of his superior preventive medicine.[55,66]

During 1944, the 14th retook northwest Burma. From January to May 1945, the Allies pushed south to regain the remainder. With the occupation of Rangoon on 2 May 1945, Japanese resistance effectively ended.[55–61]

Conclusion

William Slim was one of the truly superb generals of World War II. Under arduous conditions, he took aggressive action that restored the health of his command and earned him an honored place in the history of military medicine. The success of the 14th Army in the field owed greatly to its good health and to the vision and leadership of General Slim.[63]

Slim recognized the central truth that the commander is responsible for the health of his command, with the medical officer as the primary advisor. This is his legacy to military medicine. His classic statement on this topic bears repeating: "Good doctors are no use without good discipline. More than half the battle against disease is fought, not by the doctors, but by the regimental officers."[55p180]

Meningococcal Disease in the US Army Training Base

Setting

In considering the application of preventive medicine to war, it is common to think of actions at or near the front. This bias toward the theater of operations overlooks many less noticeable but equally important efforts, such as mobilization and training, that occur in the zone of the interior (the continental United States). The story of meningococcal disease in the US Army training base is unlike the others treated in this chapter. Efforts to control this problem involved many different workers pursuing various lines of effort at numerous locations over several decades.

US military trainee populations are generally representative of the young adult populations from which they come. Recruits are mustered at training posts and trained for several weeks. Environmental factors in camp that predispose recruits for meningococcal disease include crowded living conditions, poorly ventilated barracks, rigorous training schedules, exposure to the elements, and sleep deprivation. While the US military always has recruits in training, the number swells in wartime.

Health Issues in the Early Twentieth Century

Infection with *Neisseria meningitidis* can produce a wide range of results, including asymptomatic nasopharyngeal carriage, local infection (eg, pharyngitis and pneumonia), and invasive disease (eg, disseminated meningococcemia and meningitis). Meningococcal disease is endemic in the US population, causing 1 to 3 cases per 100,000 population per year.[67] The petechial rash (Figure 2-19) often seen with invasive disease is classic.[68]

Meningitis has been a problem in the US Army during past wars, including the War of 1812, the Mexican–American War, and the Civil War; how-

Fig. 2-19. A classic petechial rash in a patient with fulminant meningococcal disease. Other signs and symptoms include fever, headache, nausea, vomiting, and neck stiffness. Meningitis has historically been a problem in US military training camps during periods of mobilization. The disease is still formidable; even with early diagnosis, modern therapy, and life support, the case-fatality rate is 5% to 15%. Reproduced from: Daniels WB. Meningococcal infections. In: Havens PW, ed. *Infectious Diseases.* Vol 2. In: *Internal Medicine in World War II.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1963: 254.

ever, exact diagnoses could not be established until the turn of the 20th century, when bacteriologic methods had developed sufficiently.[69,70] The disease is most common at recruit training camps, where organisms from various locales are shared.

During World War I from April to October of 1917, over 1 million American men were mobilized and trained at 39 different camps, most of which had meningitis epidemics. In January 1918, the annualized incidence rose to a peak of 459/100,000 enlisted men in the continental United States. By war's end, the overall annualized meningitis admission rate for the Army was 141/100,000 men. While the incidence was moderate, the case fatality rate (CFR) was not. The standard treatment was intrathecal injection of polyvalent antimeningococcal serum. Despite this treatment, there were 2,279 deaths in 5,839 cases for a CFR of 39%.[69,70]

Actions Taken and Results Achieved

During the 20th century, US wartime mobilization has always led to epidemic meningococcal disease, with the exception of the Korean and Persian Gulf Wars. These epidemics have prompted actions leading to advances in prevention and treatment.

