Influenza Surveillance, United States, 1960

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DURING the first 3 months of 1960, an influenza epidemic of unexpected severity occurred in the United States. The specific etiologic agent was determined to be the A_2 or Asian strain of influenza virus type A. Many characteristics of this epidemic were in notable contrast with the experience in the Asian influenza pandemic of 1957–58, reported by Trotter and associates (*1*). Such characteristics of the 1960 epidemic included an inability to trace geographic spread, low morbidity rates among school-age children, high morbidity rates among older persons, the intensity of the epidemic in southern California, and the unexpectedly severe excess mortality. This report describes the surveillance information obtained from various sources about the 1960 epidemic and compares its epidemiologic characteristics with those of the pandemic of 1957–58.

Surveillance Methods

The establishment and function of the Influenza Surveillance Unit of the Communicable Disease Center has been described by Trotter and associates (*1*). Surveillance methods used in 1960 were patterned after those developed in 1957–58, although in general less intensive surveillance was maintained.

State reporting. As available, information was provided by State health departments on the location, onset, and extent of influenza epidemics within their respective States. Absenteeism rates in selected schools and industries were provided in many cases, as well as information regarding laboratory identification of etiologic agents or serologic testing of diagnostic value.

U.S. National Health Survey. Special tabulations of the weekly incidence, by age group, of acute respiratory disease involving one or more days in bed were provided by the U.S. National Health Survey. Although not available on a current basis, this information was of material benefit in the retrospective study of the epidemic.

Excess mortality. The number of deaths recorded as due to "influenza" and "pneumonia" (International Statistical Classification Codes 480–483 and 490–493) from the weekly telegraphic reports of 108 large cities with a population of approximately 50,000,000 were made available on a current basis by the National Office of Vital Statistics. These were incorporated weekly into a continuing statistical analysis of excess mortality due to influenza and pneumonia; the method used has been previously described (*1*).

Laboratory data. Information regarding isolation and identification of influenza viruses as well as serologic studies was transmitted from collaborating laboratories to the Respiratory Disease Unit, Laboratory Branch, Communicable Disease Center. The CDC laboratory served as the WHO International Influenza Center for the Americas and a reference diagnostic center. Strains of influenza viruses submitted were subjected to further characterization and comparison with previously isolated strains.

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Other sources of information. Additional current information was supplied by the National Office of Vital Statistics; Epidemic Intelligence Service Officers; members of the Armed Forces Epidemiological Board; and physicians in military, university, and private practice.

During the epidemic period, information available on a current basis was analyzed and distributed in the form of periodic *Influenza Surveillance Reports.*

Epidemiologic Findings

The onset and geographic spread of the epidemic in 1960 could be defined only within very broad limits. Earliest reports of outbreaks came from such widely scattered areas as Brownsville, Tex.; Columbus, Ohio; and Perry County, Tenn. All these outbreaks apparently began during the last week of December 1959 or the first week of January 1960. Following these initial reports, epidemics appeared simultaneously or in rapid succession in almost every geographic region of the country. By February 25, 1960, the disease was rather diffusely and randomly scattered across the United States (fig. 1).

Only in rare instances was it possible to identify any pattern of geographic spread. In California, for example, the epidemic began in the southern areas of the State, with an observable gradual spread northward. A group of university students in Seattle, Wash., who had attended the Rose Bowl game in Pasadena, Calif., on January 1, 1960, became ill with influenza following their return to Seattle. Southern California was at that time experiencing the beginning of an epidemic. With these few exceptions, however, it was impossible to trace any clear patterns of spread during the 1960 epidemic.

