INFLUENZA 1918: THE CONTEMPORARY PERSPECTIVE

JOHN H. WALTERS, M.D.

Clinical Assistant Professor
Department of Family and Community Medicine
University of Missouri-Columbia School of Medicine
Columbia, Missouri

"The men are dying like flies out there, anyway. This funny new disease. Simply knocks you into a cocked hat." "It seems to be a plague, something out of the middle ages. Did you ever see so many funerals, ever?" Catherine Ann Porter: *Pale Horse, Pale Rider*. New York, Harcourt Brace and World, 1936.

IN October 1918 the obituaries in the *Journal of the American Medical Association* showed a sudden threefold rise in deaths, a frightful mortality due entirely to influenza and pneumonia among younger American physicians.

In other years, physicians 30 years of age or younger accounted for fewer than 1.5% of all physician deaths: normally fewer than 10% of the deaths were in the 31 to 40 age group and fewer than 20% between 41 and 50. Now 39% of all deaths among physicians occurred before 40 (13% in those under 30) from influenza and pneumonia. During comparable periods in 1916 and 1919, such deaths were fewer than 2% of the total and none under age 30 died from either. Older doctors seemed less at risk. Only 12.5% of the deaths were due to influenza and pneumonia among those between 41 and 50 and only 9% in those over 51.[1]

Losses fell most heavily not on the weak but on the strong; nearly half the deaths from influenza and pneumonia were among men and women in the prime of life, between 20 and 40, and the disease in 1918 occurred most frequently among children five to 14 years old. The greatest losses in proportion to population fell between 25 and 29 years of age.[2] The chances of surviving influenza were good, but because, at least in the United States,one in every four people contracted influenza, the death toll was enormous.[3]

Spanish influenza had come to the United States. The youngest physi-

Address for reprint requests: 1027 Hickory Hill Drive, Columbia, Mo. 65201.

cians, lacking the immunity that the 1889-1890 influenza pandemic had conferred, were, along with nurses and other young hospital staff, the most exposed and most susceptible potential victims.

From 1914 to 1918 most nations exhausted their resources in killing young men, yet nature outstripped statesmen: influenza killed more victims in a few months than all the armies in four years. In the United States between half a million[2] and more than one and a half million[4] died from it. In India six months of influenza caused nearly as many deaths as 20 years of cholera. The mortality in October 1918 topped all years of pestilence: appraisals of worldwide mortality range from 10 to 21 million. It was estimated that not fewer than two hundred million and possibly as many as seven hundred million had been sick. The influenza pandemic of 1918 ranks with the plague of Justinian and the black death as one of the three most destructive human epidemics. Four years of war had, by 1918, dulled sensibility to death. All perspectives were distorted,[2] and the world took it in stride.

Disastrous pandemic influenza was not new. An outbreak of a "coughing violence," from which "very many died" was reported in Europe between 1556 and 1560 and at the same time in Japan.[5] Some of the medieval plague epidemics and the later widely reported English and Picardy sweats may well have been influenzal: plague and smallpox were then, probably, often thought to be the same disease: the boils, the sores, and the stench. But there was also pneumonic plague without boils, and many accounts of the 1918 influenza mention a peculiar stench physicians instantly recognized as influenzal. A violaceous "heliotrope" cyanosis characterized hopeless cases. (The *Lancet* in January 1918 even published one of its rare color illustrations very well showing that peculiar hue.)[6]

Reliable descriptions of a disease similar in pathologic characteristics, if not in severity and spread, to the 1918 epidemic had to wait until late in the 19th century. In 1893 William Pepper described the severe 1889-1890 epidemic[7] and reminisced to his associates about the 1848-1850 outbreak.[8] His pupil, Alfred Stengel, compared the 1889 and the 1918 pandemics. Outbreaks during intervening years had generally been mild and tended to "prove fatal only to old people and to persons weakened by pulmonary complaints but they carried off several hard drinkers."[8]

The chronology of the 1889 and 1918 epidemics could easily be explained by the exceptionally high infectiousness and dissemination no more rapid than the rate of human travel at the time. It spread in Turkestan with

the speed of a caravan, in Europe of an express train, and over the seas with the rapidity of ocean travel.[9]

Ethnic and cultural backgrounds accounted for differences in the susceptibility among immigrant groups in the United States as age differences alone often did among native Americans. Previous exposure to similar epidemics had conferred immunity. Older lifelong city dwellers thus were mostly spared. The movement of native rural Americans to large cities was not extensive, but from the end of the century the number of immigrants of Italian, Russian, Austrian, Polish, and French-Canadian stock increased vastly. Most of these new immigrants were between ages 14 and 44 and many were peasant farmers from isolated, self-contained villages. They were not exposed to influenza until they congregated in crowded quarters in the cities.[10]