During the World War I era, there was no effective prevention for meningococcal disease. Military doctors at several recruit camps studied various aspects of the problem, such as demographics, carriage, and transmission. Some attempted, unsuccessfully, to control transmission by methods such as isolation and treatment of cases and contacts. Glover noted a relation between crowding and carriage rate and between carriage rate and incidence, leading to the notion of increasing living space as a means of prevention.[70] Herrick, an Army physician at Camp Jackson, South Carolina, who studied 265 patients, recognized the importance of early diagnosis and treatment. His work led to the creation of special surveillance wards for acute respiratory disease patients, an effective practice still in use today.[69,71] While these pioneer physicians were unable to control the disease, they made practical advances and contributions to the understanding of meningococcal pathophysiology and epidemiology. With the war's end, though, the problem waned.

The era of effective antimicrobial therapy began in the mid 1930s when sulfonamide drugs were introduced.[70] Their efficacy against meningococcal infection was soon proven.[72] The CFR dropped dramatically but remained significant.

Antimicrobial therapy had little effect on incidence, however, and when mobilization for World War II began, there was cause for concern. In June 1941, the newly formed Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army (later chartered as The Armed Forces Epidemiological Board [AFEB]) established the Commission on Meningococcal Meningitis to study the incidence, treatment, and prevention of cerebrospinal meningitis.[73] Ultimately under the direction of Dr. John J. Phair and drawing on both military and civilian scientists, this commission conducted extensive research into the epidemiology of meningococcal disease. The commission concluded that the most effective way to decrease incidence was to eradicate carriage. This theory underlay the concept of mass antimicrobial prophylaxis.

The anticipated epidemic of meningococcal meningitis began in December 1942 and rose to a peak in March 1943, when incidence reached 290/100,000 troops per year in the Army. This epidemic accounted for half of the 13,922 cases that occurred during World War II. Based on the work of the Commission on Meningococcal Meningitis, the Army Surgeon General responded in September 1943 to the epidemic by directing sulfadiazine prophylaxis for concentrated troops, including those in training camps. This practice successfully controlled the epidemic. By war's end, 559 deaths were attributed to meningococcal disease, yielding a CFR of 4.0% and making it the second leading cause of infectious disease mortality, behind tuberculosis.[71,74]

After World War II, Aycock and Mueller reviewed meningococcal carriage and incidence studies conducted during the war years. They made several key observations, notably that military epidemics occur only in concert with civilian epidemics and therefore mirror US trends (Figure 2-20). This and other observations caused them to deemphasize factors such as carriage, rate of spread, and environment in favor of individual susceptibility factors. This theory underlay the concept of immuno-prophylaxis for definitive prevention.[71,75]

During the Korean War buildup, meningococcal disease epidemics did not occur in either the general US or trainee populations. Sulfadiazine prophylaxis controlled meningococcal activity at low levels. In fact, this grace period would last nearly 2 decades. Inevitably, antibiotic resistance developed. Meningitis became a serious problem at Fort Ord, Calif, beginning in 1960, 1 year after the rate in the surrounding county began increasing. The number of cases grew steadily, with the first death occurring in 1963. During 1964 there were 108 cases at Fort Ord. With public anxiety and pressure running high, recruit training was temporarily suspended

