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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

April 23–May 20, 1939

The accompanying table summarizes the prevalence of eight important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the PUBLIC HEALTH REPORTS under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ending May 20, the number reported for the corresponding period in 1938, and the median number for the years 1934–38.

DISEASES ABOVE MEDIAN PREVALENCE

Poliomyelitis.—For the 4 weeks ended May 20, South Carolina reported 71 cases of poliomyelitis, Georgia 11 cases, and Florida 10 cases, as compared with 3, 3, and 4 cases, respectively, for the corresponding period in 1938. Due largely to the high incidence in these States, the total number of cases (149) reported for the country as a whole during the 4 weeks is 2.3 times the number reported in 1938 and almost twice the preceding 5-year average incidence for this period. An increase in this disease may be expected at this season of the year, but the rise in the South Atlantic region is somewhat sharper than usually occurs.

Influenza.—The influenza incidence continued to decline in all regions of the country, although the number of reported cases (10,725) was almost four times the number reported for the corresponding period in 1938, and almost three times the 1934–38 median figure for this period. In the Middle Atlantic, West North Central, and Pacific regions the incidence was not much above the average seasonal level, but all other regions continued to report an unusually large number of cases for this season of the year. This comparatively high incidence is no doubt due in part to the lateness of the appearance of the disease in anything like epidemic form. In preceding years the peak has

usually been reached in February, but during the current year the incidence remained about normal until in March. While the high incidence at that time was of rather short duration, it has continued long enough to make a rather unfavorable comparison with the corresponding periods in preceding years.

Smallpox.—For the 4 weeks ended May 20 there were 1,229 cases of smallpox reported, as compared with 1,571, 1,142, and 956 for the corresponding period in 1938, 1937, and 1936, respectively. More than one-half of the total number of cases was reported from 4 States, viz, Iowa, 185; Oklahoma, 165; Indiana, 163; and Missouri, 122. The largest number of cases of this disease are still being reported from the Central States. In the Mountain and Pacific States, where the disease has been unusually prevalent for some time, the number of cases has now dropped somewhat below the average seasonal level. The incidence along the Atlantic Coast is about normal.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period April 23–May 20, 1939, the number for the corresponding period in 1938, and the median number of cases reported for the corresponding period in 1934–38 [1]

Division	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median
	Diphtheria			Influenza [2]			Measles [3]			Meningococcus meningitis		
United States [1]	1,221	1,486	1,649	10,725	2,796	3,918	61,913	114,699	114,699	154	233	504
New England	31	49	49	322	21	21	8,550	2,940	7,778	11	7	15
Middle Atlantic	242	301	380	82	52	69	9,231	35,244	22,998	44	44	82
East North Central	273	289	296	617	187	482	5,701	42,412	31,892	20	31	63
West North Central	75	118	123	271	142	258	5,269	7,304	7,304	5	19	35
South Atlantic	177	208	263	3,796	570	926	9,119	13,923	6,126	25	47	116
East South Central	81	124	125	1,753	225	384	1,209	3,394	2,903	14	52	52
West South Central	159	181	245	3,016	1,194	1,194	3,656	2,719	2,719	23	18	25
Mountain	72	97	68	492	150	186	3,956	3,412	3,412	7	4	11
Pacific	111	119	136	376	255	262	14,222	3,451	4,177	5	11	20
	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
United States [1]	149	64	78	15,980	18,074	24,641	1,229	1,571	956	521	645	629
New England	1	1	3	1,094	2,082	1,417	0	0	0	20	15	22
Middle Atlantic	6	5	6	4,606	5,371	6,739	1	0	0	59	71	71
East North Central	8	14	9	6,236	5,223	8,683	354	365	139	71	105	86
West North Central	3	2	3	1,445	2,437	3,064	440	463	463	26	23	38
South Atlantic	95	19	12	573	767	842	5	12	5	89	154	144
East South Central	9	7	5	411	265	265	29	90	7	65	54	54
West South Central	11	8	8	299	426	426	243	145	61	119	124	128
Mountain	2	3	3	454	469	525	59	150	128	30	41	25
Pacific	14	5	17	862	1,034	1,080	98	346	172	42	58	56

[1] 48 States. Nevada is excluded and the Distr ict of Columbia is counted as a State in these reports.
[2] 44 States and New York City.
[3] 47 States. Mississippi is not included.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—For the country as a whole, the current year continues to maintain record breaking lows for diphtheria. The number of cases (1,221) reported for the 4 weeks ended May 20 was about 80

percent of last year's figure and about 75 percent of the average incidence reported during the corresponding period in the years 1934–38. In the Mountain region the incidence stood at about the average seasonal level, but in all other areas it was relatively low.

Measles.—While the incidence of measles has been unusually high in some sections of the country, the number of cases (61,913) reported for the current 4-week period compares very favorably with the average for this period in normal "measles years." As the preceding 5-year period contains three years (1934, 1935, 1938) in which the disease was unusually prevalent, the current incidence is slightly more than 50 percent of the median figure for that period, while the average number of cases for the other 7 years within the past 10 years is approximately 67,000 cases and the current incidence is about 90 percent of that figure. In the Pacific region the number of cases was more than three and one-half times the 1934–38 median figure for this period, while other regions reported minor excesses, ranging from 10 percent in the New England region to almost 50 percent in the South Atlantic.

Meningococcus meningitis.—A decrease from the average seasonal incidence of meningococcus meningitis was apparent in all regions during the current 4-week period. The number of reported cases (154) was about 65 percent of the number reported in 1938, and only about 30 percent of the 1934–38 average figure for the corresponding period. For the country as a whole the current figure is the lowest on record for this period; the nearest approach to this was in 1934, when 220 cases were reported.

Typhoid fever.—The typhoid fever incidence was comparatively low, the number of cases (521) being approximately 80 percent of the number reported for this period in 1938 and also of the 1934–38 median. The East South Central and Mountain groups, however, each show an excess of approximately 20 percent over the average seasonal figure.

Scarlet fever.—The scarlet fever incidence was also relatively low. The number of cases (15,980) reported for the 4 weeks ended May 20 was about 90 percent of last year's figure and only about 65 percent of the 1934–38 average for this period. All regions shared in this favorable comparison except the East South Central, which reported an excess of about 55 percent over the normal seasonal expectancy. For the entire reporting area the current figure is the lowest recorded for this period in 9 years.

MORTALITY, ALL CAUSES

The mortality in large cities reporting to the Bureau of the Census for the 4 weeks ended May 20 averaged 11.3 per 1,000 population (annual basis). The rate for the corresponding period in 1938 was 11.2, and the average rate for the years 1934–38 was 11.9.

ANALYSIS OF 5,116 DEATHS REPORTED AS DUE TO ACUTE CORONARY OCCLUSION IN PHILADELPHIA, 1933–1937[1]

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Over a quarter of a century has passed since Herrick (*1*), in 1912, directed the attention of the medical profession to the clinical diagnosis of acute coronary occlusion. Although this observation provoked little interest at first, probably on account of the exigencies of the World War, since 1920 research workers the world over have spared neither time nor effort to bring new light on the clinical diagnosis and the structural changes underlying and accompanying this now well-recognized clinical entity. While there have been a number of attempts to analyze mortality from diseases of the coronary arteries by statistical methods, no studies have been made of deaths reported as due to acute coronary occlusion in a large city over a period of several years.

The object of this study is to analyze 5,116 deaths reported by the medical profession of Philadelphia during the 5-year period from January 1, 1933, to December 31, 1937, as due to acute coronary occlusion. This will be done with the following points in view: (1) A statement, by way of definition, of the certified causes of death comprising this series; (2) a consideration of the principal sources reporting deaths from this cause; (3) a comparison of the age distribution of reported mortality with deaths from acute coronary occlusion confirmed by necropsy examinations; (4) detailed analyses by sex, color, and age at time of death in the city as a whole and of deaths occurring in hospitals, coroner's cases, and deaths reported from other sources, principally general practitioners; (5) the age, race, and sex distribution of certain cases examined post mortem; (6) the sex distribution and age at death among Jewish persons; (7) determination of death rates from this cause by age, race, and sex; (8) the country of birth of white decedents from this disease; (9) occupation of white males under 65 years of age, based on broad occupational groups; and (10) a discussion of the factors responsible for the increased reported mortality from this important cause of death.

TYPE OF FATAL CASES STUDIED

This study embraces 5,116 deaths occurring in Philadelphia from January 1, 1933, to December 31, 1937, reported to the branch office of vital statistics of the Commonwealth of Pennsylvania as due to acute coronary occlusion or classified under some practically synonymous diagnostic term. According to Levy (*2*), 97 percent of coronary disease is of arteriosclerotic etiology. So far as possible, this study

[1] From the Office of Heart Disease Investigations, National Institute of Health, Branch Office, 133 South 36th St., Philadelphia, Pa.

was limited to deaths which appeared to have been due to a thrombosis occurring as a result of coronary atherosclerosis.

