The Open-Air Treatment of PANDENIC INFLUENZA

Richard A. Hobday, PhD, and John W. Cason, PhD

The H1N1 "Spanish flu" outbreak of 1918–1919 was the most devastating pandemic on record, killing between 50 million and 100 million people. Should the next influenza pandemic prove equally virulent, there could be more than 300 million deaths globally. The conventional view is that little could have been done to prevent the H1N1 virus from spreading or to treat those infected; however, there is evidence to the contrary. Records from an "open-air" hospital in Boston, Massachusetts, suggest that some patients and staff were spared the worst of the outbreak. A combination of fresh air, sunlight, scrupulous standards of hygiene, and reusable face masks appears to have substantially reduced deaths among some patients and infections among medical staff. We argue that temporary hospitals should be a priority in emergency planning. Equally, other measures adopted during the 1918 pandemic merit more attention than they currently receive. (*Am J Public Health*. 2009;99:S236–S242. doi:10.2105/AJPH.2008.134627)

THREE INFLUENZA PANDEMICS

occurred during the last century: in 1918, 1957, and 1968. Each was caused by a novel type A influenza virus of avian origin. The H1N1 influenza pandemic of 1918–1919 is notorious because of the infectivity of the virus and the number of lives it claimed. Although the fatality rate was relatively low, the incidence of infection was so great that the number of deaths was high. No other pandemic in history killed so many in such a short time. I

Global mortality from the pandemic is not known, because there are large areas of the world for which there is little information. In the 1920s, it was

estimated that the disease had killed 21 million people. In 1991, this figure was revised to between 24.7 million and 39.3 million, and more-recent scholarship suggests 50 million to 100 million people may have died.² Morbidity was high, at anywhere from 25% to 90%, and the fatality rate was between 1% to 3%.3 However, some regions reported mortality rates for the entire population as high as 5% to 10%.2 Most deaths occurred between mid-September and mid-December of 1918.4 Unusually, many of those who died were young adults, who normally have a low death rate from influenza. Another striking feature was the

discoloration of the seriously ill, who often exhibited "heliotrope cyanosis," which is characterized by a blue-gray tinge to the face and other parts of the body.^{3,5} Many victims died of pneumonia caused by secondary bacterial infections. Others succumbed to a condition similar to acute respiratory distress syndrome that could kill within days or hours.^{5,6} Pleurisy, hemorrhage, edema, inflammation of the middle ear, meningitis, nephritis, and pericarditis were among the many complications reported.^{6,7}

There were 3 waves of infection between 1918 and 1919. The first, in the spring of 1918, spread through parts of the United States, Europe, and Asia. This was a fairly mild form of influenza and caused relatively few fatalities. The second wave, which spread around the world in a few months, was disastrous. In less than a year, 220000 influenza-related deaths occurred in Britain, and between September 1918 and June 1919 it proved fatal to at least half a million US citizens.^{1,3} Death rates

in Africa were comparable to or higher than those in North America and Europe.⁸ Figures suggest that China was spared the worst of the pandemic, although this may simply reflect a lack of accurate records. The mortality in India alone has been estimated at 18 million. 9,10 According to one estimate of the period, 800 of every 1000 people who showed symptoms suffered from uncomplicated influenza. This was more severe than the so-called "three-day fever" of the spring of 1918, but no worse than ordinary influenza. The remaining 200 suffered pulmonary complications; of these, the mortality rate for those developing heliotrope cyanosis was 95%.

With so many infected, and so many dying within a few weeks, the burden on medical staff and the funerary industry were immense, as was the accompanying economic and social disruption.^{1,3} There was much debate about the origins of the illness and whether it was indeed influenza. The symptoms were so severe that there was speculation that it was some other disease such as "trench fever," dengue, anthrax, cholera, or even plague. 1,3,11 Mortality reached alarming levels. The pandemic arrived in Boston, Massachusetts, early in September and by October 19 had claimed 4000 lives out of a total population of less than 800000.12 At the peak of the outbreak, more than 25% of patients at an emergency hospital in Philadelphia died each night, many without seeing a nurse or doctor. The bodies of those who succumbed were stored in the cellar of the building, from where they were tossed onto trucks and taken away. Attempts at therapy for those still alive were described as "exercises in futility." 13(p139)

