1 The New Flu

The proximate beginning of this story is abrupt. On the East Coast of the United States, January 1976 was very cold. At Fort Dix, New Jersey, training center for Army recruits, new men fresh from civilian life got their first taste of barracks and basics. A draft of several thousand came in after New Year's Day to be instructed by a cadre back from Christmas leave. The fort had been almost emptied; now in the cold it was full again. By mid-January many men began reporting respiratory ailments. A relative handful were hospitalized. One, refusing hospitalization, went on an overnight hike and died.

After a county medical meeting on another subject, the state's chief epidemiologist bet the senior Army doctor that Fort Dix was in the midst of an influenza virus epidemic. To win, the latter sent a sample set of cultures for analysis in the state laboratory. He lost. The lab turned up several cases of flu traceable to the Victoria virus which had been since 1968 the dominant cause of human influenza.[1] But the lab also found other cases of flu caused by a virus it could not identify. With foreboding, Dr. Martin Goldfield, the civilian epidemiologist, sent those cultures to Atlanta, to the Federal government's Center for Disease Control (CDC). A similar virus, also unidentified, was isolated from the dead man and a culture sent to CDC. In the evening of February 12, the Center's laboratory chief, Dr. Walter Dowdle, reported the result to his superiors—in four cases including the fatality, the unknown was swine flu. At CDC this caused more concern than surprise.

Four things combined to create the concern. First, these four recruits could have been infected through human-to-human transmission. Not since the late 1920's had this form of influenza been reported in as many persons out of touch with pigs. There might have been a number of occasions unreported; no one knew. Second, for a decade after World War I a virus of this sort was believed to have been the chief cause of flu in human beings. Since then it had confined itself to pigs. Were it returning now to humans, none younger than 50 would have built up specific antibodies from previous infection. Third, the Fort Dix virus differed in both its surface proteins, termed "antigens," from the influenza virus then circulating in the human population. This difference, in expert terms an "antigenic shift," would negate any resistance carried over from

exposure to the other current viruses. In 1976, it was assumed by leading experts that pandemics follow antigenic shifts as night from day.

And finally, in 1918, a pandemic of the swine flu virus, the most virulent influenza known to modern medicine, had, in a so-called "killer wave," been associated with some 20 million deaths worldwide, 500,000 here. Many were taken by bacterial pneumonia, a complication of influenza now treatable with antibiotics, but an unknown number succumbed to the flu itself. Among the hardest hit then had been able-bodied persons in their twenties and early thirties. Parents of small children died in droves. So did young men in uniform. Virulence cannot as yet be tested in the lab. Could the Fort Dix swine flu be a comparable killer? No one at CDC knew any reason to suppose it was—contrast the 1920's and the circumstances of the one death now—but still. . . .

The absence of surprise reflected expert views at that time about epidemic cycles and about the reappearance of particular types of viruses in people. It was widely thought—on rather scanty evidence—that antigenic shifts were likely about once a decade (interspersed with slighter changes, "drifts," each second or third year). There had been shifts in 1957 and in 1968, both followed by pandemics—Asian flu and Hong Kong flu respectively—and public health officials were expecting another by, say, 1978 or 1979. 1976 was close. The very day the Fort Dix cases were identified at CDC, the *New York Times* carried an Op Ed piece by Dr. Edwin D. Kilbourne, one of the country's most respected influenza specialists, extolling cycles and affirming that pandemics occur every eleven years—another one of which, he warned, was surely coming soon:

> Worldwide epidemics, or pandemics, of influenza have marked the end of every decade since the 1940's—at intervals of exactly eleven years —1946, 1957, 1968. A perhaps simplistic reading of this immediate past tells us that 11 plus 1968 is 1979, and urgently suggests that those concerned with public health had best plan without further delay for an imminent natural disaster.[2]

Also, an influenza virus recycling theory was just then receiving attention, and this suggested swine-type as a likely next strain to appear. The idea was that the flu virus had a restricted antigenic repertoire and a limited number of possible forms, requiring repetition after a time period sufficient for a large new crop of vulnerable people to accumulate. The Asian flu of 1957 was thought to have resembled flu in the pandemic year of 1889. The Hong Kong flu of 1968 was thought to be like that of 1898. Swine flu, absent for 50 years, fit well enough, no surprise. The theory had been originally proposed by two doctors who wrote in 1973:

> A logical sequel to the data presented and supported here would be

> the emergence in man of a swine-like virus about 1985-1991. . . . Regardless of one's view as to the origin of recycling of human strains of influenza, the matter of being prepared to produce swine virus vaccine rapidly should receive consideration by epidemiologists. Man has never been able to intervene effectively to prevent morbidity and mortality accompanying the emergence of a major influenza variant, but the opportunity may come soon.[8]

Though some experts were skeptical about the regularity with which previous strains might be expected to reappear, no one doubted that a swine flu virus might well re-emerge in the human population.