In order to estimate the extent and age distribution of influenza morbidity, a questionnaire survey of acute respiratory illness among employees of the Ohio Department of Health and their families was carried out in January 1960 by Dr. Winslow Bashe, chief, division of

Figure 1. Reported influenza, United States, December 1959 through February 25, 1960

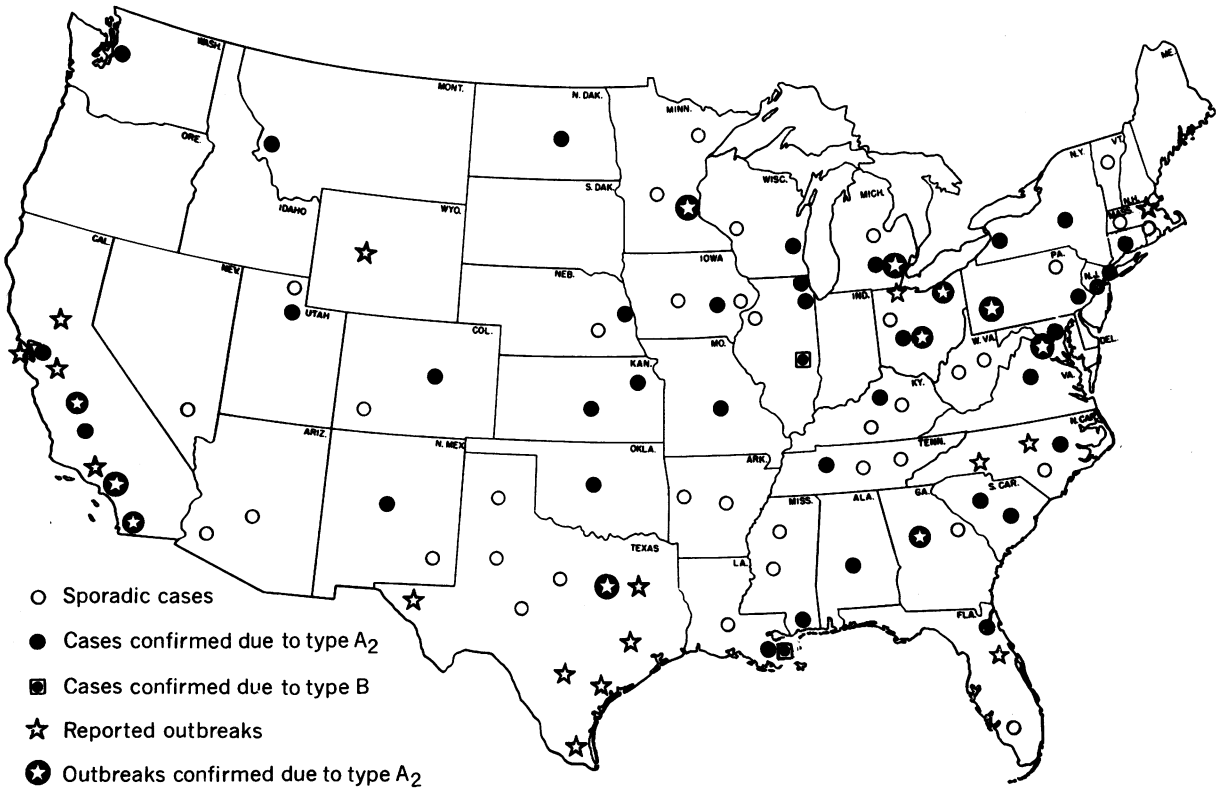


Table 1. Acute respiratory disease questionnaire survey, Ohio Department of Health employees and their families, January 1960

Age group (years)	Number ill	Number at risk	Percent ill
0–9	73	189	39
10–19	15	117	13
20–29	77	158	49
30–39	34	131	26
40 and over	60	239	25
All ages	259	834	31

communicable diseases. The results of the survey are shown in table 1. A striking contrast is apparent between the low attack rate in those 10–19 years of age and the high attack rate among those 20–29 years of age. The epidemic in Columbus began in late December, however, and school vacations may therefore have contributed to the low rate among school-age children by reducing the usual risks of exposure associated with school contacts.