The demands of wartime meant that many doctors had been called into military service; those not in uniform were caring for the wounded in hospitals at home or inspecting potential recruits at medical boards. The shortage of nurses was even more acute: as they and other medical staff fell ill, patient care rapidly deteriorated.^{1,3,14} Hospitals were turning patients away; mortuaries were overflowing, some handling 10 times their normal capacity. Gravediggers, many of whom were ill, could not keep up with the demand for burials. 1,3,15 Early in October 1918, a delegate from a health department in the US Midwest went east to find out how best to combat the infection. Officials there offered the following advice:

When you get back home, hunt up your wood-workers and cabinet-makers and set them to making coffins. Then take your street laborers and set them to digging graves. If you do this you will not have your dead accumulating faster than you can dispose of them. 12(p787)

This was not meant to cause undue alarm; it was merely a practical solution to a problem that had to be addressed once the pandemic arrived.12 In an attempt to prevent the infection from spreading, many cities banned public assembly, closed their schools, isolated those infected, and mandated the wearing of surgical face masks. 1,3,6 Recent studies suggest that when such measures were introduced quickly-before the pandemic was fully established-and then sustained, death rates were reduced.16-19 Yet for those who contracted the disease and went on to develop pneumonia, the prospects were poor. Anyone

fortunate enough to gain admission to an "open-air" hospital, however, may have improved their chances of survival.

THE ORIGINS OF THE **OPEN-AIR REGIMEN**

By the time of the 1918–1919 pandemic, it was common practice to put the sick outside in tents or in specially designed open wards. Among the first advocates of what was later to become known as the "open-air method" was the English physician John Coakley Lettsom (1744-1815), who exposed children suffering from tuberculosis to sea air and sunshine at the Royal Sea Bathing Hospital in Kent, England, in 1791.20,21 Lettsom's enthusiasm for fresh air attracted little support at the time, and the next doctor to recommend it met with fierce opposition. George Bodington (1799-1882) was the proprietor of the first institution that could be described as a tuberculosis sanatorium, at Sutton Coldfield near

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Birmingham, England. He treated pulmonary tuberculosis with a combination of fresh air, gentle exercise in the open, a nutritious, varied diet, and the minimum of medicines.

In 1840, Bodington published the results of his work in AnEssay on the Treatment and Cure

of Pulmonary Consumption, On Principles Natural, Rational and Successful.22 Bodington's essay includes accounts of six cases; one patient died, as he acknowledged, but the others were either cured or greatly improved. This was at a time when, he estimated, one in five people in England were dying of the disease and little was being done to prevent it. Tuberculosis was generally regarded as hereditary, noninfectious, and incurable. Bodington argued otherwise, objecting strongly to the use of blistering, bleeding, and the popular purgative drugs of the day as well as the practice of confining patients in warm, badly ventilated rooms to protect them from the supposedly harmful effects of cold air, "thus forcing them to breathe over and over again the same foul air contaminated with the diseased effluvia of their own persons."22(p2)

Bodington had noticed that people who spent their time indoors were susceptible to tuberculosis, whereas those who worked outdoors, such as farmers, shepherds, and plowmen, were usually free of the disease. He reasoned that patients should copy the lifestyles of those who appeared immune to tuberculosis. They should live in well-ventilated houses in the country and spend much of their time outside breathing fresh air. According to Bodington,

The application of cold pure air to the interior surface of the lungs is the most powerful sedative that can be applied, and does more to promote the healing of cavities and ulcers of the lungs than any other means that can be employed.^{22(p17)}

It is not known when Bodington started treating tuberculosis in this way, but there is evidence that he was doing so by 1833. By 1840, he had taken the tenancy of the "White House" at Maney, Sutton Coldfield, to provide suitable accommodation for his tubercular patients. Bodington's tenancy of this seminal building was brief—only three to four years. The *Lancet* published a sarcastic review of his essay and methods, and he abandoned the White House to devote himself to the care of the mentally ill.^{23,24}