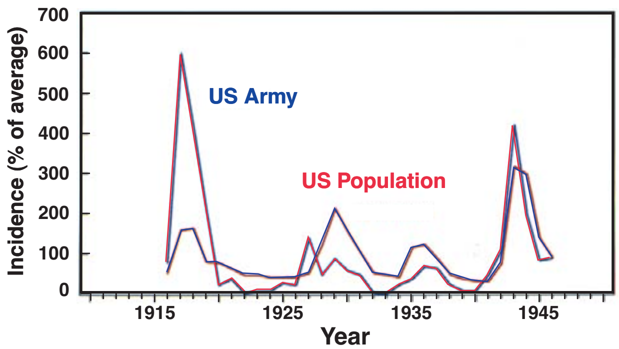

Fig. 2-20. Meningococcal meningitis incidence trends in the [continental] U.S. Army (blue line) and in the general U.S. population (red line), 1916–1946. For both groups, average incidence for the entire period is defined as 100% to facilitate direct comparison on the same graph. The actual Army incidence was approximately 7-fold higher than that of the US population. Note that military epidemics historically occur only in concert with civilian epidemics and so mirror US trends. Adapted from: Phair JJ. Meningococcal meningitis. In: Hoff EC, ed. *Communicable Diseases Transmitted Chiefly Through Respiratory and Alimentary Tracts.* Vol 4. In: *Preventive Medicine in World War II.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1958: 203. Prepared by Judith Bowles.

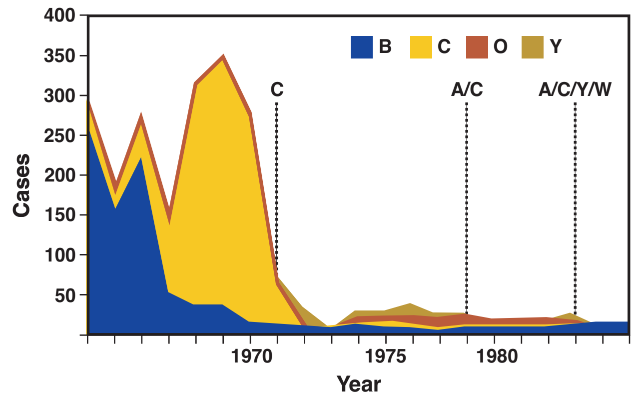

Fig. 2-21. Cases of meningococcal disease in U.S. Army personnel, 1964–1984. Vertical lines indicate introduction of group-specific vaccines. "Group O" indicates not grouped or grouped other than B, C, or Y. The Army's vaccine development program produced dramatic results. Adapted with permission from: Brundage JF, Zollinger WD. Evolution of meningococcal disease epidemiology in the US Army. In: Vedros NA, ed. *Evolution of Meningococcal Disease.* Vol 1. Boca Raton, Fla, CRC Press; 1987: 11. Prepared by Judith Bowles.

at Fort Ord in late 1964.[76,77] Ultimately, widespread sulfadiazine resistance rendered routine prophylaxis useless and the policy was discontinued. Without effective control measures and with the Vietnam War in full swing, meningococcal disease incidence in the Army increased to a high of 350 cases and 38 deaths in 1969.[71,78]

With emphasis on meningococcal meningitis renewed by wartime mobilization, the Army Medical Department sought a definitive solution to this problem. Research efforts intensified, centered at the Walter Reed Army Institute of Research (WRAIR), and led by Dr. Malcolm Artenstein. Goldschneider, Gotschlich, and colleagues investigated the safety and immunogenicity of group A, B, and C *N meningitis* polysaccharides in humans. The WRAIR team developed a group C polysaccharide vaccine and began field testing in 1969 at Army training centers. Full-scale immunization of all Army recruits began in October 1971, albeit after the epidemic had peaked. Vaccines against groups A, Y, and W-135 were developed and tested by WRAIR during the 1970s. By late 1982, all recruits were immunized with the tetravalent meningococcal vaccine.[71,78–91]

The Army's vaccine development program produced dramatic results. Since 1973, meningococcal disease has been well controlled in the US military training base (Figure 2-21). Moreover, for the first time since statistics have been kept, meningococcal disease incidence in the trainee population has become dissociated from that in the general US popu-