On February 12, alerted by preliminary lab reports, Dr. David Sencer, CDC's Director, asked a number of officials from outside his agency to join him there for a full lab report on February 14. The Army responded as did Goldfield from New Jersey. And from two other parts of CDC's parent entity in HEW, the Public Health Service (PHS), Dr. Harry Meyer and Dr. John Seal came as a matter of course. Meyer was Director of the Bureau of Biologics (BoB) in the Food and Drug Administration; Seal was the Deputy Director of the National Institute for Allergy and Infectious Diseases (NIAID) in the National Institutes of Health. (NIAID's director left these relations to Seal.) The BoB was responsible for licensing and testing flu vaccines, the NIAID for federally sponsored flu research. The duties of Meyer and Seal overlapped, but they were accustomed collaborators. Both were accustomed also to work closely with CDC, its labs and its state services.

Among their recent objects of collaboration had been workshops held at intervals since 1971 on how to better the quite dismal record of 1957 and of 1968 in getting vaccine to Americans ahead of a pandemic. This matter was much on Seal's mind and especially on Meyer's. His bureau had been the subject of a Senate inquiry three years before and needed nothing less than the black-marketing and discrimination characteristic of vaccine distribution in 1957.

To this group, enlarged by CDC staff, Dowdle reported his laboratory findings. The question at once became whether four human cases were the first appearance of incipient pandemic or a fluke of some kind, a limited transfer to a few humans of what remained an animal disease which would not thrive in people. All agreed that on the present evidence there was no means of knowing. Surveillance was the task at hand. Since their uncertainty was real, they agreed also that there should be no publicity until there were more data: why raise public concern about what might turn out an isolated incident? Some days later CDC scrapped this agreement on the plea that uninformed press leaks were imminent, and Sencer called a press conference for February 19. He must have hated the thought that an announcement might come from some place

other than CDC. However that may be, the press conference got national attention.

In the *New York Times* Harold Schmeck reported, February 20:

> The possibility was raised today that the virus that caused the greatest world epidemic of influenza in modern history—the pandemic of 1918-19—may have returned.

This story (on page 1) was headed:

> U.S. Calls Flu Alert On Possible Return of Epidemic Virus

The 1918 reference was included in brief notices that night, on CBS and ABC news telecasts. NBC went them one better and showed 1918 still pictures of persons wearing masks. Lacking further information, the media did not follow up the story for a month. But 1918 left a trace in certain minds, some of them TV producers and reporters. From within CDC, we have encountered a good deal of retrospective criticism at press tendencies to "harp" on 1918 prematurely, with no evidence whatsoever about prospective virulence or even spread through 1976. These NBC pictures are cited along with the *New York Times* headline. But the reference was included in the CDC press briefing and indeed without it what was known about Fort Dix so far was scarcely news at all. What sense to a conference that did not bring it up?

Publicity had no effect upon the effort to establish what the Fort Dix outbreak meant. In Fort Dix itself, where the Army conducted its own investigation shielded from civilians, the Victoria strain proved dominant, at least for the time being. There were plenty of new influenza cases, none was caused by the swine virus. On the other hand, that virus was isolated from a fifth soldier who had been sick in early February, and blood tests confirmed eight more old cases of swine flu, none of them fatal. Moreover, a sampling of antibody levels among recruits suggested that as many as 500 had been infected by swine flu. This implied human transmission on a scale that could not reasonably be viewed lightly. Around Fort Dix, however, in the civilian population—which was Goldfield's territory for investigation—analysis of every case of flu reported, by a medical community on the alert, showed only Victoria. Elsewhere in New Jersey Goldfield's inquiries turned up no swine flu. The Army's inquiries turned up none at camps other than Fort Dix. The NIAID network of university researchers and the state epidemiologists in touch with CDC reported none untraceable to pigs. The World Health Organization, pressed by CDC, could learn of none abroad. One death, thirteen sick men and up to 500 recruits who evidently had caught and resisted the disease, all in one Army camp, were the only established instances of

human-to-human swine flu found around the world as February turned into March, the last month of flu season in the Northern Hemisphere.

On March 10 the group that had met February 14 reassembled at CDC and under Sencer's chairmanship reviewed their findings with the Advisory Committee on Immunization Practices (ACIP). That committee was in form a set of outside experts appointed by the Surgeon General, independently advising CDC; in fact it was almost a part of CDC, nominated, chaired and staffed at Sencer's discretion. BoB deadlines now forced his pace. One ACIP function was to make vaccine recommendations for the next flu season available to manufacturers. The annual questions were: vaccine against what viruses, aimed at which population groups? For 1976 these questions had already been reviewed in a January ACIP meeting. The committee had recommended Victoria vaccine for the "high-risk groups" as then defined, some 40 million people over 65 in age or with certain chronic diseases. By March 10, the four active manufacturers had produced in bulk form about 20 million doses of Victoria vaccine for the civilian market. If Fort Dix meant a change or addition, now was the time to decide. Indeed for a regulatory body like the BoB, responsible for setting standards and for quality control, March was already late. Vaccine is grown in eggs; a vaccine against swine flu would require new supplies replacing those just used for Victoria vaccine. Then immunization trials would be needed if there were a new vaccine, also extensive testing. And what about the vaccine now in bulk? Whatever surveillance had turned up by now would have to suffice for some sort of decision.