George Bodington had anticipated the principles of sanatorium treatment that were to become the main line of defense against the disease.25 By the 1850s, Florence Nightingale (1820–1910) was writing about the importance of sunlight and copious amounts of fresh air in the recovery of hospital patients,26,27 but her ideas were slow to gain acceptance. And so it was in Germany that the openair regimen reemerged, most notably at the Nordrach-Kolonie in the Black Forest, a sanatorium established in 1888 by Otto Walter (1853-1919). It was so well known that "Nordrach" became the term for open-air sanatoria. By 1908, there were at least 90 of them in Britain, many of which were enthusiastic imitations of Nordrach.²⁸ An open-air recovery school for tubercular children, founded in 1904 at Charlottenburg, a suburb of Berlin, was the first of its type and, as with Germany's open-air sanatoria, was widely imitated.²⁹ In 1884, Edward Livingston Trudeau (1848–1915) opened America's first sanatorium at Saranac Lake in New York State.30 The first open-air orthopedic hospital was set up in the Shropshire village of Baschurch in England in 1907.31 In the two decades before World War I,

charitable associations, leagues,

and societies dedicated to preventing and eliminating tuberculosis among the poor flourished, as did sanatoria.³²

THE OPEN-AIR TREATMENT OF THE WOUNDED

There is evidence that the open-air regimen may have improved the health of some tuberculosis patients. Records for the Dreadnought Hospital in Greenwich, one of the first British hospitals in which such methods were adopted, appear to show that there were benefits to this approach. From 1900 to 1905, the overall mortality of consumptive patients in open-air wards was less than half that of those who received the orthodox treatment of the day. An improvement in their state of "well-being" was also reported.33 Later, during World War I, the use of open-air therapy extended to nontubercular conditions, and on a large scale. Temporary open-air hospitals were built to take casualties from the Western Front.

An early example stood on one of Cambridge University's best cricket pitches at the King's and Clare Athletic Ground. The First Eastern General Hospital, which was mobilized in August 1914, was originally designed to provide 520 beds and to be erected in 4 weeks. It proved so popular with the authorities, however, that within 8 weeks its complement of beds more than doubled to 1240. The hospital's wards were completely open to the south except for some low railings and adjustable sun blinds.34,35

In June 1915, the eminent scientist and Master of Christ's College, A.E. Shipley (1861–1927), judged the open-air treatment of

sick and wounded soldiers at the First Eastern a success, particularly for those with pneumonia. Some 6600 patients had passed through the hospital, with a death rate of 4.6 per 1000. Sixty patients with pneumonia had been treated, and 95% of them recovered. Critics ascribed the low mortality at the hospital to the absence of "bad cases," but according to Shipley, some convoys arrived from the trenches almost entirely made up of them. In his opinion, the open wards produced much better results than closed ones. Instead of patients losing their bodily health and strength during the period of recovery from infections or wounds, they maintained their vigor and even improved it. The only people who felt the cold at the hospital were apparently the nurses, the patients having comfortable beds with plenty of blankets and hot-water bottles.35 Nearer the front, the British Army put its casualties in tents. As the military surgeon Lieutenant Colonel Sir Berkeley Moynihan observed in 1916,

In the treatment of all gunshot wounds where the septic processes are raging, and the temperature varies through several degrees, an immense advantage will accrue from placing patients out of doors. While in France I developed a great affection for the tented hospitals. There is great movement of air, warmth and comfort; when a sunny day comes the side of the tent may be lifted and the patient enjoys the advantage of open-air treatment. "^{36(p337)}

INFLUENZA AT THE CAMP BROOKS OPEN-AIR HOSPITAL

When the influenza virus pandemic took hold in the United States in 1918, emergency hospitals were started in schools, halls, and large private houses, and open-air hospitals were being "thrown up" all over the country.1 In the harbor of East Boston, 1200 out of 5100 merchant sailors onboard training ships had contracted influenza. The seriously ill were too numerous for local hospitals to accommodate. The Massachusetts State Guard responded by building the Camp Brooks Open Air Hospital at Corey Hill in Brookline, near Boston.37,38 The hospital comprised 13 tents, 12 of which were occupied by one or two patients each and the other by the head nurse. The State Guard took seven hours to erect the tents, make sure the site was properly drained, and provide running water, latrines, and sewerage. Portable buildings were then set up for the medical staff and nurses. From the time the camp opened on September 9, 1918, until its closure a month later on October 12, a total of 351 victims of the pandemic were admitted, one third of whom were diagnosed with pneumonia. In total, 36 of the 351 sailors received at the hospital died.³⁷

The treatment at Camp Brooks Hospital took place outdoors, with "a maximum of sunshine and of fresh air day and night."37(p1747) The medical officer in charge, Major Thomas F. Harrington, had studied the history of his patients and found that the worst cases of pneumonia came from the parts of ships that were most badly ventilated. In good weather, patients were taken out of their tents and put in the open. They were kept warm in their beds at night with hot-water bottles and extra blankets and were fed every few hours throughout the course of the fever. Anyone in contact with