American concern with the developing influenza epidemic can be measured by inspection of newspaper files.[11] *The New York Times* reported on June 1, 1918 that a disease resembling influenza was spreading through northern China. On June 21 a Spanish epidemic was said to have come from Germany. And on June 25th and 27th *The New York Times* mentioned the epidemic in the German army and in German cities. On the 28th it stated that no cases had been reported among American troops. Apprehension about influenza became manifest in July 1918, and passengers arriving on boats from Spain were fumigated before being allowed to land. Documents taken from German prisoners of war indicated much illness among German troops. Some now called the disease the "Flanders grippe" and its spread across Europe was rapid. On July 14th the epidemic hit Switzerland hard enough that the authorities prohibited public meetings in Berne, and on July 27th 305 deaths from influenza were reported by the Swiss army, which had mobilized but was not involved in the war. Increasing numbers of cases were reported in August on ships arriving in New York from abroad, mostly from England and Norway. But in mid-August the New York City health commissioner still insisted that no danger of an epidemic existed in New York. Army camps were struck in mid-September, and influenza was then made reportable in New York City.[11]

Of a total camp population of 33,044 at Camp Sherman, Ohio, 7,618 were admitted to the camp hospital with coryza and bronchitis or more definitive evidence of influenza; 2,001 developed pneumonia and 842 soldiers died there between September 26 and October 11, 1918.[12] One

thousand cases had occurred at the Great Lakes Naval Training Station by September 20 and on September 22, 1918 the number had risen to 2,000. The *Olympic* arrived in port with 5,951 troops. On the day of arrival, 571 cases of acute respiratory disease developed and within three weeks there were 1,668 cases. Of these, 532 (32%) had pneumonia, of whom 317 (19% of all cases) died; 5.3% of all troops on the *Olympic* died of influenza.[13] Army intelligence—ever alert—suspected that the disease had been brought to our shores by German U-boats.[11]

The pathologist, Eugene L. Opie, followed the influenza epidemic throughout its course at Camp Pike, Ark. A camp housing approximately 50,000 men had almost 12,000 cases of influenza, 1,499 with pneumonia and 466 deaths, all from pneumonia. Opie later recalled that early in the epidemic places prepared for the dead were completely occupied and bodies had to be stacked one upon the other in the mortuary from floor to ceiling.[14]

In October 1918 the pandemic peaked. Sierra Leone in Africa reported 1,000 cases a week. There was similar distress in Argentina and Brazil. The disease spread rapidly across the United States; Boston was the first major city hard hit, then New York. Physicians and nurses rushed to Boston to help. Three thousand people were reported stricken in Camden, N.J. Outbreaks in Washington, Chicago, New Orleans, Los Angeles, Seattle, and San Francisco soon followed.[2]

Reisman confessed that Philadelphia, as many other centers, was unprepared for the great emergency. There were not nearly enough doctors and nurses. Available hospital space was quickly occupied, drugs and supplies were used up and undertakers ran out of coffins. Many patients suffered and died without medical aid.[15] Two hundred bodies had to be crowded in a morgue built for 36 and a street railway repair shop was turned into a coffin factory. Baltimore, too, lacked coffins. *The New York Times* reported unburied dead in Queens cemeteries. The mayor asked the street commissioner to send street cleaners to help dig graves (*The New York Times*, October 26, 1918). Cardinal Gibbons forbade public funerals in the Baltimore archdiocese (October 11, 1918), and Chicago arrested people who did not use handkerchiefs when they sneezed.[11] By December 20 deaths in the past three months had, according to *The New York Times*, reached six million. Reports of the seemingly unending spread of the disease and its proximate and distant effects poured in from all sides. Denmark reported mortality high and drugs scarce. Coffee shipments from

Brazil were delayed. Senator James Hamilton Lewis introduced a bill appropriating $10,000,000 to fight the disease although no one knew how to do it. The Association of Life Insurance Presidents worried about costs to their companies, and the Temperance League protested the medicinal use of whiskey. Express trains were suspended because a third of the trainmen were sick, and all public gatherings not absolutely essential to war work were prohibited. On November 9th Mayor Kiel suspended all nonessential business in St. Louis for a week.[11]