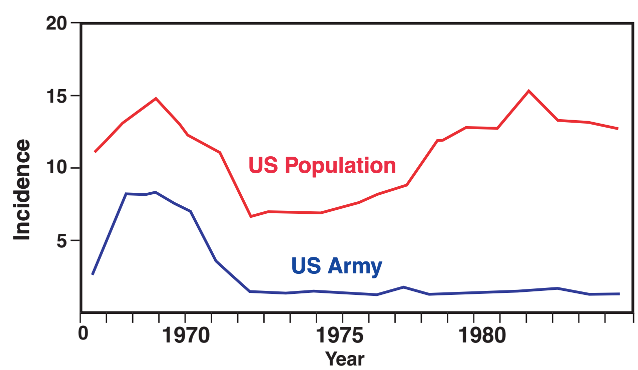

Fig. 2-22. Meningococcal disease incidence rates for the general US population (top line, cases/1 million per year) and the US Army (bottom line, cases/10,000 per year), 1967–1984. This conclusively illustrates the effectiveness of the Army's meningococcal vaccine program. Compare with Figure 2.20. Adapted with permission from: Brundage JF, Zollinger WD. Evolution of meningococcal disease epidemiology in the US Army. In: Vedros NA, ed. *Evolution of Meningococcal Disease.* Vol 1. Boca Raton, Fla, CRC Press; 1987: 13. Prepared by Judith Bowles.

lation (Figure 2-22). The effectiveness of the tetravalent vaccine is further shown by the virtual absence of groups A, C, Y, and W-135 disease in soldiers, though it still occurs in the general US population.[71]

Impact

Today, the US military training base operates with almost complete freedom from the once onerous burden of meningococcal disease. While sporadic cases occur,[92,93] they are generally due to group B, for which there is not yet an effective vaccine.[94]

Conclusion

The effort to control meningococcal disease involved a great number of people over many years, demonstrating the interplay of epidemiology, basic science, and clinical investigation in prevention. Contributions came from the general scientific community, with the Army Medical Department carrying the effort forward during mobilization periods.

The meningococcal story illustrates the great potential of immunization, as opposed to other strategies, in preventing disease in military and other populations. Short of disease eradication, an effective vaccine is generally the best possible solution, especially for military personnel.

As successful as this story seems, it is not yet over. Because of the continued difficulty in developing an effective group B vaccine, today's trainees are still at risk for meningococcal infection, particularly if a group B epidemic in the general population were to occur coincident with a military buildup. Diagnosis and treatment continue to pose a challenge; the CFR remains formidable at 5% to 15%.[68] The claim of victory must await either a total coverage vaccine or disease eradication. Lieutenant Colonel J. D. Bartley, an Army physician who studied meningococcal meningitis in the recruits at Fort Dix from 1968 to 1970, had these words to say about overconfidence:

> ... the solution to the meningococcal problem seemed at hand once before, when SDZ [sulfadiazine] was introduced. Nature does not accept conquest lightly and may once again emerge the victor from apparent defeat.[78p379]

Skin Disease in the US 9th Infantry Division, Republic of Vietnam

Setting

During the summer of 1968, the US 9th Infantry Division, commanded by Major General Julian J. Ewell, moved from its sector near Saigon to the Mekong (Nine Dragon) River delta area. Having fought in the January 1968 Tet and May 1968 Mini-Tet offensives, the 9th was in a suboptimal state of combat effectiveness due to a variety of administrative and operational factors, including unit organization, static mission, high operational tempo, and recent losses.[95,96]

Known as the "Mouth of the Dragon," the Mekong delta region is low, flat, hot, and wet. The ground averages only 2 m above sea level, with the main relief features being dikes that rise a meter or so above the rice paddies. Average daily temperatures range from a low of 24°C (75°F) to a high of 32°C (90°F), with little seasonal variation. During the rainy season, up to 90% of the area is covered by water.[95,97]

To deny the enemy respite that is so critical to guerrilla forces, the 9th employed the constant pressure concept. This tactic was highly effective in weakening the Viet Cong but demanded much field time of American units as well. Rifle companies typically spent two thirds of their time in the field, often remaining there for 5 or more continuous days. While armored personnel carriers were useful during the dry season (November through March), vehicular traffic was difficult if not impossible during the rainy season (May through October). As the dikes were often booby trapped, the infantry had to walk in the flooded paddies to maintain constant pressure (Figure 2-23).[95]