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them had to wear an improvised facemask, which comprised five layers of gauze on a wire frame covering the nose and mouth. The frame was made out of an ordinary gravy strainer, shaped to fit the face of the wearer and to prevent the gauze filter from touching the nostrils or mouth. Nurses and orderlies were instructed to keep their hands away from the outside of the masks as much as possible. A superintendent made sure the masks were replaced every two hours, were properly sterilized, and contained fresh gauze.38

Other measures to prevent infection included the wearing of gloves and gowns, including a head covering. Doctors, nurses, and orderlies had to wash their hands in disinfectant after contact with patients and before eating. The use of common drinking cups, towels, and other items was strictly forbidden. Patients' dishes and utensils were kept separate and put in boiling water after each use. Pneumonia and meningitis patients used paper plates, drinking cups, and napkins; paper bags with gauze were pinned to pillowcases for sputum. Extensive use was made of mouthwash and gargle, and twice daily, the proprietary silver-based antimicrobial ointment Argyrol was applied to nasal mucous membranes to prevent ear infection.37

Of the camp's medical staff— 15 doctors, 45 nurses and aids, 20 sanitary corps men, and 74

sailors acting as orderlies—only six nurses and two orderlies developed influenza. In five of these cases, exposure to the virus was reported to have taken place outside the camp. A few medicines were used to relieve the patients' symptoms and aid their recovery, but these were considered less important than were regular meals, warmth, and plenty of fresh air and sunlight.³⁷

VENTILATION AND SUNLIGHT

The curative effects of fresh air were investigated at length by the physiologist Sir Leonard Hill (1866–1952) in the years following World War I. He reported favorably on the effects of sun

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and air when judiciously applied, particularly for tuberculosis. 39,40 In 1919, Hill wrote in the British Medical Journal that the best way to combat influenza infection was deep breathing of cool air and sleeping in the open.41 Whether the patients at Camp Brooks or other temporary hospitals were spared the worst of the influenza pandemic because they slept in the open is uncertain. The apparent success in reducing the number of infections and deaths reported at this open-air hospital may simply have been caused by patients and staff experiencing levels of natural ventilation far higher than in a conventional hospital ward. Significantly, the minimum

amount of ventilation needed to prevent the spread of infectious diseases such as severe acute respiratory syndrome (SARS) and tuberculosis is unknown. Much more fresh air may be needed than is currently specified for hospitals, schools, offices, homes, and isolation rooms. 42–44

The patients at Camp Brooks recovered in direct sunlight when available. This may have kept infection rates down, because laboratory experiments have shown that ultraviolet radiation inactivates influenza virus and other viral pathogens and that sunlight kills bacteria. 45-50 In addition, exposure to the sun's rays may have aided patients' recovery, because sunlight is known to promote healing in other conditions such as septic war wounds.35 There is evidence that heart attack victims stand a better chance of recovery if they are in sunlit wards.51 Depressed psychiatric patients fare better if they get some sun while hospitalized, as do premature babies with jaundice. 52-55 In one study, patients in hospital wards exposed to an increased intensity of sunlight experienced less perceived stress and less pain and took 22% less analgesic medication per hour.56 One advantage of placing patients outside in the sun is that they can synthesize vitamin D in their skin, which they cannot do indoors behind glass. Rickets, the classic childhood disease of vitamin D deficiency, has long been associated with respiratory infections; it has been hypothesized that low levels of vitamin D may increase susceptibility to influenza.57,58

The surgeon general of the Massachusetts State Guard, William A. Brooks, had no doubt that open-air methods were effective at the hospital, despite

much opposition to the therapy. Many doctors felt that patients would get the same benefits if the windows of a conventional ward were open or the patients were put in a hospital "sun parlor." Brooks, however, held that patients did not do as well in an ordinary hospital, no matter how well ventilated, as they did outdoors. Patients in indoor sun parlors were not exposed to direct sunlight all day as they were when outdoors. He reported that in one general hospital with 76 cases, 20 patients died within three days and 17 nurses fell ill.38 By contrast, according to one estimate, the regimen adopted at the camp reduced the fatality of hospital cases from 40% to about 13%.12 Brooks wrote that "The efficacy of open air treatment has been absolutely proven, and one has only to try it to discover its value."38(p750)