Hopes rose late in October that the Boston epidemic was subsiding and were dashed in December, when another rise in the epidemic wave was reported. The Mexican government quarantined all entrance points from the United States. Places of worship and theaters were closed. The New York health commissioner, an unabashed optimist throughout the epidemic, refused to close the public schools in the city and repeated at weekly intervals assurances to the public—in spite of all evidence to the contrary—that the epidemic was just about over. In the last third of December 1918 his predictions became true; both the extent of the epidemic and the severity of the illness declined and, except for minor and less lethal flareups, the epidemic faded away.[11]

At the center of the nation, in Columbia, Mo., the University of Missouri opened for classes on August 30, 1918, with a total enrollment of 2,706. Of these, 1,185 were nonmilitary, mostly women; 1,521 military members were housed in barracks. The first cases of influenza were admitted to the 50-bed University Hospital on September 26th and by October 8th that 50-bed facility and an 84-bed rented facility which had been equipped in the interval were both crowded. By October 11th another hospital of 125 beds, a 50-bed hospital for women, and a 40-bed convalescent hospital had all been added. Only three full-time physicians were available for all these patients and four graduate and 13 pupil nurses were reinforced by only three additional nurses—all that were available—and by soldiers detailed to help. Between September 26, 1918 and December 6, 1918 1,020 patients with influenzal pneumonitis, 37% of all enrolled students, were admitted, and 14 students died. In this University of Missouri epidemic, then, 1.5% of all influenza victims, or 0.5% of all enrolled students, had died within less than two and one-half months,[16] all young, healthy people.

The disease, vast in the morbidity it caused (between two hundred million and seven hundred million people were estimated to have fallen ill),

actually had a relatively low mortality, less than 2%. But many more victims were stricken than were ever afflicted by plague, smallpox, or cholera. Those pestilences, all with a much higher risk of mortality, never had nearly so many victims, and therefore killed substantially fewer than the 1918 influenza epidemic.

Predominantly and hardest hit by the disease were the young. Most people who had lived through the 1889-1890 epidemics—whether or not they had been clinically ill—escaped infection or suffered only a mild illness, as had those in 1889 who had presumably been exposed to a similar illness in 1848-1850.

What was the clinical picture of influenza in 1918? Ill-defined respiratory disorders were scattered all over the world in 1917 and early 1918 and had been simply regarded as the usual winter colds. During the winter of 1917 an increase in fatal cases of purulent bronchitis was recorded at a base hospital in France.[17] During the late spring and summer a three-day fever, striking suddenly and bringing aching lassitude but leading to uneventful recovery, was widespread. This was commonly thought to be "la grippe" (influenza). Ordinary influenza and the 1918 variety were similar except for severity and extent of complications. The latter's extreme contagiousness, huge number of victims, and serious complications accounted for the immense number of deaths. Descriptions of influenza in medical texts and contemporary literature differ little over the past 80 years.[7,8,13,18,19,20]

The disease became suddenly virulent in September 1918. Although most who were stricken recovered from fever and aches uneventfully, a significant minority (estimated at one out of five in England) suddenly developed severe pneumonia.

When the lips, ears, and face turned an ashy heliotrope purple,[6] death was almost certain. Bilateral ptosis—the lids half closed—was equally ominous. In an overwhelming number, pneumonia arose early, sometimes during the first 24 hours, sometimes not until the second or third day. Pulmonary edema, with crackling moist rales, was often evident and almost never absent in the bad cases. Pleurisy, common in lobar pneumonia, was infrequent. Before this epidemic, bronchopneumonia in young healthy adults between 20 and 35 years of age had been quite unusual, but it had suddenly become common and often hemorrhagic.

Epidemic influenza usually began suddenly, with general malaise, pain in the legs, head and eyeballs, and backaches—often very severe. Chilli-