Most of the deaths were certified as being due to acute coronary occlusion or acute coronary thrombosis. Deaths listed as coronary occlusion or thrombosis without qualification concerning acuteness were generally included. Most of these were coroner's cases, or a short duration of the condition was indicated on the death certificate. Other diagnostic terms that were included were cardiac infarction, myocardial infarction, and coronary embolus, a term often incorrectly used which appeared with decreasing frequency.

Diagnoses of other forms of acute coronary disease, such as atresia of the coronary arteries due to syphilitic aortic disease or embolic phenomena complicating rheumatic heart disease or subacute bacterial endocarditis, were not included. Deaths attributed to arteriosclerotic heart disease, coronary sclerosis, myocardial fibrosis, and other conditions involving gradual changes or which are essentially chronic were excluded.

Deaths were not included in which acute coronary occlusion was listed with tuberculosis, cancer, severe injuries, blood dyscrasias, or apparently overwhelming infections. In these conditions it seemed apparent that, even though the diagnoses were correct, acute coronary occlusion had little to do with the fatal outcome. In other instances, such as diabetes mellitus, in which acute coronary occlusion appeared to be an important factor as the immediate cause of death, the reported cases were included in this series.

SOURCES OF MATERIAL

Most mortality studies are made without due regard to the sources from which the death certificates are obtained, and without effort to evaluate the accuracy of the diagnoses. In this study an attempt was made to consider mortality from a qualitative as well as a quantitative point of view.

Deaths from acute coronary occlusion are reported from three main sources (table 1). Of the 5,116 deaths in this series, 703, or 13.7 percent, occurred among patients regularly admitted to 26 civilian hospitals approved for interneship by the American Medical Association. The diagnoses on the death certificates were made on the basis of necropsy findings in 197, or 28.1 percent, of these 703 cases. The hospital cases as a whole were subject to above average diagnostic standards. Many of the fatal cases had been admitted previously for cardiac conditions, not infrequently an acute coronary occlusion. Other cases had been studied intensively in cardiac clinics. Improvement in diagnosis was offset to a certain extent by an element of selection.

Comparatively few very old persons seek hospital care, a point which will be discussed later in detail.

TABLE 1.—*Number of deaths attributed to acute coronary occlusion in Philadelphia each year from Jan. 1, 1933, to Dec. 31, 1937, among patients regularly admitted to 26 civilian hospitals approved for interneship by the American Medical Association, coroner's cases, deaths reported from all other sources, and the total number of deaths attributed to this cause*

Year	Deaths in civilian hospitals approved for interneship by the American Medical Association	Coroner's cases	Deaths in other hospitals and in homes	Total
1933	114	258	300	672
1934	96	299	408	803
1935	159	383	454	996
1936	140	403	581	1,124
1937	194	525	802	1,521
Total	703	1,868	2,545	5,116
Percent of total	13.7	36.5	49.8	100.0
Percent increase in 5 years	70	103	167	126

The coroner's office reported 1,868 deaths, or 36.5 percent of the total. Information concerning cases examined post mortem was not obtained, but it was understood that necropsies were obtained on about 20 percent of these cases. This group is composed of sudden and unexplained deaths, or of deaths unattended by a physician. Patients dying within 24 hours after admission to hospitals fall in this group. Of the 1,868 coroner's cases, 863 deaths occurred in or en route to hospitals. Since nearly all of these deaths occurred within 24 hours after admission to a hospital and since many patients were either dead or moribund on arrival, they were not included in the hospital series. Obviously the hospitals did not have sufficient time to study these cases as fully as the regularly admitted cases.

The chief source of selection in this group is the probability that relatively sudden deaths among aged persons in more or less failing health were regarded by the attending physician as due to natural causes, and either were not brought to the attention of the coroner's office or else not considered of sufficient importance to require an inquest. The same type of death among middle-aged persons, especially when occurring away from home, is almost certain to be investigated by the coroner.

The remainder of the deaths, 2,545, or 49.8 percent of all deaths in this series, occurred for the most part in homes and were reported by general practitioners. Included in this group are a few deaths occurring in hospitals not approved for interneship by the American Medical Association, special hospitals, homes for the aged, and other institutions. Such a small number were examined post mortem that it was not considered worth while to tabulate the necropsy cases.

ACCURACY OF DIAGNOSES

Although it is not possible for the health officer or statistician reviewing a large series of deaths to determine the accuracy of diagnosis in each individual case, effort should be made to compare the series with the results of studies from sources believed to be reliable. Care must be exercised to rule out elements of selection which may influence the series under study or the cases used for comparison.

In figure 1, the age distribution of the 5,116 fatal cases of acute coronary occlusion reported by physicians in Philadelphia is compared with that in two series of fatal cases of acute coronary occlusion based on necropsy findings. One of these consists of 284 fatal cases collected from the literature, consisting of the combined studies of Levine (*3*), Saphir et al. (*4*), Appelbaum and Nicolson (*5*), and Meakins and Eakin (*6*). The other series consists of 197 necropsied cases

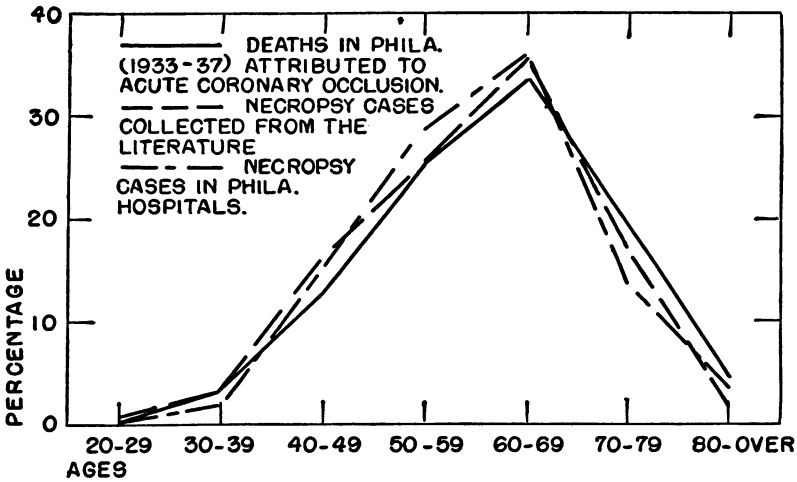

FIGURE 1.—Percentage age distribution, by decades of life, of 5,116 deaths reported as due to acute coronary occlusion in Philadelphia from January 1, 1933, to December 31, 1937, compared with 284 necropsied cases collected from the literature and 197 fatal cases in this series in which the diagnoses were based on necropsy findings.

occurring among patients regularly admitted to Philadelphia hospitals during the period under study.

A comparison of the age distribution of the fatal cases reported from all sources with the two necropsy series reveals very little difference. The mean age at death in the 5,116 cases was 61.2 years. Among the 197 cases in this series examined post mortem the mean age was 59.8 years, while among Levine's 46 necropsied cases the average age at death was 58.5 years. This slight difference is due to the large number of nonhospital cases.

The age distribution and mean ages at death during each of the 5 years are shown in figure 2. Despite the great increase in mortality

from this cause during this period (see table 1) the distribution of deaths by age decades remained practically the same and the mean ages varied only 1.8 years. The distribution of deaths during each of these years conformed closely to that in series based on well-authenticated cases and to the general picture of the age distribution described in the literature.

There is a tendency among some physicians to overexploit popular diagnoses. While there were doubtless many incorrect diagnoses in this large series, it does not appear likely that the diagnosis of acute coronary occlusion is being abused to any great extent in Philadelphia. Although more deaths were diagnosed each year as due to this cause, the age distribution was remarkably constant during the period under study and conformed quite closely (compare figs. 1 and 2) to the two necropsy series.