Health Issues

During French operations in Indochina from 1945 to 1954, skin disease had caused considerable morbidity and manpower degradation.[98] With an incidence of 42 admissions per 1,000 troops per year (average experience over 9 years),[99] dermatological disease was the leading cause of hospitalization. Fungal and staphylococcal infections were the most common diagnoses.[97]

During the American experience in Vietnam from 1965 to 1972, dermatological complaints were the most frequent cause of outpatient presentation, accounting for 12.2% of all visits.[97,100] Given the conditions in the Mekong delta area, it is not surprising that skin problems were even more significant in the 9th Infantry. For the 1-year period from July 1968 through June 1969, skin disease accounted for 47% of all disease and injury, including battle injury, in maneuver battalions.[95]

Typically, one third of the men went on sick call after an operation in the delta, the majority with

Fig. 2-23. Combat patrol entering rice paddy, Mekong River delta, Vietnam. Mission demands sent rifle companies of the 9th Infantry Division into the field, often for 5 or more continuous days. The infantrymen normally had to walk in the flooded paddies. The constant exposure to wet conditions made skin disease a major problem. Official US Army photo, Walter Reed Army Institute of Research, Silver Spring, Md.

skin complaints. The three main problems were pyoderma (bacterial infection, usually streptococcal), dermatophytosis (fungal infection), and immersion foot (tropical type).[95,97,101] Most were given light duty or quarters for an average of 4 days, but many received permanent restrictions from field duty.[97] By March 1968, many 164-man rifle companies were taking only 65 to 70 men to the field on combat operations.[95]

The epidemic of dermatological disease in the 9th occurred in part because, with only four trained US military dermatologists in-country, there was insufficient knowledge at the battalion level of prevention, diagnosis, and treatment of skin disease. The reason the epidemic went largely unnoticed was administrative. Neither medical nor personnel reports listed the number of men on restricted duty, thus neither indicated the problem.[95]

At the same time, the local population was noted to have no significant skin problems.[101,102] The rice farmers went barefoot or wore sandals in the paddies by day and allowed their feet to dry at night.[103]

A Series of Actions and Results

Dermatological disease caused great concern outside the 9th Infantry Division as well. In May 1967, the AFEB Commission on Cutaneous Diseases offered to send an expert team of dermatological consultants to Vietnam to study the problem. Noting the steady rise of dermatological disease, the US Army, Vietnam (USARV) Surgeon invited the AFEB consultants.

The team, consisting of Dr. Harvey Blank, Dr. Nardo Zaias, and Mr. David Taplin, arrived in October 1967 and spent several weeks visiting soldiers in the field, in clinics, and in hospitals. They brought a field laboratory with culture capability, which proved very useful. The team defined the various causes of dermatological morbidity and made several practical recommendations, which were implemented in USARV Medical Service Regulation 40-29 (10 January 1968).[97,100] The team submitted its final report on 24 May 1968.[73]

From November 1968 through February 1969, a field dermatology research team from WRAIR, headed by Captain Alfred M. Allen, studied dermatological conditions in soldiers and Vietnamese civilians in the 9th Division area. Its findings were similar to those of the AFEB team.[97,101,102] These and many other scientific efforts contributed significantly to the understanding of dermatological disease in Vietnam.[104–114]

In the 9th Infantry Division, Major General Ewell took several actions to address the problems. First, he defined the manpower status. Ewell's Chief of Staff, Colonel Ira A. Hunt, assembled a group of captains and majors and applied operations research techniques to problems the division faced.[115] With a goal of optimizing the number of infantrymen in the field, the group developed a "paddy strength report" that provided details on those not present for combat operations—details the morning report was missing. Through this, Ewell saw that as many as half of the men in the rifle companies were nonavailable due to skin disease.[95]