ness was more common than actual chills. Coughs varied in intensity. In some it became more violent and laborious than that of whooping cough. The sputum was frequently thin, rusty, reddish, or salmon colored, and occasionally resembled prune juice. Where bloody discoloration was seen, pneumonia was invariably present.[15] Temperature elevations to 103° or 104° F. were usual and hyperpyrexia common. The pulse tended to be relatively slow, as was respiration even with extensive lung disease. Preterminally, however, both pulse and respirations became rapid. Insomnia was very common and often intractable. Delirium, sometimes violent, was frequent. It was common to see a typical "typhoidal state": cracked lips, dry, glazed brown, shrunken, tremulous tongue, lint-picking fingers, incontinence of urine and feces, and semiconscious stupor. Even though patients often would not complain and, when asked, say they felt splendid, the ominous violaceous heliotrope hue, the half-closed, seemingly ptotic lids, and a peculiar stench—readily recognized by the then all too experienced physicians as typical of influenza—told a different story. The peculiar discoloration was never adequately explained. Methemo- and carboxyhemoglobinemia were suspected but not documented by spectroscopic blood examinations. It was likely a product of anoxic cyanosis combined with peripheral vascular changes. To Riesman the color suggested an altered state of the blood analogous to that of acetanilid poisoning, and its extent was proportional to the degree of toxemia.[15] The heliotrope cyanosis blanched on pressure and, when present, was sometimes confined only to lips, earlobes, and fingernails. In severe cases, however, it was general over the face, trunk, and extremities. The lips and mucous membranes then often were maroon and, occasionally, the face and neck were livid, bluish-black—a discoloration neither attributed to circulatory failure nor directly related to the extent of the pneumonia.

The characteristic stench, which reminded some of rotting corpses on the battlefields, also defied rational explanation. The sputum or the breath were not its cause, nor was flatus. While more noticeable when bedclothes were pulled down, it did not persist when the bodies were placed in the morgue, even if refrigeration was not available.[21] Meningismus was rare, and abnormal cells were absent from the spinal fluid. Diuresis was generally free, but hemorrhagic nephritis with marked albuminuria was not uncommon and occasionally urinary retention developed. Herpes simplex and erythemas were rare. Nasal, upper gastrointestinal, and uterine bleeding were common. Blood in the sputum was usual and at times distinct

hemorrhages, as with pulmonary tuberculosis or infarction, were seen. Phlebitis was rare.[15] When the disease occurred in a pregnant woman it almost always proved fatal and infection seemed to require minimal exposure.

The contemporary view of the epidemic was naturally determined by the state of the art at the time. This report cannot discuss problems still not entirely solved of the exact etiology of this pandemic. Most thought that *Hemophilus influenzae* played some role, although filtered secretions reproduced the disease in fearless volunteers. Secondary pneumococcal and streptococcal invasions were certainly frequent, but do not account for the rapid downhill course; hours or one or two days from good health to death were not at all uncommon. Antibiotics might not drastically reduce mortality today were we faced by a similar situation.

Extensive confluent bronchopneumonia and often hemorrhagic and purulent changes of the entire respiratory tract were present at autopsy. In the average case more than 50% and frequently as much as 90% of the lung tissue was involved.[12]

The vast majority of patients recovered completely, although not always quickly. Myocarditis occasionally delayed recovery and caused some permanent invalidism. Postinfluenzal bronchiectasis was not uncommon.

Prostration was characteristic of influenza and mental depression a hallmark of convalescence. Very few cases failed to show at least some, but the severity of the depression was often no measure of the seriousness of the preceeding infection nor did it seem related to the patient's basic temperament. Exhaustion was physical and mental: insomnia, mental aberrations, delusions, irritability, and improper judgment were common. The reasoning of writers and scientists often proved erratic and their technique faulty. Most characteristic was inability to work, to concentrate, or to sustain an effort. While the general tendency was toward depression and inaction, a few showed active psychotic behavior, ungovernable temper, hysterical outbursts, aggressive irritability, and quarrelsomeness.[22,23] *The New York Times* of December 16, 1918 reported that Doctor J. S. Gentile had been killed by a patient who was delirious from influenza.[11]

The relation of the 1918 influenza pandemic to epidemic encephalitis lethargica has frequently been discussed. That pandemic preceded the 1918 influenza outbreak by at least one year and outlasted it in repeated epidemic waves until 1926.[24] Association between these diseases has often been suggested[25] but never documented.[26,27] Gamboa and her colleagues in

1974 demonstrated various influenza A antigens with fluorescent antibody studies in the brains of patients with postencephalitic Parkinsonism.[28] No swine-influenza antigens could be found in any of these brains.

The present assumption that swine influenza was the virus responsible for the 1918 pandemic seems well established by serologic studies. Genetic variants of the influenza virus carry with them the implied threat of a repetition of the 1918 worldwide disaster. Kilbourne[29] concluded that the swine-influenza virus, should it be the next pandemic strain, may be less virulent, as virulent, or more virulent than the virus of the great pandemic. There is little doubt that in our present era of almost instant transportation it could spread rapidly and without forewarning over the entire globe.[9]

SUMMARY

Almost 60 years ago a great influenza pandemic ravaged humanity, and it ranks with the plague of Justinian and the medieval black death as one of the three most devastating plagues to ever strike mankind. The impact of the epidemic on the medical profession and on the public at large are reviewed, its peculiar clinical manifestations discussed, and need for concern about a similar pandemic in the future appraised.