The increased incidence of deaths attributed to this cause was not due to an increase in the use of this term in describing deaths among

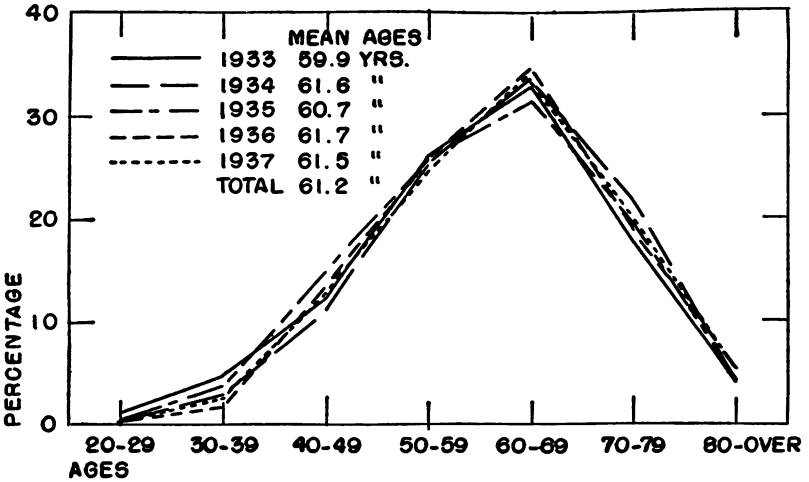

FIGURE 2.—Percentage age distribution, by decades of life, and mean ages at death of 5,116 deaths reported as due to acute coronary occlusion in Philadelphia based on the number of deaths occurring in each of the 5 years under study.

elderly persons. Of the total number of deaths from acute coronary occlusion, only 24.5 percent occurred among persons past 70 years of age, while among deaths from all forms of heart disease, 42.1 percent of the recorded mortality was among persons past 70 years of age.

Despite efforts on the part of the writer to eliminate cases of sudden death due to cardiovascular syphilis by rejecting deaths certified as due to that cause, it is likely that some such deaths were included. It is doubtful that unrecognized or undiagnosed syphilis is as important a factor as might be anticipated, except, perhaps, among cases of sudden death. The differential diagnosis

between cardiovascular syphilis and various forms of coronary disease is usually not difficult. Syphilis of the aorta and heart does not appear to be a very frequent cause of heart disease in Philadelphia, even among clinic patients, except among colored adults, especially Negro males. Since less than 5 percent of these 5,116 deaths attributed to acute coronary occlusion were among Negroes, it does not appear likely that cardiovascular syphilis constituted an important source of error.

The inclusion of a certain number of cases of cardiovascular syphilis is an inherent disadvantage encountered in mortality studies of heart disease among adults. It will probably be much easier to prevent cardiovascular syphilis than to obtain necropsies wherever this factor seems likely and to induce physicians to make diagnoses of syphilitic affections without regard to the feelings of the family of the deceased.

Further references to the accuracy of diagnoses will be made in analyzing in detail the various sources from which death certificates were received. In addition to deaths occurring in the city as a whole, in hospitals, among coroner's cases, and in homes, studies will be made of necropsied cases from hospitals and deaths from acute coronary occlusion among Jewish persons, a group commonly believed to be especially susceptible to this disease.

DEATHS IN THE CITY AS A WHOLE

In table 2 is shown the age distribution, by decades, according to color and sex of the 5,116 deaths attributed to acute coronary occlu-

TABLE 2.—*Number and percentage of deaths in each age decade and mean age at death according to color and sex among 5,116 deaths attributed to acute coronary occlusion in Philadelphia (from all sources) from Jan. 1, 1933, to Dec. 31, 1937*

Age (years)	Total						White						Colored					
	Both sexes		Male		Female		Both sexes		Male		Female		Both sexes		Male		Female	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
20 to 29	21	0.4	10	0.3	11	0.6	15	0.3	8	0.3	7	0.4	6	2.6	2	1.6	4	3.7
30 to 39	160	3.1	107	3.2	53	3.0	132	2.7	91	2.8	41	2.5	28	12.1	16	12.9	12	11.2
40 to 49	663	13.0	521	15.4	142	8.2	605	12.4	487	15.0	118	7.3	58	25.1	34	27.5	24	22.4
50 to 59	1,299	25.4	952	28.2	347	20.0	1,222	25.0	910	28.0	312	19.1	77	33.3	42	33.8	35	32.7
60 to 69	1,720	33.6	1,111	32.9	609	35.0	1,675	34.3	1,092	33.6	583	35.7	45	19.6	19	15.3	26	24.4
70 to 79	1,003	19.6	566	16.8	437	25.1	990	20.3	556	17.0	434	26.6	13	5.6	10	8.1	3	2.8
80 and over	248	4.9	108	3.2	140	8.1	244	5.0	107	3.3	137	8.4	4	1.7	1	.8	3	2.8
Unknown	2	----	2	----	0	----	2	----	2	----	0	----	0	----	0	----	0	----
Total	5,116	----	3,377	----	1,739	----	4,885	----	3,253	----	1,632	----	231	----	124	----	107	----
Percent of total	----	100	----	66.0	----	34.0	----	95.5	----	63.6	----	31.9	----	4.5	----	2.4	----	2.1
Mean age at death	61.2	----	59.8	----	63.9	----	61.6	----	60.0	----	64.7	----	52.3	----	52.0	----	52.7	----

sion in Philadelphia during the quinquennium under study. The mean age at death was 61.2 years. The maximum number of deaths (33.6 percent) occurred in the 60–69-year age period. In this series 16.5 percent occurred prior to 50 years of age, while 41.9 percent occurred before 60 years of age. Only 24.5 percent occurred in persons over 70 years of age (fig. 3).

Among males, 18.9 percent of deaths occurred prior to 50 years of age, while among females only 11.8 percent occurred prior to that age. In both sexes the peak incidence occurred in the 60–69-year age period. Among females 33.2 percent occurred in persons over 70 years of age, while among males only 20 percent occurred during this advanced age period (fig. 4). The mean age at death among males was

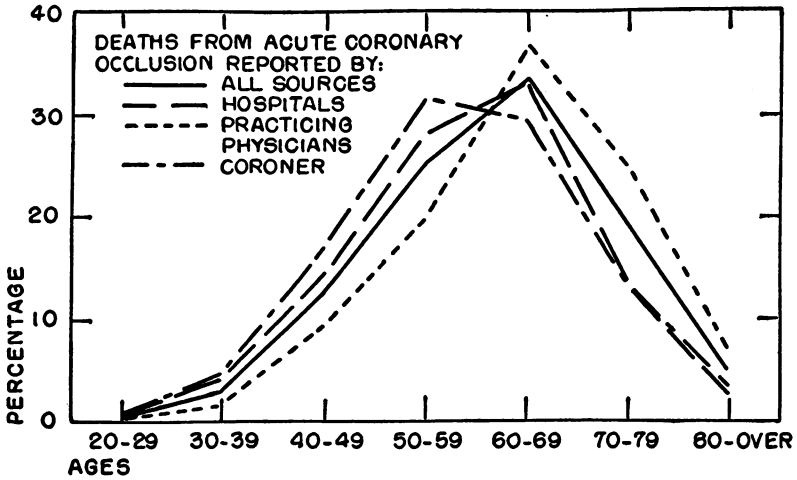

FIGURE 3.—Percentage age distribution by decades of life of 5,116 deaths reported as due to acute coronary occlusion, 703 deaths from 26 civilian hospitals approved for interneship by the American Medical Association, 1,868 coroner's cases, and 2,545 deaths from other sources, most of which occurred in the homes, in Philadelphia from January 1, 1933, to December 31, 1937

59.8 years, while among females it was 63.9 years, a difference of over 4 years. The ratio of males to females was nearly 2 to 1. This is not as great a difference as has been reported in some clinical series, probably owing to the inclusion of more cases over 70 years of age.

The unusual ratios between males and females under 30 years of age may be due to the few cases in this age group or to the inclusion of some cases of acute cor pulmonale due to pulmonary emboli. During the first 2 years under study there was an unusually large number of deaths reported among relatively young women after operation or during the postpartem period as due to acute coronary occlusion. With better differentiation between acute coronary occlusion and acute pulmonary embolism the number of deaths attributed to the former condition declined. It is believed that with better

diagnostic standards the number of deaths among males would greatly exceed those among females.

The mean age at death among white persons was 61.6 years. Among white males it was 60.0 years, while among white females it was 64.7 years. Since 95.5 percent of the total number of deaths from this cause occurred among white persons, the age distribution and ratio of male to female mortality is practically the same as that described in the above paragraph.

Only 4.5 percent of the total mortality occurred among Negroes. This is strikingly low in a city where, according to the United States census of 1930, 11.3 percent of the population are Negroes, even after allowance is made for a somewhat younger age distribution and the

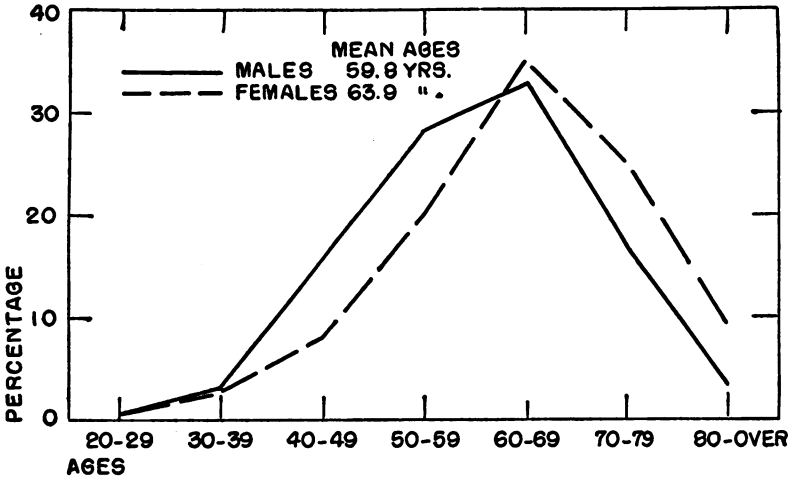

FIGURE 4.—Percentage age distribution, by decades of life, and mean age at death of deaths attributed to acute coronary occlusion among males as compared with females in Philadelphia from January 1, 1933, to December 31, 1937.

likelihood that many colored patients are attended by physicians less proficient in the diagnosis of acute coronary occlusion.