Surprised and alarmed, Ewell pursued two actions simultaneously. He sent unfit infantrymen with duty limitations to other units with garrison duty in dry areas, exchanging them for healthy soldiers. More importantly, he commissioned Operation Safe Step.[95]

Operation Safe Step was a medical research pro-

Fig. 2-24. Soldier volunteers in test paddy at Dong Tam, Vietnam during Operation Safe Step. This paddy, in a rear area, was used to test disease incidence, clothing, and various protective measures under simulated combat conditions. Small groups of soldier volunteers were rotated in and out of Dong Tam. Official US Army photo, Walter Reed Army Institute of Research Silver Spring, Md.

gram designed to control and minimize foot problems. This three-pronged effort tested various types of footgear, several protective skin ointments, and skin disease in volunteers exposed to paddy water over varying lengths of time. Two successive division surgeons, Lieutenant Colonels Travis L. Blackwell and Archibald W. McFadden, directed Operation Safe Step, in consultation with Colonel William A. Akers, US Army Medical Research Unit, Presidio of San Francisco (later Letterman Army Institute of Research). A test center was established at Dong Tam, where field trials were conducted in actual rice paddies, and used soldier volunteers (Figure 2-24) to test ointments provided by Colonel Akers and experimental footgear provided by the US Army Natick Research Laboratories. Captain Allen's field dermatology research team assisted as well. Operation Safe Step's findings encompassed etiology, prevention, treatment, and equipment and served as the basis for many decisions and policies.[95,97,104,105] McFadden also assembled data that suggested a time course relation between paddy water exposure and incidence of pyoderma, dermatophytosis, and immersion foot (Figure 2-25).[104]

Convinced by the findings, Ewell altered division tactical procedure to accommodate the health of his soldiers. On 28 October 1968, he issued an order limiting operations in paddies to 48 hours (unless pinned down by the enemy), followed by a 24-hour drying period.[95,97] With this strong command interest and the onset of the dry season, time lost to skin disease in rifle battalions dropped from well over 3,000 man-days per month to nearly 1,000 man-days per month.[97]

On a larger scale, the US Army in Vietnam fared better than the French. Over 99% of all American skin cases were handled as outpatients. Dermatological hospitalization, with incidence ranging from 19 to 33 admissions per 1,000 troops per year during the period 1965 to 1972, accounted for 7.4% of the 619,121 Army hospital admissions during the war. Skin disease was the third leading cause of hospitalization, behind diarrheal disease (12.5%) and respiratory infection (11.6%), and just ahead of malaria (7.3%).[97,116]

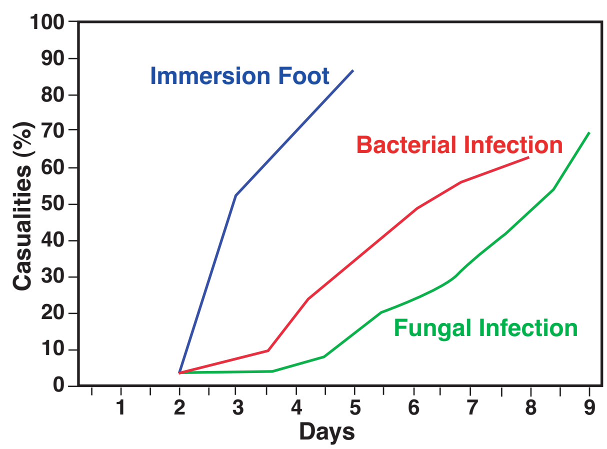

Fig. 2-25. Temporal relation between exposure to paddy water and incidence of debilitating pyoderma, dermatophytosis, and immersion foot in infantrymen. This figure was developed from data (Division Weekly Dermatology Sick Call Report Summaries, May 1968 to August 1969, and other) collected by Lieutenant Colonel Archibald W. McFadden, 9th Infantry Division Surgeon. Paddy foot: a warm water immersion foot syndrome variant. *Mil Med.* 1974;139:611. Prepared by Judith Bowles. Adapted with permission from *Military Medicine: The Official Journal of AMSUS*. Akers WA.