Coincidentally, in 1918 a British soldier, Patrick Collins, reached a similar conclusion. When Collins developed the first signs of influenza, he dragged himself and his tent up a hill away from his regiment. There he sweated, shivered, and was delirious for several days, sustained only by his rum ration. He was one of the few survivors of his regiment. ⁵⁹

DISCUSSION

The seeming success of the medical team who confronted pandemic influenza on Corey Hill in 1918 was in stark contrast to others' experience of the infection. The high standard of personal and environmental hygiene upheld by staff at the camp may have played a large part in the relatively low rates of infection and mortality there compared with other hospitals. Significantly,

the outbreak of SARS in Hong Kong in 2003 showed that basic infection controls, such as those employed at Camp Brooks Hospital, can help to contain the spread of a virulent respiratory infection. 60,61

Of the measures introduced to combat pandemic influenza at the hospital, the use of improvised facemasks-including their design and the frequency with which they were changed-is noteworthy. Another is the fresh air the patients enjoyed. When Major Harrington, the medical officer at Camp Brooks, discovered that sailors from the most poorly ventilated areas of the ships in East Boston also had the worst cases of pneumonia, he put his patients outdoors. Sailors, such as those on board the ships at East Boston, were particularly vulnerable to influenza infection, because the influenza virus is readily transmitted in confined quarters. In 1977, for example, an influenza outbreak on board a commercial airliner with deficient ventilation resulted in an infection rate of 72%. The aircraft was grounded for over four hours with the passengers on board and the ventilation system turned off.62

There is still much uncertainty surrounding the transmission and epidemiology of influenza. As yet, the proportion of influenza infections that occur by the airborne route is not known, 63 nor is there any evidence to support the idea that fresh air helps those infected to recover. Given the threat to public health posed by the avian influenza virus, both merit further study. So too does the part played by sunlight in preventing the spread of the virus. Solar radiation may retard its transmission by directly inactivating virions and by increasing immunity

to them. A combination of outdoor air and sunlight could also reduce the likelihood of secondary respiratory infections.

The current H5N1 avian influenza virus has high virulence and lethality but as yet is not readily transmitted from person to person.⁶⁴ We do not know how virulent the next type A pandemic will be, but should it prove to be as pathogenic as that of 1918, there could be 180 million to 360 million deaths globally.65 Vaccines, antiviral drugs, and antibiotics may be effective in controlling avian influenza and dealing with secondary infection; however, for much of the world's population, access to them will be limited. In many countries, the only viable strategy would be to disrupt the transmission of the virus by banning public gatherings, closing schools, isolating infected people, and wearing surgical masks, as was the case during the 1918-1919 pandemic. 66,67

Epidemiological studies show that the wearing of masks in public places in Hong Kong and Beijing during the SARS outbreak was associated with a lower incidence of infection. 68,69 However, no controlled studies have been undertaken to assess the effectiveness of surgical masks in preventing influenza from passing from one host to the next. 70 In addition, it is uncertain whether transmission of the influenza virus from person to person is chiefly by large droplets or aerosols. If droplets are the main mode of transmission, the isolation of patients in private rooms and the use of ordinary surgical face masks may suffice.63 If airborne transmission is significant, reusable respirators could be pivotal in preventing infection, because surgical masks do not offer reliable protection from

aerosols.71,72 Also, measures that prevent the influenza virus from spreading through buildings would assume greater importance. Improvements in air-handling equipment, portable filtration units, and the introduction of physical barriers in the form of partitions or doors may offer some protection.⁷³

However, more might be gained by introducing high levels of natural ventilation or, indeed, by encouraging the public to spend as much time outdoors as possible. It might also be prudent to stockpile tents and beds, because hospitals in the United Kingdom, the United States, and elsewhere are not prepared for a severe pandemic.74-80 Temporary accommodation would be required to deal with the most seriously ill, just as it was in 1918. The Camp Brooks Open Air Hospital might serve as a useful model.

About the Author

At the time of the study, Richard A. Hobday was with the School of the Built and Natural Environment, University of the West of England, Bristol. John W. Cason was with the Department of Infectious Diseases, King's College London School of Medicine, London, England.

Requests for reprints should be sent to Richard Hobday, PhD, Department of Architectural Studies, University of Wales Institute, Cardiff, Llandaff Campus, Western Avenue, Cardiff, CF5 2YB, United Kingdom, and Natural Environment, University of the West of England, Frenchay Campus, Coldharbour Lane, Bristol BS16 1QY, England (e-mail: ra.hobday@virgin.net)

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Contributors

R.A. Hobday originated the study and led the writing. J. W. Cason assisted with the study and analyses. Both authors conceptualized ideas, interpreted findings, and reviewed drafts of the article.