The mean age at death among Negroes was 52.3 years. Among colored males the mean age at death was 52.0 years, while among females it was 52.7 years. Despite the probable inclusion of some cases of cardiovascular syphilis, it is believed that these figures are essentially correct. Johnston (*7*) observed that acute coronary occlusion occurred at a somewhat earlier age among Negroes than among white persons in a series of hospital cases. Since mortality from rheumatic, syphilitic, and hypertensive cardiovascular diseases (*8*) (*9*) occur several years earlier among Negroes it is not surprising to find that a similar situation applies to coronary disease.

The age distribution of mortality from acute coronary occlusion among Negroes as compared with white persons is shown in figure 5.

The peak number of deaths among Negroes occurs in the 50–59-year age period as compared with the 60–69-year age period among white persons. The ratio of colored males to females (table 2) was nearly even.

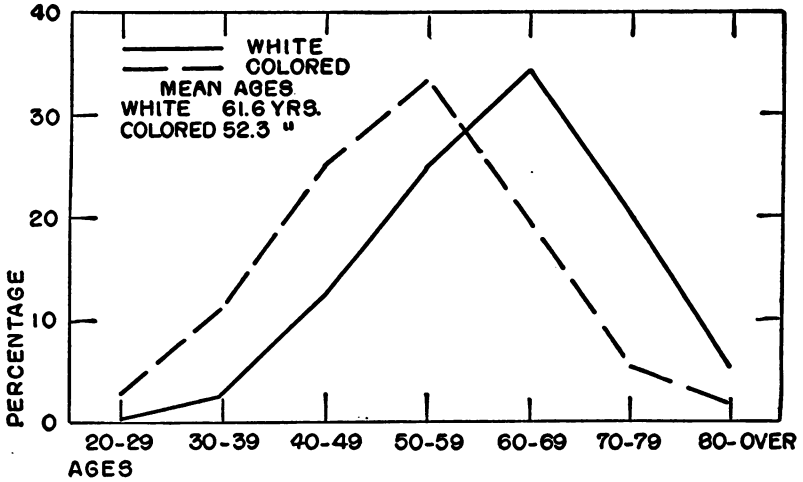

FIGURE 5.—Percentage age distribution, by decades of life, mean age distribution by decades of life, and mean age at death of deaths reported as due to acute coronary occlusion among white persons as compared with colored persons in Philadelphia from January 1, 1933, to December 31, 1937.

DEATHS OCCURRING IN HOSPITALS APPROVED FOR INTERNESHIP BY THE AMERICAN MEDICAL ASSOCIATION

There were 703 deaths reported among patients regularly admitted to 26 civilian hospitals approved for interneship by the American Medical Association. These do not include 836 deaths occurring en route to these hospitals or within 24 hours after admission, for which the causes of death were determined by the coroner's office.

The mean age at death was 59.5 years. The age distribution (table 3 and fig. 3) was essentially similar to that for the entire series. Deaths from heart disease in hospitals tend to occur at somewhat earlier age periods than in private practice for the following reasons:

(1) Elderly persons, especially among the foreign born and Negro populations, have never been educated to the value of hospitals.

(2) Many elderly persons in the face of a severe illness prefer to remain at home with their relatives rather than "go to the hospital to die."

(3) Many patients with heart disease are admitted to the hospital wards from the outpatient clinics. Owing to the infirmities of old age, there is a decline in the number of persons past 70 years of age who are physically able to attend outpatient clinics.

(4) Elderly patients often reside in various types of institutions. Most of these institutions have attending physicians and many of them have infirmaries. Consequently, there is no reason for transferring these patients to hospitals for nonsurgical conditions.

(5) Old age pensions and other benefit funds often enable them to live at home and be attended by the family physician.

TABLE 3.—*Number and percentage of deaths in each age decade and mean age at death according to color and sex among 703 deaths attributed to acute coronary occlusion among regularly admitted patients (exclusive of coroner's cases) to 26 civilian hospitals approved for interneship by the American Medical Association, in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937*

Age (years)	Total						White						Colored		
	Both sexes		Male		Female		Both sexes		Male		Female		Both sexes	Male	Female
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Number	Number
20 to 29	3	0.4	1	0.2	2	0.8	3	0.5	1	0.2	2	0.8	0	0	0
30 to 39	30	4.3	16	3.7	14	5.3	22	3.3	11	2.7	11	4.4	8	5	3
40 to 49	102	14.5	74	17.0	28	10.5	89	13.4	66	16.1	23	9.2	13	8	5
50 to 59	200	28.5	128	29.4	72	27.1	186	28.1	120	29.2	66	26.3	14	8	6
60 to 69	233	33.2	139	31.8	94	35.3	230	34.8	137	33.3	93	37.0	3	2	1
70 to 79	113	16.1	64	14.7	49	18.4	111	16.7	62	15.1	49	19.5	2	2	0
80 and over	21	3.0	14	3.2	7	2.6	21	3.2	14	3.4	7	2.8	0	0	0
Total	[1] 702	-----	436	-----	266	-----	662	-----	411	-----	251	-----	40	25	15
Percent of total	100.0	-----	62.1	-----	37.9	-----	94.3	-----	58.5	-----	35.8	-----	5.7	3.6	2.1
Mean age at death	59.5	-----	59.1	-----	60.1	-----	60.1	-----	59.7	-----	60.9	-----	49.0	49.7	47.8

[1] Excluding 1 case of unknown age.

(6) Another factor is the economic one. Many of these people have saved very little money and their children cannot afford to hospitalize them for an indefinite period, especially when they feel that the situation is essentially hopeless.

(7) Death from heart disease among the aged often supervenes after periods of chronic invalidism. Hospitals do not have facilities for prolonged care of patients with chronic conditions.

With the exception of the cases examined post mortem, diagnosis of deaths occurring in hospitals is the most accurate in this series of cases. The medical staffs of the large general hospitals are more experienced in diagnosing heart disease, and often have had the opportunity of following the patients in the outpatient clinics or have seen them on the wards in previous attacks of acute coronary occlusion. They also have electrocardiographs and other diagnostic equipment at their disposal.

After allowance has been made for the factors mentioned above, the age distribution of this group does not differ materially from that of deaths from acute coronary occlusion in the city as a whole. More deaths occurred under 60 years of age (compare tables 2 and 3), while fewer occurred among persons over 70 years of age. The ratio of males to females was 1.7 to 1, much lower than the experiences of other writers indicate. An explanation for this unusually low ratio of male to female deaths is that this group consists entirely of fully admitted hospital cases, while series reported by other writers include sudden deaths or deaths occurring within 24 hours after admission. As will be shown subsequently, coroner's cases were predominantly of the male sex.

Only 5.7 percent were Negroes. When consideration is given to the fact that, owing to lower economic status, Negroes are more likely than white persons to seek admission to hospitals, this becomes even more significant. At the Philadelphia General Hospital, for example, Negroes comprise about 45 percent of the total number of admissions for all causes. The mean age at death among Negroes was 49.0 years. This is even lower than the mean age at death among Negroes for deaths reported as due to acute coronary occlusion from the entire city. (See table 2.)

NECROPSY CASES OF ACUTE CORONARY OCCLUSION IN HOSPITALS

Of the 703 fatal cases of acute coronary occlusion reported as occurring in hospitals approved for interneship by the American Medical Association, diagnoses on death certificates were made on the basis of necropsy examinations in 197, or 28.1 percent of cases (table 4). The mean age at death among these cases was 59.8 years, practically the same as among the entire group of hospital cases (table 3), and not differing greatly from that in the city as a whole (table 2). The age distribution was essentially similar to that of the hospital group as a whole, allowance being made for the smaller series of necropsied cases, with a greater probability of error. Of the 197 deaths, 17.3 percent occurred among persons less than 50 years of age and 46.7 percent under 60 years; the maximum number of deaths, 36 percent, occurred in the 60–69-year age period, while only 17.3 percent occurred among persons over 70 years of age.