Impact

The knowledge gained by the AFEB, the Army Medical Department, and the 9th Infantry Division staff was translated into actions that significantly affected company-level operations. Paddy strength—the number of infantrymen available for combat operations—rose from 65 to 120 men per rifle company, enabling these units to operate effectively. While other administrative efficiencies contributed, Ewell and Hunt wrote, "This medical research effort [Operation Safe Step] proved to be the most important single factor in increasing the paddy strength of the 9th Division."[95p23]

Conclusion

The story of the 9th Infantry underscores at least three preventive medicine themes. First, surveillance data is invaluable, both in identifying problems and in pointing toward solutions. Second, medical research is not only possible but can be extremely useful, *even when units are deployed*. Finally, command emphasis is central to prevention of disease and nonbattle injury. Without it, other seemingly more pressing issues will inevitably preoccupy the junior leaders and soldiers. Like Lieutenant General Sir William Slim, Major General Ewell was an astute leader who exploited preventive medicine, as he put it, to "sharpen the combat edge."[95]

SUMMARY

While the main purpose of this chapter is to illustrate the impact of preventive medicine in war, it is possible to draw from these eight stories at least four major conclusions concerning the practice of preventive medicine in war.

There is no substitute for preparation. Consider the problems of the British in South Africa and the Americans on Attu Island, caused in part by inadequate preparation. Medical planning should begin not with force size, casualty estimates, and hospital bed projections but with medical intelligence, medical threat analysis, and preventive medicine planning.

Medical surveillance must be conducted. With an ongoing medical surveillance system, commanders and medical managers can rapidly identify medical threats and devise appropriate countermeasures. Without it they are blind to the threats until they become obvious. Generals Slim and Ewell made excellent use of medical surveillance.

Field research is often invaluable during operations. It is shortsighted to believe that field research is not important or cost-effective during wartime, even at the operational level. Many of the stories discussed in this chapter illustrate this point: the work of the Yellow Fever Commission in Cuba, the nearly complete conquest of meningococcal disease in the US training base, Adolph's desert research during World War II, and the various dermatological research efforts conducted by the US Army in Vietnam.

Preventive medicine plans need solid command support to succeed. Without continued and strong command emphasis, *including enforcement*, the tedious, mundane, and often distasteful soldier-level tasks so critical to preventive medicine will go undone. Slim's tough enforcement and dramatic results demonstrate this principle. Unfortunately, this most important lesson is perhaps most easily forgotten, because, indeed, the individual preventive medicine measures are often tedious, mundane, and distasteful.

Returning to the impact of preventive medicine in war, the examples presented here illustrate the price paid by commanders who do not pay attention to Jonathan Letterman's *leading idea*, and the rewards that accrue to those who do. Leaders who properly apply preventive medicine significantly increase their combat strength. Those who ignore it forgo this advantage at the least. At the most, when battle conditions or nature turn against them, they suffer terrible consequences.

There are many more examples that could be cited. On the negative side are Napoleon's losses to cold injury in Russia,[117,118] Hitler's making the same mistake over a century later,[119–121] and Rommel's lack of attention to field sanitation in North Africa.[122–125] On the positive side are Washington's concern for variolation and field sanitation in the Continental Army,[2,126,127] the British Army's successes with "shell shock" during World War I,[128,129] and MacArthur's actions against malaria in the Southwest Pacific in World War II.[130]

In the press of war, when tactical and operational concerns close in, men sometimes become shortsighted. The responsibility for a military force's health is shared. Commanders—who bear the ultimate responsibility—should not forget Letterman's leading idea.[1,131,132] And their medical officers should not let them.[133]

> Those who cannot remember the past are condemned to repeat it.[134p284]
>
> George Santayana (1863–1952)

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