REFERENCES

1. Crosby AW. America's Forgotten Pandemic: The Influenza of 1918. 2nd ed. Cambridge, England: Cambridge University Press; 2003.

- 2. Johnson NP, Mueller J. Updating the accounts: global mortality of the 1918-1920 "Spanish" influenza pandemic. Bull Hist Med. 2002;76:105-115.
- 3. Johnson N. Britain and the 1918-19 Influenza Pandemic. A Dark Epilogue. Oxford, England: Routledge; 2006.
- 4. Barry JM. The site of origin of the 1918 influenza pandemic and its public health implications. J Transl Med. 2004:2:3.
- 5. Morens DM, Fauci AS. The 1918 influenza pandemic: insights for the 21st Century. J Infect Dis. 2007;195:1018-
- 6. Barry JM. The Great Influenza: The Epic Story of the Greatest Pandemic in History. London, England: Penguin;
- 7. French H. The clinical features of the influenza epidemic of 1918-19. In: Great Britain Ministry of Health, Reports on Public Health and Medical Subjects No. 4: Report on the Pandemic of Influenza, 1918-19. London, England: His Majesty's Stationery Office; 1920: 66 - 109
- 8. Patterson KD. The influenza pandemic of 1918-19 in the Gold Coast. I Afr Hist. 1983;24:485-502.
- 9. Mills ID. 1918-1919 influenza pandemic: the Indian experience. Indian Econ Soc Hist Rev. 1986;23:1-40.
- 10. Cheng KF, Leung PC. What happened in China during the 1918 influenza pandemic? Int | Infect Dis. 2007:11:360-364
- 11. Tognotti E. Scientific triumphalism and learning from facts: bacteriology and the "Spanish flu" challenge of 1918. Soc Hist Med. 2003;16:97-110.
- 12. Anon. Weapons against influenza. Am J Public Health. 1918;8:787-788.
- 13. Starr I. Influenza in 1918: recollections of the epidemic in Philadelphia. 1976 Ann Intern Med 2006:145: 138-140.
- 14. Carnwath T. Lessons of the influenza pandemic 1918. J State Med. 1919;27:142-157.
- 15. Schoch-Spana M. "Hospital's fullup": the 1918 influenza pandemic. Public Health Rep. 2001;116(suppl 2):
- 16. Markel H, Lipman HB, Navarro JA, et al. Nonpharmaceutical interventions implemented by US cities during the 1918-1919 influenza pandemic. JAMA. 2007;298:644-654.
- 17. Hatchett RJ, Mecher CE, Lipsitch M. Public health interventions and epidemic intensity during the 1918 influenza pandemic. Proc Natl Acad Sci USA 2007;104:7582-7587.

- 18. Bootsma MJ, Ferguson NM. The effect of public health measures on the 1918 influenza pandemic in US cities. *Proc Natl Acad Sci USA*. 2007;104: 7588–7593.
- 19. Halloran ME, Ferguson NM, Eubank S, et al. Modeling targeted layered containment of an influenza pandemic in the United States. *Proc Natl Acad Sci USA*. 2008:105:4639–4644.
- 20. St Clair Strange FG. *The History of the Royal Sea Bathing Hospital Margate 1791–1991*. Rainham, England: Meresborough Books; 1991.
- 21. Jones ER. Lettsom and the Royal Sea Bathing Hospital. *Trans Med Soc Lond.* 1973;89:285–287.
- 22. Bodington G. An Essay on the Treatment and Cure of Pulmonary Consumption, On Principles Natural, Rational and Successful. London, England: Simpkin, Marshall, Hamilton and Kent; 1906.
- 23. Cyriax RJ. George Bodington: the pioneer of the sanatorium treatment of pulmonary tuberculosis. *Br J Tuberc*. 1925;19:1–16.
- 24. Keers RY. Two forgotten pioneers— James Carson and George Bodington. *Thorax.* 1980;35:483–489.
- 25. McCarthy OR. The key to the sanatoria. *J R Soc Med.* 2001;94:413–417.
- 26. Nightingale F. *Notes on Hospitals*. 3rd ed. London, England: Longman, Green, Longman, Roberts and Green; 1862
- 27. Nightingale F. *Notes on Nursing;* What It Is and What It Is Not. New York, NY: Dover Publications; 1969.
- 28. Smith FB. *The Retreat of Tuberculosis* 1850–1950. London, England: Croom Helm; 1988.
- 29. Connolly C. Pale, poor, and "pretubercular" children: a history of pediatric antituberculosis efforts in France, Germany, and the United States, 1899–1929. *Nurs Ing.* 2004;11:138–147.
- 30. Mera FE. History of the sanatorium movement in America. *Chest.* 1935; 1:8–9.
- 31. Carter AJ. A breath of fresh air. *Proc R Coll Physicians Edinb.* 1994;24:397–405.
- 32. Dormandy T. *The White Death: A History of Tuberculosis*. London, England: Hambledon Press; 1999.
- 33. Cook GC. Early use of "open-air" treatment for "pulmonary phthisis" at the Dreadnought Hospital, Greenwich, 1900–1905. *Postgrad Med J.* 1999;75: 326–327.
- 34. Anon. A military open-air hospital. *Br Med J.* 1915;1:1015–1016.
- 35. Shipley AE. The Open-Air Treatment of the Wounded (The First Eastern