TABLE 4.—*Number and percentage of deaths in each age decade and mean age at death according to color and sex among 197 deaths from acute coronary occlusion in Philadelphia hospitals from Jan. 1, 1933, to Dec. 31, 1937, in which the diagnoses on the death certificates were confirmed by post-mortem examinations*

Age (years)	Total						White						Colored		
	Both sexes		Male		Female		Both sexes		Male		Female		Both sexes	Male	Female
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Number	Number
20 to 29	0	-----	0	-----	0	-----	0	-----	0	-----	0	-----	0	0	0
30 to 39	4	2.0	3	2.4	1	1.4	3	1.6	2	1.7	1	1.5	1	1	0
40 to 49	30	15.3	23	18.3	7	9.8	25	13.5	20	16.7	5	7.6	5	3	2
50 to 59	58	29.4	38	30.1	20	28.2	54	29.0	36	30.0	18	27.3	4	2	2
60 to 69	71	36.0	44	34.9	27	38.0	70	37.6	44	36.6	26	39.4	1	0	1
70 to 79	27	13.7	14	11.1	13	18.4	27	14.5	14	11.7	13	19.7	0	0	0
80 and over	7	3.6	4	3.2	3	4.2	7	3.8	4	3.3	3	4.5	0	0	0
Total	197	-----	126	-----	71	-----	186	-----	120	-----	66	-----	11	6	5
Percent of total	100	-----	64.0	-----	36.0	-----	94.4	-----	60.9	-----	33.5	-----	5.6	3.0	2.5
Mean age at death	59.8	-----	59.3	-----	62.0	-----	60.5	-----	59.3	-----	62.7	-----	48.2	45.0	52.2

The ratio of males to females was only 1.8 to 1, much less than the reported ratio of other series. The mean age at death among white persons was 60.5 years. Among white females the mean age at death was 62.7 years, while among white males it was 59.3 years, more than 3 years less. Although there were only 11 Negroes whose death certificates indicated that a necropsy had been performed, the mean age, 48.2 years, bears out the impression that Negroes succumb to this disease several years younger than white persons.

Only 5.6 percent of the necropsied cases were Negroes. When consideration is given to the facts that Negroes account for 11.3 percent of the population of Philadelphia, that, owing to their poor economic circumstances, they constitute a much larger hospital problem than this percentage indicates, and that it is not difficult as a rule to obtain permission for necropsies, it appears evident that acute coronary occlusion occurs much less frequently among colored persons.

CORONER'S CASES

In table 5 is shown the age distribution, by decades, according to color and sex, of 1,868 deaths attributed to acute coronary occlusion by coroner's physicians. This group is of interest because it comprises 36.5 percent of the total deaths from this cause, and because it is composed of the type of deaths described in the daily press as due to "heart attacks." Since it is not possible to hold post-mortem examinations on every case brought to the attention of the coroner, there is perhaps a greater likelihood of error in diagnosis in these cases than in deaths reported in hospitals or by general practitioners. On the other hand, as has been previously indicated, there is less opportunity for selection.

TABLE 5.—*Number and percentage of deaths in each age decade and mean age at death according to color and sex among 1,868 coroner's cases attributed to acute coronary occlusion in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937*

Age (years)	Total						White						Colored					
	Both sexes		Male		Female		Both sexes		Male		Female		Both sexes		Male		Female	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
20 to 29	14	0.7	7	0.5	7	1.6	10	0.6	6	0.4	4	1.1	4	3.1	1	1.3	3	5.8
30 to 39	89	4.8	71	4.9	18	4.2	76	4.4	61	4.5	15	3.9	13	10.0	10	12.8	3	5.8
40 to 49	319	17.1	275	19.2	44	10.2	283	16.3	252	18.6	31	8.2	36	27.7	23	29.5	13	25.0
50 to 59	590	31.6	469	32.6	121	28.0	539	31.0	439	32.3	100	26.3	51	39.2	30	38.5	21	40.4
60 to 69	552	29.5	410	28.6	142	32.9	531	30.6	400	29.5	131	34.5	21	16.2	10	12.8	11	21.1
70 to 79	251	13.4	174	12.1	77	17.8	247	14.2	171	12.6	76	20.0	4	3.0	3	3.8	1	1.9
80 and over	53	2.9	80	2.1	23	5.3	52	2.9	29	2.1	23	6.1	1	.8	1	1.3	0	----
Total	1,868	----	1,436	----	432	----	1,738	----	1,358	----	380	----	130	----	78	----	52	----
Percent of total	----	100	----	76.9	----	23.1	----	93.0	----	72.7	----	20.3	----	7.0	----	4.2	----	2.8
Mean age at death	58.1	----	57.4	----	60.6	----	58.6	----	57.7	----	61.8	----	51.3	----	51.0	----	51.6	----

This group should be regarded as consisting for the most part of cases in which death was caused by advanced coronary arteriosclerosis, many probably suffering acute occlusions of coronary arteries just prior to death. Acute myocardial ischemia due to advanced coronary disease or other similar mechanisms were also probable immediate factors in some instances. There were probably some cases of cardiac rupture with hemopericardium. This is usually the result of a former coronary occlusion with myocardial infarction. There were quite likely a certain number of cases of dissecting aortic aneurysm, usually on an arteriosclerotic basis. Apoplexy, pulmonary emboli, sudden heart failure in essential hypertension, Stokes-Adams attacks, and unexplained cessation of cardiac activity probably were involved to a certain extent. Cardiovascular syphilis cannot be ruled out without post-mortem examinations. Martland (*10*), for example, found that 18 percent of his 300 cases of sudden death examined post mortem died from syphilis of the aorta. After all of these factors have been considered, advanced coronary arteriosclerosis in all likelihood remains the outstanding factor.

The mean age at death was 58.1 years. The age distribution indicates death at somewhat younger ages than for any other group. (See fig. 3.) Of the total number of deaths attributed to acute coronary occlusion by the coroner's office, 22.6 percent occurred among persons under 50 years of age (table 5). The peak incidence, 31.6 percent, occurred in the 50–59-year age period, a decade younger than in the cases for the city as a whole. Altogether, 54.2 percent of these deaths, most of which were sudden, occurred among persons less than 60 years of age. Only 16.3 percent occurred among persons past 70 years of age.

The mean age at death among white males was 57.7 years, while among white females it was 61.8 years. Among white persons of both sexes the mean age at death was 58.6 years. The ratio of males to females for deaths occurring among white persons was 3.6 to 1. The age distribution indicated death at considerably younger ages among white males than white females. The maximum number of deaths among white males occurred in the 50–59-year age decade, while among females the peak incidence occurred in the 60–69-year age period. The ratio of males to females was over 4 to 1 in the 30–39-year age period, over 8 to 1 in the 40–49-year age decade, over 4 to 1 in the 50–59-year age decade, while in the 60–69-year age period the ratio of males to females was over 3 to 1.

The increased liability of white males as compared with white females to sudden death constitutes an important clinical and public health problem which for the most part is being neglected. It should be made a subject of carefully planned research extending over a num-

ber of years. These studies should take into consideration not only etiology and structural changes as determined by post-mortem examinations, but also the occupation, habits, previous illnesses, and hereditary background of persons dying suddenly from natural causes. Incidentally, a study of this sort would probably serve as a very effective index in years to come of the success of the campaign recently inaugurated against syphilis.

Only 130, or 7.0 percent, of the coroner's cases were Negroes. The mean age at death among Negro males was 51.0 years, while among females it was 51.6 years; among Negroes of both sexes it was 51.3 years. There was less difference in the age distribution of deaths between the sexes of this race than among white persons. Notwithstanding this, 43.6 percent of the deaths of Negro males occurred among persons under 50 years of age, while only 36.6 percent of deaths of Negro females occurred during this period. In both sexes the greatest number of deaths occurred during the 50–59-year age period. The ratio of Negro males to females was 1.5 to 1. Although there were more deaths among males, there was not the marked difference noted among white males as compared with white females.

DEATHS IN OTHER HOSPITALS AND IN HOMES

There were 2,545 deaths reported as occurring in other hospitals or at home. Of this number, 2,438 occurred in homes or in non-medical institutions such as homes for the aged, while 106 deaths occurred in a miscellaneous group of hospitals. The only large hospital in this group is the United States Naval Hospital. This hospital has clinical standards equal to those of large civilian hospitals, and is approved for interneship by the American Medical Association. The Naval Hospital was included in this group because its beneficiaries constitute a selected class from the standpoint of sex and age distribution. Less than 40 deaths were reported from this hospital.