The consensus of information from other State health departments was that school closures were infrequent, and pupil absenteeism in general was not strikingly elevated. In some instances, teacher absenteeism was a more significant factor in school closures than pupil absenteeism. It was the opinion of several State health officers that industrial absenteeism, reflecting the spread of the disease in the adult population, seemed disproportionately high when compared with school absenteeism. The epidemic in southern California was reported by State health department authorities to be unusually severe, particularly in the Los Angeles metropolitan area. There was marked involvement of the elderly population, especially those in nursing homes and old-age homes.

U.S. National Health Survey

Figure 2 depicts the weekly incidence of respiratory disease involving one or more bed-days of illness, by age group, for the period July

Figure 2. Weekly incidence of respiratory illnesses, United States, case rates by age group, July 1957–May 1960

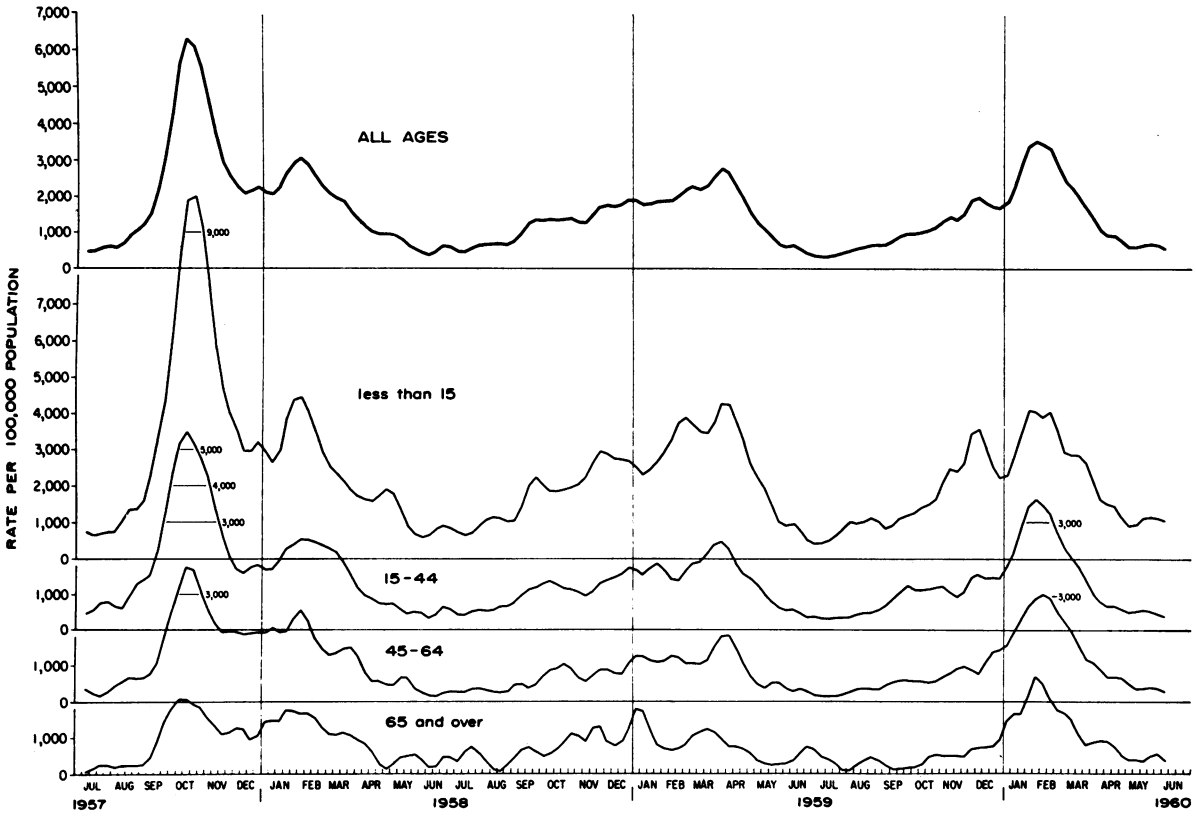
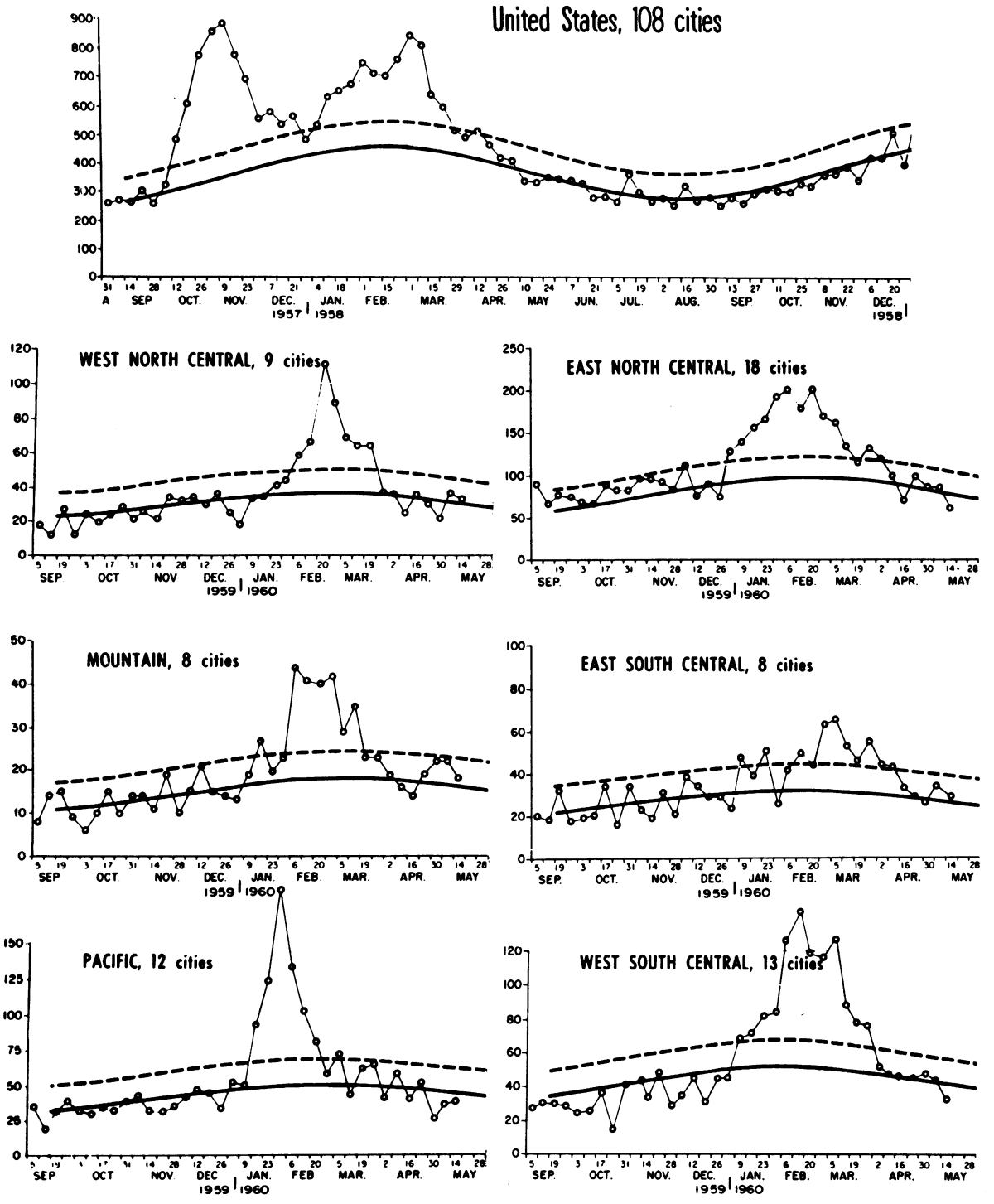
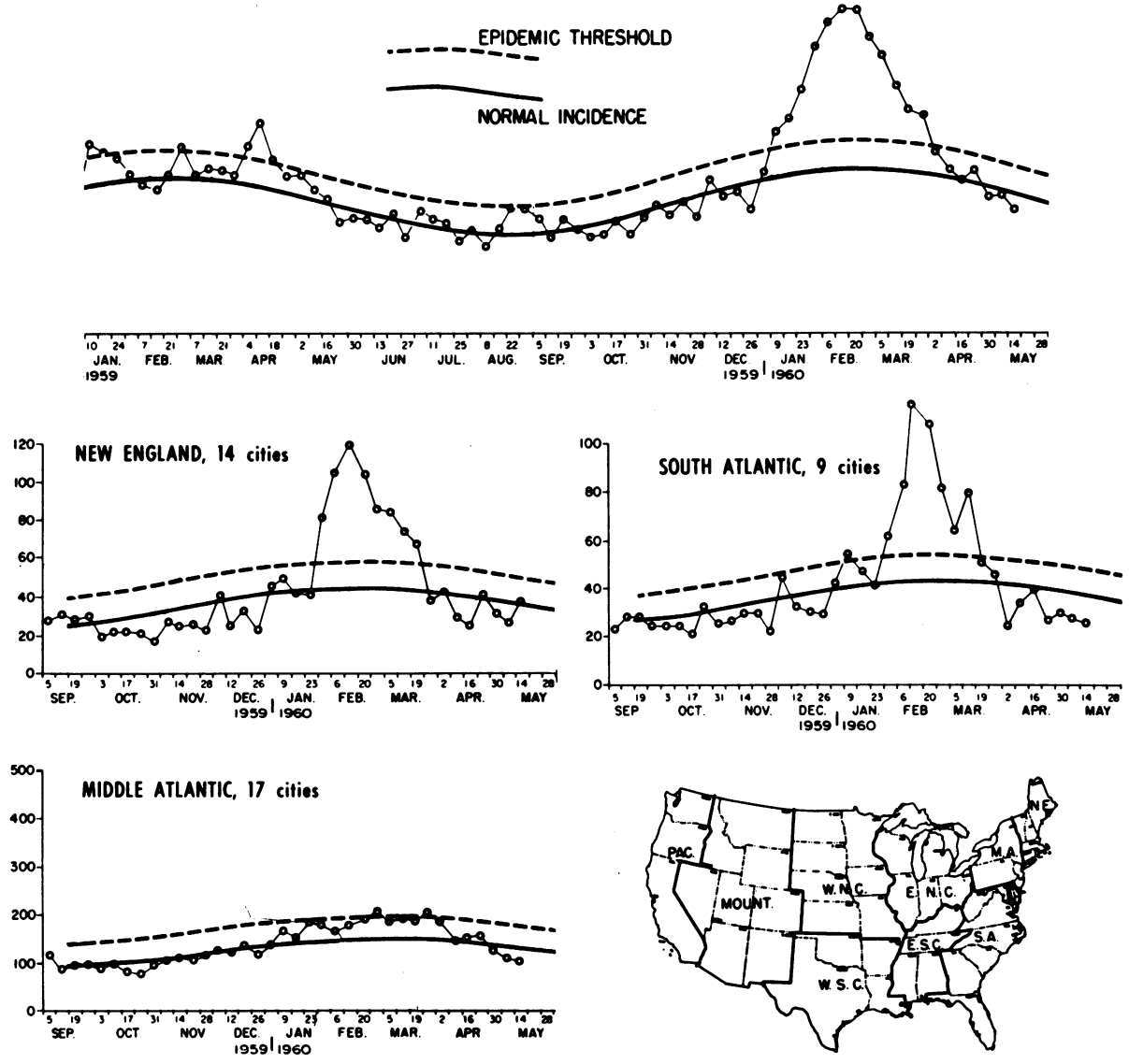