- General Hospital). 2nd ed. London, England: Country Life Library; 1915.
- 36. Moynihan B. An address on the treatment of gunshot wounds. *Br Med J.* 1916:1:333–339.
- 37. Anon. Influenza at the Camp Brooks Open Air Hospital. *JAMA*. 1918;71: 1746–1747.
- 38. Brooks WA. The open air treatment of influenza. *Am J Public Health.* 1918;8: 746–750.
- 39. Hill AB, Hill B. The life of Sir Leonard Erskine Hill FRS (1866–1952). *Proc R Soc Med.* 1968;61:307–316.
- 40. Hill LE, Campbell A. *Health and Environment*. London, England: Edward Arnold & Co.; 1925.
- 41. Hill LE. The defence of the respiratory membrane against influenza, etc. *Br Med J.* 1919;1:238–240.
- 42. Li Y, Leung GM, Tang JW, et al. Role of ventilation in airborne transmission of infectious agents in the built environment—a multidisciplinary systematic review. *Indoor Air.* 2007;17:2–18.
- 43. Tang JW, Li Y, Eames I, Chan PK, Ridgway GL. Factors involved in the aerosol transmission of infection and control of ventilation in healthcare premises. *J Hosp Infect.* 2006;64: 100–114.
- 44. Escombe AR, Oeser CC, Gilman RH, et al. Natural ventilation for the prevention of airborne contagion. *PLoS Med.* 2007;4:e68.
- 45. Hollaender A, Oliphant JW. The inactivating effect of monochromatic ultraviolet radiation on influenza virus. *J Bacteriol.* 1944:48:447–454.
- 46. Tamm I, Fluke DJ. The effect of monochromatic ultraviolet radiation on the infectivity and hemagglutinating ability of the influenza virus type A strain PR-8. *J Bacteriol.* 1950;59: 449–461
- 47. Jensen MM. Inactivation of airborne viruses by ultraviolet irradiation. *Appl Microbiol.* 1964;12:418–420.
- 48. Jakab GJ, Knight ME. Decreased influenza virus pathogenesis by infection with germicidal UV-irradiated airborne virus. *Environ Int.* 1982;8:415–418.
- 49. Sagripanti JL, Lytle CD. Inactivation of influenza virus by solar radiation. *Photochem Photobiol.* 2007;83: 1278–1282
- 50. Hockberger PE. A history of ultraviolet photobiology for humans, animals and microorganisms. *Photochem Photobiol.* 2002;76:561–579.
- 51. Beauchemin KM, Hays P. Dying in the dark: sunshine, gender, and outcomes in myocardial infarction. *J R Soc Med.* 1998;91:352–354.