The remaining hospitals were either small general or private hospitals, or institutions devoted to the treatment of special diseases. These hospitals are not recognized for interneship by the American Medical Association. Since this miscellaneous group of hospitals, including the Naval Hospital, reported only 106 deaths, or 4.2 percent of the 2,544 deaths not occurring in the other hospitals or reported by the coroner's office, deaths from these sources are included with the deaths occurring in homes. It is quite likely that the standards of diagnosis of coronary disease in some of these hospitals are more nearly comparable to diagnoses made in homes than to diagnoses made in large hospitals.

The mean age at death was 63.9 years, about 4.4 years older than deaths occurring in the 26 civilian hospitals approved for interneship

by the American Medical Association. Despite a greater opportunity for error in diagnosis among these cases, an older age at death is to be expected. In 1936 the mean age at death from rheumatic heart disease in Philadelphia was 36.5 years. Among deaths from rheumatic heart disease occurring in hospitals approved for internship the mean age was 33.4 years, while among deaths occurring elsewhere the mean age was 39.7 years.

The deaths from acute coronary occlusion reported in homes occurred among persons several years older than those in the other groups (fig. 3). Only a little over 10 percent occurred under 50 years of age. Only 31.3 percent occurred prior to 60 years of age (table 6). The greatest percentage of cases, 36.8, occurred in the 60–69-year age period. Although 31.9 percent of these deaths were reported among persons over 70 years of age, this does not appear to be an excessive percentage, since the mortality from this disease is limited to persons in adult life and reaches its maximum in the 60–69-year age decade. This does not suggest that physicians in Philadelphia have seized upon acute coronary occlusion as a convenient blanket diagnosis for deaths among persons over 50 years of age, as was the case, in some instances, with myocarditis in the past. Apparently the diagnosis of acute coronary occlusion is being made with considerable restraint and with an effort to portray a definite clinical condition.

TABLE 6.—*Number and percentage of deaths in each age decade and mean age at death according to color and sex among 2,545 deaths attributed to acute coronary occlusion and occurring in hospitals not approved for interneship by the American Medical Association, in other institutions, and in homes in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937*

Age (years)	Total						White						Colored					
	Both sexes		Male		Female		Both sexes		Male		Female		Both sexes		Male		Female	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
20 to 29	4	0.2	2	0.1	2	0.2	2	0.1	1	0.1	1	0.1	2	3.3	1	4.8	1	2.5
30 to 39	41	1.6	20	1.3	21	2.0	34	1.4	19	1.3	15	1.5	7	11.5	1	4.8	6	15.0
40 to 49	242	9.5	172	11.4	70	6.6	233	9.3	169	11.4	64	6.4	9	14.8	3	14.3	6	15.0
50 to 59	509	20.0	355	23.7	154	14.8	497	20.0	351	23.7	146	14.6	12	19.6	4	19.0	8	20.0
60 to 69	935	36.8	562	37.4	373	35.8	914	36.8	555	37.5	359	35.9	21	34.4	7	33.3	14	35.0
70 to 79	639	25.1	328	21.8	311	30.0	632	25.5	323	21.7	309	30.8	7	11.5	5	23.8	2	5.0
80 and over	174	6.8	64	4.3	110	10.6	171	6.9	64	4.3	107	10.7	3	4.9	0	0	3	7.5
Total	[1] 2,544	----	1,503	----	1,041	----	2,483	----	1,482	----	1,001	----	61	----	21	----	40	----
Percent of total	----	100	----	59.1	----	40.9	----	97.6	----	58.3	----	39.3	----	2.4	----	.8	----	1.6
Mean age at death	63.9	----	62.4	----	66.1	----	64.1	----	62.5	----	66.5	----	57.0	----	58.8	----	56.0	----

[1] Excluding 1 case of unknown age.

The distribution of these deaths by age, race, and sex was essentially similar to the distribution in hospitals approved for interneship, except that age at time of death was greater in the former group. The ratio of males to females was approximately 1.5 to 1, due largely to the number of deaths among females over 70 years of age. Only 2.4 percent of these deaths occurred among Negroes. More deaths were reported among Negro females than among Negro males. The mean age at death was over 7 years younger among colored than among white persons.

DEATHS AMONG JEWISH PERSONS

There is a rather widespread impression that Jews are especially liable to acute coronary occlusion. An effort was made to determine the number of deaths among Jewish persons by a careful scrutiny of the death certificates for the place of burial, name of decedent, given and surnames of decedents' parents, and other factors which might indicate that the deceased person was of the Jewish race. Only an approximate figure can be obtained in this way. Owing to the tendency on the part of native-born Hebrews, in particular, to Anglicize their names and the fact that they are often not buried in Jewish cemeteries, this group is composed largely of foreign born, for the most part Russian-Jewish persons.

TABLE 7.—*Number and percentage of deaths in each age decade and mean age at death according to sex among 909 deaths attributed to acute coronary occlusion among Jewish persons in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937, with a comparison of the age distribution of deaths attributed to this cause among non-Jewish white persons during this period*

Age (years)	Deaths among Jewish persons						Deaths among non-Jewish white persons	
	Both sexes		Male		Female			
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
20 to 29	6	0.7	3	0.5	3	1.0	9	0.2
30 to 39	17	1.9	12	2.0	5	1.7	115	2.9
40 to 49	119	13.1	101	16.6	18	6.0	486	12.2
50 to 59	272	29.9	194	32.0	78	25.9	950	23.9
60 to 69	345	38.0	215	35.4	130	43.2	1,330	33.5
70 to 79	121	13.3	68	11.2	53	17.6	869	21.9
80 and over	28	3.1	14	2.3	14	4.6	216	5.4
Unknown	1		1		0		1	
Total	909		608		301		3,976	
Percent of total			66.9		33.1			
Mean age	60.0		58.7		62.6		62.0	

Even with these limitations, 909 deaths were found which were known to have occurred among Jewish persons. These deaths constitute 18.6 percent of the 4,885 deaths from acute coronary occlusion among white persons. The Jewish population of Philadelphia in 1930

was 270,000, or 15.3 percent of the white population. On the basis of the known deaths among Jewish persons, the mortality from acute coronary occlusion appears to be slightly higher than among the non-Jewish white population. The slightly increased mortality probably can be explained on the basis of the large Russian-Jewish immigrant population whose age distribution is largely in the period of life most susceptible to this disease.

The mean age at death from acute coronary occlusion among Jewish persons was 60.0 years (table 7) as compared with 62.0 years among white Gentiles. A comparison of the age distribution of deaths from this disease reveals relatively more deaths among Jewish persons in the 50–59- and 60–69-year age decades, but decidedly fewer deaths among Jews than among Gentiles over 70 years of age.

The age distribution of deaths among Jewish males indicated death at considerably younger ages than among Jewish females. For example, 51.1 percent of the deaths among Jewish males occurred before 60 years of age, while only 34.6 percent of deaths among Jewish females occurred before that age. The ratio of Jewish males to females was approximately 2 to 1. In the 40–49-year age decade over five times as many Jewish males as females died from this cause.

DISTRIBUTION OF DEATHS BY MONTHS

The distribution of deaths from acute coronary occlusion by months (adjusted to a 30-day basis) for the 5 years under study corresponds in general to the monthly distribution of deaths from all forms of heart disease and to deaths from all causes (fig. 6). There was, however, a lower percentage of deaths from acute coronary occlusion during the summer months and appreciably more deaths during the last 3 months of the year than of deaths from all forms of heart disease and deaths in general.

Only 53 percent as many deaths occurred in August as in December. While the monthly variation is not as great as in certain acute infectious diseases, and may be influenced by summer vacations, nevertheless it appears significant. Wolff and White (*11*) observed that most of the attacks of acute coronary occlusion in New England occur in the winter. Wood and Hedley (*12*) noted that there were considerably fewer clinical attacks of acute coronary occlusion in Philadelphia during the summer months. Mullins (*13*), in Pittsburgh, expressed a similar view. Master, Dack, and Jaffe (*14*) do not believe that season plays an important role. Recently, Bean and Mills (*15*), in Cincinnati, reviewed the literature and added a number of cases based on their own experience. Their results indicated that attacks of acute coronary occlusion were definitely more frequent during the colder months.