Figure 3. Weekly pneumonia and influenza deaths, United States, 108 cities,



1957 through May 1960. A wave of respiratory illness affecting all age groups is evident during the first 3 months of 1960. The slight increases in respiratory disease morbidity observed in the winter of 1959 occurred during a time when there was only little evidence of influenza activity, and may be considered to approximate the baseline seasonal variation of acute respira-

September 1957–May 1960, and by geographic region, September 1959–May 1960



tory illness of more than mild nature. In particular, the involvement of specific age groups in 1960, compared with their experience in the fall of 1957, shifted toward a relatively greater proportion of overall morbidity in older age groups. Thus, the age group under 15 years had much less morbidity from respiratory disease than in the fall of 1957—in fact, little if any more than in 1959, a "baseline" year. However, the age group 65 years and older had as much if not more respiratory disease morbidity in the winter of 1960 than in the fall of 1957. There is no evidence from these data that one age group was affected by the epidemic before another. The peak of morbidity appears to have been experienced during the last week of January, the week ending January 30, 1960.

Influenza and Pneumonia Excess Mortality

Figure 3 shows influenza and pneumonia deaths reported weekly from 108 cities in the United States for the period September 1957 to May 1960 for the entire country, and September 1959 to May 1960 for each of the nine major geographic regions. The solid curved baseline

in each case represents the expected level of deaths, taking into account seasonal variation and secular trend. The dashed line parallel to the solid baseline represents the "epidemic threshold," allowing for random variation in the weekly numbers of deaths. It is placed so that an elevation of the number of deaths over the epidemic threshold for two or more consecutive weeks would be unlikely to occur except in an epidemic situation. The area between the line of recorded deaths and the solid baseline represents excess mortality due to influenza and pneumonia.

Examination of the data for the United States as a whole reveals a broadly based and severe wave of excess mortality from January to March 1960, reaching a peak during the week ending February 13, 1960. This may be contrasted with the two well-marked waves of excess mortality in the 1957–58 Asian influenza pandemic. As measured by excess influenza and pneumonia mortality, the 1960 epidemic exceeded the second wave of the 1957–58 pandemic in severity, and closely approached that of the first wave. The small amount of excess mortality in late March and April 1959 appears insignificant in comparison with the major epidemic waves in the preceding and succeeding years. This small wave of excess mortality was associated with scattered mixed outbreaks of influenza A_2 and B in the Middle Atlantic region, particularly in the New York metropolitan area.

The individual graphs of weekly excess influenza and pneumonia mortality in the nine geographic regions reveal considerable variation in onset, peak, and regression of the epidemic. The Pacific region, in particular, shows a sharp, well-defined peak of excess mortality. Only the Middle Atlantic region escaped significant excess mortality in the 1960 epidemic.

Characteristics of Virus Strains

Influenza viruses isolated during the 1959–60 season were clearly of the A_2 or Asian subgroup with little, if any, antigenic variation from strains isolated since the emergence of this group of viruses in 1957. It was found in 1957 that, in addition to their unique antigenic composition, these viruses varied markedly with respect to their avidity for specific antibody and nonspecific inhibitor (*2–4*). It was not unusual to isolate from a single geographic area strains which exhibited all degrees of reactivity with specific antibody and nonspecific inhibitor (*5*). While strains lacking avidity for specific antibody and nonspecific inhibitor were frequently isolated in 1957–58, such strains were encountered only rarely in 1959–60. Influenza virus strains submitted to the International Influenza Center were used in the hemagglutination-inhibition test to determine avidity for nonspecific inhibitor as well as specific antibody in human, ferret, and chicken antiserums. Strains were then categorized according to degrees of reactivity. The results of hemagglutination-inhibition tests with a collection of 386 strains submitted to the International Influenza Center from all parts of the world during the period 1957–60 are shown in table 2. The Q-phase influenza virus strains (*6*) which were encountered frequently in 1957–58 were uncommon during outbreaks of influenza in 1959–60.