- 52. Beauchemin KM, Hays P. Sunny rooms expedite recovery from severe and refractory depressions. *J Affect Disord.* 1996;40:49–51.
- 53. Benedetti F, Colombo C, Barbini B, Campori E, Smeraldi E. Morning sunlight reduces length of hospitalization in bipolar depression. *J Affect Disord*. 2001;62:221–223.
- 54. Dobbs RH, Cremer RJ. Phototherapy. *Arch Dis Child.* 1975;50:833–836.
- 55. Barss P, Comfort K. Ward design and jaundice in the tropics: report of an epidemic. *Br Med J (Clin Res Ed)*. 1985;291:400–401.
- 56. Walch JM, Rabin BS, Day R, Williams JN, Choi K, Kang JD. The effect of sunlight on postoperative analgesic medication use: a prospective study of patients undergoing spinal surgery. *Psychosom Med.* 2005;67:156–163.
- 57. Hobday RA. *The Light Revolution: Health Architecture and the Sun.* Forres, Scotland: Findhorn Press; 2006.
- 58. Cannell JJ, Vieth R, Umhau JC, et al. Epidemic influenza and vitamin D. *Epidemiol Infect.* 2006;134:1129–1140.
- 59. Moriarty KJ. The 1918 influenza pandemic: a survivor's tale. *BMJ*. 2006;332;889.
- 60. Chan WF, Wong TK. Preparing for pandemic influenza: revisit the basics. *J Clin Nurs*. 2007;16:1858–1864.
- 61. Seto WH, Tsang D, Yung RW, et al. Advisors of Expert SARS Group of Hospital Authority. Effectiveness of precautions against droplets and contact in prevention of nosocomial transmission of severe acute respiratory syndrome (SARS). *Lancet.* 2003;361:1519–1520.
- 62. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol.* 1979; 110:1–6.
- 63. Oshitani H. Potential benefits and limitations of various strategies to mitigate the impact of an influenza pandemic. *J Infect Chemother.* 2006;12: 167–171.
- 64. Gambotto A, Barratt-Boyes SM, de Jong MD, Neumann G, Kawaoka Y. Human infection with highly pathogenic H5N1 influenza virus. *Lancet.* 2008; 371:1464–1475.
- 65. Osterholm MT. Preparing for the next pandemic. *N Engl J Med.* 2005; 352:1839–1842.
- 66. Toner E. Do public health and infection control measures prevent the spread of flu? *Biosecur Bioterror*. 2006;4:84–86.
- 67. Low DE. Pandemic planning: non-pharmaceutical interventions. *Respirology*, 2008;13(suppl 1):S44–S48.

- 68. Wu J, Xu F, Zhou W, et al. Risk factors for SARS among persons without known contact with SARS patients, Beijing, China. *Emerg Infect Dis.* 2004; 10:210–216.
- 69. Lau JT, Tsui H, Lau M, Yang X. SARS transmission, risk factors, and prevention in Hong Kong. *Emerg Infect Dis.* 2004;10:587–592.
- 70. Bell DM, World Health Organization Writing Group. Non-pharmaceutical interventions for pandemic influenza, national and community measures. *Emerg Infect Dis.* 2006;12:88–94.
- 71. Tellier R. Review of aerosol transmission of influenza A virus. *Emerg Infect Dis* 2,006:12:1657–1662
- 72. Weiss MM, Weiss PD, Weiss DE, Weiss JB. Disrupting the transmission of influenza A: face masks and ultraviolet light as control measures. *Am J Public Health*. 2007;97(suppl 1):S32–S37.
- 73. Morse SS, Garwin RL, Olsiewski PJ. Next flu pandemic: what to do until the vaccine arrives? *Science*. 2006;314:929.
- 74. Bartlett JG. Planning for avian influenza. *Ann Intern Med.* 2006;145:141–144.
- 75. Toner E, Waldhorn R, Maldin B, et al. Hospital preparedness for pandemic influenza. *Biosecur Bioterror*. 2006;4: 207–217.
- 76. Menon DK, Taylor BL, Ridley SA. Modelling the impact of an influenza pandemic on critical care services in England. *Anaesthesia*. 2005;60:952–954.
- 77. Sobieraj JA, Reyes J, Dunemn KN, et al. Modeling hospital response to mild and severe influenza pandemic scenarios under normal and expanded capacities. *Mil Med.* 2007;172: 486–490.
- 78. Giacomet V, Tarallo L, De Marco G, Giannattasio A, Barbarino A, Guarino A. Preparing for an influenza pandemic in Italy: resources and procedures in paediatric hospital units. *Euro Surveill*. 2007;12:E7–E8.
- 79. De Cauwer HG, Mortelmans LJ, d'Orio V. Are Belgian hospitals prepared for an H5N1-pandemic? *Eur J Emerg Med.* 2007;14:204–206.
- 80. Brundage JF. Interactions between influenza and bacterial respiratory pathogens: implications for pandemic preparedness. *Lancet Infect Dis.* 2006;6:303–312.