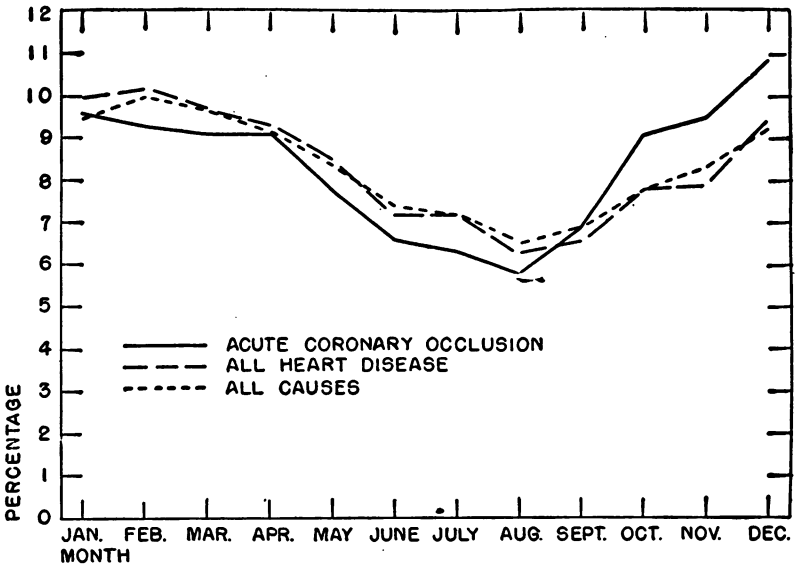

FIGURE 6.—Percentage distribution of deaths from acute coronary occlusion by months (adjusted to a 30-day basis) as compared with deaths from all causes and from all heart disease in Philadelphia from January 1, 1933, to December 31, 1937

Although the monthly distribution of deaths is not necessarily the same as that of acute attacks, the results of these mortality studies give support to the view that season plays a part in the incidence of attacks of this disease.

DEATH RATES

During the period under study the annual crude death rate from acute coronary occlusion more than doubled, increasing from 34 per 100,000 population in 1933 to 78 per 100,000 population during 1937. The mean annual mortality rate for the 5-year period was 52 per 100,000 population (table 8).[2]

The mean annual mortality rate from acute coronary occlusion among white males was 76 per 100,000 population while among white females it was only 37 per 100,000 population, less than half the rate among white males. Among both sexes the rate was 56 per 100,000 population. During each of the years under study the mortality rate among white males was about twice the rate among white females. The mortality rate among white persons increased from 36 per 100,000 in 1933 to 84 in 1937, an increase of more than 100 percent.

[2] All mortality rates in this article are based on the population of Philadelphia according to the United States census of 1930. Owing to the economic depression and to the amount of migration from city to country, country to city, and State to State, it is doubtful whether the usual formulae for estimating population growths during inter-census years are applicable to large cities, especially during the present decade. This probably results in a slight error. Since these deaths occurred among persons past 20 years of age, the death rates were probably not affected to any great extent by the falling birth rate.

Since there were no deaths from acute coronary occlusion among Orientals and other non-Caucasian races, other than Negroes, all of the deaths among colored persons were among Negroes. The total population belonging to other races in Philadelphia in 1930 was only 2,905 persons.

TABLE 8.—*Number of deaths and annual death rates, by color and sex, per 100,000 population from acute coronary occlusion in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937 (populations based on the 1930 census)*

Race and sex	Population	Total 1933–37		1933		1934		1935		1936		1937	
		Deaths	Mean annual death rate	Deaths	Death rate	Deaths	Death rate	Deaths	Death rate	Deaths	Death rate	Deaths	Death rate
Total, both sexes	1,950,961	5,116	52	672	34	803	41	996	51	1,124	58	1,521	78
Male	968,281	3,377	70	439	45	534	55	661	68	738	76	1,005	104
Female	982,680	1,739	35	233	24	269	27	335	34	386	39	516	53
White	1,731,362	4,885	56	622	36	767	44	950	55	1,086	63	1,460	84
Male	859,798	3,253	76	413	48	516	60	634	74	717	83	973	113
Female	871,564	1,632	37	209	24	251	29	316	36	369	42	487	56
Colored	219,599	231	21	50	25	36	16	46	21	38	17	61	28
Male	108,483	124	23	26	24	18	17	27	25	21	19	32	29
Female	111,116	107	19	24	22	18	16	19	17	17	15	29	26

The mean annual mortality rate among colored males was 23 per 100,000 population, while among colored females it was 19 per 100,000 population; for both sexes the rate was 21 per 100,000 population. The mortality from this disease is lower among Negroes and the difference in death rates between the sexes not so marked as in the white race. The mortality from acute coronary occlusion among Negroes did not increase to the extent that it did among white persons. During 1933 the mortality rate from this disease among the Negro population was 25 per 100,000 population, while in 1937 it had increased to only 28 per 100,000, and it showed a tendency to fluctuate during the intervening years.

In table 9 is shown the total number of deaths during the quinquennium under study, and the mean annual age-specific mortality rates by age decades, according to race and sex. The mean annual death rates increased gradually with each age decade in each of the several race and sex groups. Even in the age periods over 70 years there was no precipitous increase in the death rates. As reported in Philadelphia, acute coronary occlusion is not primarily a problem of the age periods beyond 70 years. It should be noted that among white persons the mean annual age-specific mortality rate among males was nearly 4 times as great as that among females in the 40–49-year age decade, over 3 to 1 in the 50–59-year age decade, and over 2 to 1 in the 60–69-year age period. This relationship does not prevail in the Negro population, where the ratios between males and females were more nearly equal in each age group.

It should be noted that the age-specific death rates among Negroes are considerably lower than among white persons. While it is prob-

ably true that the lower incidence of mortality from this disease among Negroes is due in part to a younger age distribution of the colored population, the age-specific mortality rates are also lower than in comparable age groups among white persons.

TABLE 9.—*Number of deaths and mean annual age-specific death rates by age decades, according to color and sex, per 100,000 population from acute coronary occlusion in Philadelphia for the period from Jan. 1, 1933, to Dec. 31, 1937 (populations based on the 1930 census)*

TOTAL

Age (years)	Total, both races			Male			Female		
	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000
20 to 29	356,592	21	1	173,532	10	1	183,060	11	1
30 to 39	333,058	160	10	168,415	107	13	164,643	53	6
40 to 49	259,787	663	51	132,925	521	78	126,862	142	22
50 to 59	181,963	1,299	143	90,085	952	211	91,878	347	76
60 to 69	108,545	1,720	317	51,853	1,111	428	56,692	609	215
70 to 79	44,083	1,003	455	19,611	566	577	24,472	437	357
80 and over	10,165	248	488	3,759	108	575	6,406	140	437
Total over 20 years of age	1,294,193	[1] 5,114	79	640,180	3,375	105	654,013	1,739	53

WHITE

Age (years)	Both sexes			Male			Female		
	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000
20 to 29	306,339	15	1	150,417	8	1	155,922	7	0.9
30 to 39	285,323	132	9	144,099	91	13	141,224	41	6
40 to 49	229,663	605	53	116,808	487	83	112,855	118	21
50 to 59	167,316	1,222	146	82,046	910	222	85,270	312	73
60 to 69	103,446	1,675	324	49,327	1,092	443	54,119	583	215
70 to 79	42,429	990	467	18,878	556	589	23,551	434	369
80 and over	9,704	244	503	3,597	107	595	6,107	137	449
Total over 20 years of age	1,144,220	4,883	85	565,172	3,251	115	579,048	1,632	56

COLORED

20 to 29	50,253	6	2	23,115	2	2	27,138	4	3
30 to 39	47,735	28	12	24,316	16	13	23,419	12	10
40 to 49	30,124	58	39	16,117	34	42	14,007	24	34
50 to 59	14,647	77	105	8,039	42	104	6,608	35	106
60 to 69	5,099	45	177	2,526	19	150	2,573	26	202
70 to 79	1,654	13	157	733	10	273	921	3	65
80 and over	461	4	174	162	1	123	299	3	201
Total over 20 years of age	149,973	231	31	75,008	124	33	74,965	107	29

[1] Excluding 2 deaths of unknown age.

That the increase in reported mortality is gradual and fairly constant in each successive age decade is shown in table 10. In this table are given the age-specific death rates, by decades, per 100,000 population for all deaths from this cause in each of the 5 years studied, together with the percentage increase in each age decade in the year

1937 as compared with 1933. For the ages 20–29 there was an actual decrease, probably due to better diagnosis. In the age decade 30–39 years there was a 40-percent increase. In the four decades between 40 and 79 years the increase was fairly constant. There was relatively no greater increase in the 40–59-year age group than in the older groups. Likewise, the increase in the 60–79-year age group was not out of proportion to that in the 40–59-year age group. Among persons over 80 years of age the increase in the mortality rate in 1937 over 1933 was 176 percent, slightly more than in the younger age periods. When it is considered that the *total number* of deaths attributed to acute coronary occlusion among persons over 80 years of age was only 80 in 1937 as compared with 29 in 1933, this increase in the death rate during this age period does not seem to be an important factor.