In addition to cases of influenza due to the Asian strain during 1960, several laboratories reported isolation of strains which could not

Table 2. Reactivity of 386 A_2 influenza virus strains submitted to the International Influenza Center for the Americas, 1957–60

Category	Reactivity	Number of strains			
		1957	1958	1959	1960
I	Inhibitor sensitive, Antibody sensitive	49	114	26	32
II	Inhibitor sensitive, Antibody insensitive (human)	4	5	0	0
III	Inhibitor sensitive, Antibody insensitive (human and ferret or chicken)	1	0	0	0
IV	Inhibitor insensitive, Antibody sensitive	9	17	3	0
V	Inhibitor insensitive, Antibody insensitive (human)	27	45	0	1
VI	Inhibitor insensitive, Antibody insensitive (human and ferret or chicken)	0	7	0	1
Total		90	233	29	34

be typed. Several of these isolates were submitted to the International Influenza Center for identification and were found to be similar or identical to strains which had been prevalent in earlier years (A/PR8/34, A1/Denver/1/57, and B/Lee/40). Studies at this center, together with information obtained from the reporting laboratory, showed that in all cases laboratory contamination was probably responsible for these strains. In cases where paired serums were available for the patient from whom the virus was "isolated," no evidence of influenza virus infection was obtained. Isolation of strains antigenically similar to those prevalent in past years is always of interest since the fate of these strains, when replaced by a new subgroup, remains unknown. Isaacs and Hart have reported recently the isolation of a virus in 1960 which was similar to the A1/England/1/51 strain (personal communication). Isolation was made under conditions which would preclude laboratory contamination; and, in addition, the patient from whom the virus was isolated developed a significant increase in antibody titer to the A1/England/1/51 strain but not to the Asian strain of influenza virus.

Discussion

The surveillance data indicate that the 1960 epidemic differed in certain significant epidemiologic characteristics from the preceding 1957–58 pandemic. At the same time, there was no suggestion that clinical influenzal infection was in any way different from preceding epidemic years or that further antigenic variation occurred within the A_2 subgroup of influenza viruses.

As pointed out by Trotter and associates (*1*), it was possible in many instances to trace the spread of the Asian strain virus through the population during the summer and fall months of 1957. With only the rare exceptions already mentioned, it was not possible to trace geographic spread of the epidemic in 1960. Indeed, the multifocal and almost simultaneous onset of the epidemic in widespread areas of the nation suggested that the virus was already well seeded throughout the population.

The most useful measures of age-specific influenza morbidity rates for the whole nation are the data from the U.S. National Health Survey that first became available in 1957. The age patterns observed then are contrasted with those found in 1960. It must be remembered that the U.S. National Health Survey records acute respiratory diseases involving one or more bed-days of illness. Such data are in no sense specific measures of viral influenza, but during the short period of nationwide epidemics a large proportion of such respiratory illnesses are undoubtedly due to influenzal infection. Thus, a comparison of age-specific respiratory morbidity in 1957–58 with 1960 has some validity.

The overall amount of influenza morbidity in the winter of 1960 was substantially lower than in the fall of 1957; significant differences in age-specific morbidity were present, however, so that the older age groups were affected relatively more heavily. The low incidence of influenza among children under 15 years of age, and the relatively high incidence in the group 65 years and older may explain in part the low frequency of reported school closures and pupil absenteeism, the impressions of disproportionately increased industrial absenteeism, and the unexpectedly high excess mortality. It is possible that the relatively low morbidity rate in children under 15 years of age may have been due in part to a residual high level of immunity in that age group as a result of their extensive influenza experience in 1957–58.

No warning preceded the 13-week wave of excess mortality due to influenza and pneumonia in the winter of 1960, and its severity was unexpected. Although outbreaks of Asian strain influenza had been predicted for that season, it had not been anticipated that the order of magnitude of the epidemic, as measured by excess mortality, would be greater than that of the second wave of the 1957–58 pandemic, and approximately equal to that of the first wave.