REFERENCES

1. Deaths. *J.A.M.A. 67*:1174-75, 1242-43, 1315-16, 1384, 1916, *J.A.M.A. 71*:1334-35, 1428-30, 1505-09, 1573-79, 1918.
2. Smith, G.: *Plague on Us.* New York Commonwealth Fund, 1943, pp. 24-33.
3. Frost, W. H.: The epidemiology of influenza. *J.A.M.A. 73*:313-18, 1919.
4. Mitford, J.: *The American Way of Death.* New York, Simon and Schuster, 1963, p. 52.
5. McNeill, W. H.: *Plagues and Peoples.* New York, Anchor Press/Doubleday, 1976, pp. 288-89.
6. Abrahams, A., Hallows, N., and French, H.: A further investigation into influenza pneumococcal and influenza streptococcal septicemia. *Lancet I*:1-11, 1919.
7. Pepper, W.: *An American Textbook of the Theory and Practice of Medicine.* Philadelphia, Saunders, 1893, vol. I, pp. 184-96.
8. Stengel, A.: The influenza epidemics of 1889 and 1918. *Med. Clin. N.A. 2*:645-70, 1918.
9. von Bergmann, G.: Die Spanische Krankheit ist Influenza Vera. *Deutsche Med. Wschr. 44*:933-35, 1918.
10. Katz, R. S.: Influenza, 1918-19: A study in mortality. *Bull. Hist. Med. 48*:416-22, 1974.
11. *The New York Times Index*, April-June 1918, p. 192; July-September 1918, p. 184, October-December 1918, pp. 206-208; January-March 1919, p. 220; October-December, 1920, p. 190.
12. Friedlander, A., McCord, C. P., Sladen, F. J., and Wheeler, G. W.: The epidemic of influenza at Camp Sherman, Ohio. *J.A.M.A. 71*:1652-56, 1918.
13. Osler, W. and McCrae, T.: *The Principles and Practice of Medicine,* 9th ed. New York and London, Appleton, 1922, chap. X, pp. 118-22.
14. Opie, E. L.: The peripatetic education of a pathologist. *Med. Clin. N. A.*

*41:*942, 1957.
15. Riesman, D.: Influenza—Remarks upon symptoms, prevention and treatment. *Med. Clin. N.A. 2:*903-20, 1918.
16. Stine, D. G.: A comparison of the influenza epidemic of 1918 with that of 1920 at the University of Missouri. *J. Missouri State Med. Assoc. 18:*117-23, 1921.
17. British Medical Research Committee: Influenza. *J.A.M.A. 71:*1573-79, 1918.
18. Blattner, R. J.: In: *Textbook of Pediatrics*, 10th ed., Nelson, W. E., editor. Philadelphia, Saunders, 1975, pp. 682-85.
19. Citron, J.: Das Klinische Bild der Spanischen Grippe. *Berliner Klin. Wschr. 55:*777-78, 1918.
20. Holt, L. E. and Howland, J.: *Diseases of Infancy and Childhood,* 9th ed. New York, Appleton, 1929, pp. 947-49.
21. Abrahams, A.: In: *Influenza—Essays by Several Authors,* Crookshank, F. G., editor. London, Heinemann, 1922, chap. IX, pp. 314-50.
22. Brooks, H.: The complications and sequelae of influenza. *Med. Clin. N.A. 4:*29-58, 1920.
23. Burr, C. W.: The mental complications and sequelae of influenza. *Med. Clin. N.A. 2:*709-18, 1918.
24. Walters, J. H.: Encephalitis lethargica revisited. *J. Oper. Psychiat. 8:*37-46, 1977.
25. Weinstein, L.: Influenza—1918. A revisit. *N. Engl. J. Med. 294:*1058-60, 1976.
26. Yahr, M. D.: Swine flu letters to the editor. *N. Engl. J. Med. 295:*284-85, 1976.
27. Weinstein, L.: Swine flu letters to the editor. *N. Engl. J. Med. 295:*284-85, 1976.
28. Gamboa, E. T., Wolf, A., Yahr, M.D., et al.: Influenza virus antigen in post-encephalitic Parkinsonian brain. *Arch, Neurol. 31:*228-32, 1974.
29. Kilbourne, E.D.: Influenza pandemics in perspective. *J.A.M.A. 237:*1225-28, 1977.