Trotter and associates (*1*) had observed that the 1957–58 pandemic was relatively mild, as measured by excess mortality, in both the Mountain and Pacific regions. Both these regions were severely affected during the 1960 epidemic. Conversely, the Middle Atlantic region escaped significant excess mortality in the 1960 epidemic, having been severely affected in the 1957–58 pandemic and again, more mildly, in the spring of 1959.

Influenza and pneumonia mortality excess is again seen to be a sensitive and reliable index of influenza activity. The 2-week lag between the morbidity peak in the week ending January 30, as measured by the U.S. National Health Survey, and the peak of excess mortality in the week ending February 13, 1960, is quite close to the 3-week lag period reported in the pandemic of 1957 by Trotter and associates (*1*), using comparable data.

Although extremely useful as an influenza surveillance technique, excess influenza and pneumonia mortality by no means measures the full force of mortality of an influenza epidemic. A detailed analysis of the nature and extent of overall influenza-associated excess mortality in the epidemics of 1957–58 and 1960 has recently been published (*7*). It is apparent from these observations that excess mortality due to influenza and pneumonia comprises only about one-third of the total influenza-associated excess mortality. The remainder is due primarily to excess deaths among individuals with cardiovascular-renal disease and chronic bronchopulmonary disease. It was estimated (*7*) that as a result of the 1960 epidemic a total of 26,700 excess influenza-associated deaths occurred; only 10,600 of these deaths were attributed directly to influenza or pneumonia.

Summary

An epidemic of influenza of unexpected intensity occurred in the United States during the first 3 months of 1960. Surveillance data were obtained from direct reports from State health authorities, the U.S. National Health Survey, weekly mortality reports, and the WHO International Influenza Center for the Americas.

The origins of the epidemic were multiple, and patterns of geographic spread could rarely be discerned. Older age groups, particularly the age group 65 years and over, had relatively greater influenza morbidity when compared with their experience in the 1957–58 pandemic. The peak of morbidity was reached during the week ending January 30, 1960, followed 2 weeks later by the peak of excess mortality due to influenza and pneumonia. The extent of excess influenza and pneumonia mortality, as reported from 108 cities in the United States, closely approached that recorded during the first wave of the Asian influenza pandemic in the fall of 1957.

The virus strains responsible for the epidemic were clearly of the A_2 (Asian) subgroup, with little, if any, antigenic variation from strains isolated during the pandemic of 1957.

REFERENCES

(*1*) Trotter, Y., Jr., et al.: Asian influenza in the United States, 1957–58. Am. J. Hyg. 70: 34–50 (1959).

(*2*) Zhdanov, V. M., et al.: Antigenic peculiarities of the 1957 influenza viruses and serological indexes of immunity among the population. J.A.M.A. 167: 1469–1474 (1958).

(*3*) Fukumi, H.: Studies on the PQ phases of A/Asia/57 influenza viruses. Bull. World Health Organ. 20: 421–434 (1959).

(*4*) Jensen, K. E., Dunn, F. L., and Robinson, R. Q.: Influenza, 1957. A variant and the pandemic. *In* Progress in medical virology. Hafner Publishing Co., New York, vol. 1, 1958, pp. 165–209.

(*5*) Cleeland, R., and McKee, A. P.: Antigenic variation of Asian influenza virus. Proc. Soc. Exper. Biol & Med. 99: 371–374 (1958).

(*6*) Veen, J. Van der, and Mulder, J.: Studies on the antigenic composition of human influenza virus A strains with the aid of the hemagglutination-inhibition technique. J. Onderz. Meded. Inst. Praev. Geneesk. (Leiden) No. 6, 1950.

(*7*) Eickhoff, T. C., Sherman, I. L., and Serfling, R. E.: Observations on excess mortality associated with epidemic influenza, 1957–60. J.A.M.A. 176: 776–782 (1961).