TABLE 10.—*Age-specific death rates for acute coronary occlusion per 100,000 population by age decades during each year under study in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937, and percentage increase in specific death rates by age decades in 1937 over 1933 (population based on the census of 1930)*

Age (years)	1933	1934	1935	1936	1937	Percent increase in 1937 over 1933
20 to 29	3	0.8	0.6	0.8	0.8	(1)
30 to 39	10	7	12	6	14	40
40 to 49	32	35	56	57	75	134
50 to 59	97	114	140	158	206	112
60 to 69	204	250	288	358	485	138
70 to 79	272	395	449	485	674	148
80 and over	285	334	423	610	787	176
Total death rates among persons over 20 years of age	52	62	77	87	118	127

[1] Decrease.

As has been stated previously, acute coronary occlusion as reported in Philadelphia is not primarily a problem among persons over 70 years of age. Its increase cannot be attributed to an indiscriminate reporting of deaths among elderly persons as due to that cause. In table 11 is shown a comparison of the mean annual age-specific mortality rates by decades among persons over 20 years of age from acute coronary occlusion with the age-specific mortality rates from all heart disease during this period. Although the age-specific death rates from acute coronary occlusion increase to a certain extent with each successive decade, the rise is by no means as great as that for all forms of heart disease.

Furthermore, while acute coronary occlusion accounts for 26.0 percent of the total recorded heart disease mortality in the 50–59-year age period, with each succeeding age decade it is responsible for a smaller percentage of the total mortality from heart disease. In the age group over 80 years of age only 5.5 percent of the recorded mortality from heart disease was due to this cause.

TABLE 11.—*Comparison of number of deaths and age-specific death rates from acute coronary occlusion with reported mortality from all heart disease in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937 (population based on the census of 1930)*

Age group	Population	Coronary occlusion		All forms of heart disease		Percentage of total heart disease due to acute coronary occlusion
		Deaths	Mean annual specific death rate per 100,000 population	Deaths	Mean annual specific death rate per 100,000 population	
20 to 29	356,592	21	1	483	27	4.3
30 to 39	333,058	160	10	1,100	66	14.5
40 to 49	259,787	663	51	2,817	217	23.5
50 to 59	181,963	1,299	143	4,992	549	26.0
60 to 69	108,545	1,720	317	8,492	1,565	20.3
70 to 69	44,083	1,003	455	8,508	3,860	11.8
80 and over	10,165	248	488	4,474	8,803	5.5
Total	1,294,193	[1] 5,114		30,866		

[1] Excluding 2 deaths of unknown age.

COUNTRY OF BIRTH

In table 12 is shown the mean age at death, arranged by country of birth, of 5,116 deaths attributed to acute coronary occlusion reported from all sources, and of 703 deaths from this cause in hospitals approved for interneship by the American Medical Association. On the basis of mortality reported from all sources, the mean age at death among white native-born Americans was 61.7 years. The mean age at death among persons of German birth was 66.7 years, while the mean age at death among the Italian-born was only 57.2 years. The mean age at death among white persons born in other countries was between these extremes. The same distribution obtained for deaths occurring in hospitals, except that, as a rule, the mean ages were slightly less.

TABLE 12.—*Number of deaths and mean age at death from acute coronary occlusion, according to country of birth, among 5,116 fatal cases reported from all sources and 703 cases reported among regularly admitted patients to hospitals approved for interneship by the American Medical Association in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937*

Country of birth	Deaths reported from all sources		Deaths in hospitals approved for interneship	
	Number of deaths	Mean age (years)	Number of deaths	Mean age (years)
United States, white	2,903	61.7	372	60.2
United States, colored	231	52.3	40	49.0
Russia	603	59.5	106	57.6
Germany	253	66.7	33	67.8
Ireland	261	64.5	33	62.5
Austria-Hungary	127	60.0	15	58.3
Italy	182	57.2	22	57.3
England, Scotland, and Wales	201	65.9	20	62.3
Poland	83	58.0	7	57.7
Other countries	171	58.6	31	57.7
Unknown	101	61.3	24	59.1
Total	5,116	61.2	703	59.5

This difference in the mean age at death among white persons born in foreign countries is explainable largely on the basis of the age distribution of foreign-born groups because of immigration. For example, over 60 percent of the German-born population in the United States in 1930 migrated to this country before 1900, while less than 20 percent of the Italian-born population came to this country before the turn of the century. The distribution of the period of immigration of other foreign-born white persons, with the possible exception of the Austro-Hungarians, occurred between these extremes. Among the Austro-Hungarians, the immigration occurred largely from 1900 to 1914, with comparatively little immigration before or after that period. For a more detailed consideration of immigration, reference is made to the United States census of 1930 (*16*).

The number of deaths from acute coronary occlusion during 1933–37 in age periods of 20 years among white persons born in certain countries, and the mean annual age-specific death rates in 20-year age periods per 100,000 population is shown in table 13.

TABLE 13.—*Number of deaths during the 5-year period and mean annual age-specific death rates per 100,000 population from acute coronary occlusion among white persons in 20-year age periods among persons over 25 years of age in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937, by country of birth (population from census of 1930)*

Country of birth	Age period											
	Over 25 years			25 to 44 years			45 to 64 years			65 and over		
	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000	Population	Deaths	Mean annual rate per 100,000
United States	650,478	2,904	89	399,993	247	13	202,169	1,428	141	58,316	1,229	421
Russia	72,310	603	167	40,459	32	16	26,998	393	291	4,853	178	734
Germany	34,132	252	148	11,642	7	12	14,739	83	113	7,721	162	420
Ireland	48,091	271	113	16,546	6	7	22,994	123	107	8,551	142	332
Austria-Hungary	16,553	128	155	8,800	10	23	6,723	76	226	1,030	42	816
Italy	61,118	182	60	35,527	16	9	21,329	121	113	4,262	45	211
England, Scotland, and Wales	32,089	197	123	12,234	7	11	14,339	73	102	5,516	117	424
Poland	28,358	82	58	17,264	6	7	9,745	53	109	1,349	23	341

The mean annual death rate among white native-born Americans over 25 years of age was 89 per 100,000 native-born white population. There are more native-born than foreign-born persons, however, under 45 years of age. The mean annual death rate among Italian-born white persons was only 60 per 100,000 Italian-born population, while among the Polish-born population it was only 58 per 100,000 population. The highest death rates per 100,000 population among persons over 25 years of age were among the Germans, Austro-Hungarians,

and Russians. The Russians and the Austro-Hungarians, with mean annual mortality rates per 100,000 persons over 25 years of age of 167 and 155, respectively, had higher mortality rates than the Germans, among whom the mean annual mortality rate was 148 per 100,000 population, despite a younger age distribution of their populations.

From both a clinical and a public health point of view the age period between 45 and 64 years is the most important period. The mean annual mortality rate in this period among native-born Americans was 141 per 100,000 native-born population. The highest mean annual age-specific mortality rate in this age period was among persons of Russian birth, being 291 per 100,000 population. The Austro-Hungarians were second with a mean annual age-specific mortality rate of 226 per 100,000 population. Among the national groups with the lowest mean annual age-specific mortality rates between 45 and 64 years of age were the Germans, Italians, Poles, Irish, and persons born in Great Britain.

Of the 603 fatal cases of acute coronary occlusion among persons of Russian birth, 97 percent were among Jews. It is not possible to state whether the increased mortality among persons of Russian birth during the 45–64-year age period occurs primarily because these persons are Russians or because they are Jews. The high death rate may be due partly to greater acumen on the part of Jewish physicians in diagnosing this condition.

Unfortunately, the census statistics do not subdivide the Jewish population into age groups. The mean annual crude death rate from acute coronary occlusion among Jews was 67 per 100,000 population, based on an estimate of 909 deaths during the 5-year period in a Jewish population of 270,000 persons, as indicated in the 1930 census. This is a conservative estimate, since it was not possible to tabulate all of the deaths. Among the non-Jewish white population the crude mortality rate was about 53 per 100,000 population. It seems probable that the mortality from this cause among Jewish persons somewhat exceeds that among white non-Jewish persons. This is probably offset to a certain extent by a possible difference in age distribution, but it is also counterbalanced by unreliable figures regarding the total number of deaths among Jews. It is doubtful whether the mortality from acute coronary occlusion among native-born Jewish persons is much greater than that of native-born white Gentiles. Any excess might be due to a high incidence of diabetes mellitus among Jewish persons.

OCCUPATION OF DECEDENTS

The opinion has been frequently expressed that coronary artery disease occurs more frequently among persons in the business and

professional groups than among persons engaged in other occupations. Few attempts have been made to support or refute this statement.

It is indeed difficult to throw light on this perplexing and important question. It is extremely doubtful whether any of the clinicians writing on this subject have seen a sufficient number of cases in all walks of life to serve as an accurate basis for comparison. Cardiologists engaged in consultation practice deal for the most part with patients among the well-to-do classes. Studies based on clinic patients deal largely with the incidence of this disease among the less fortunate economic groups. Most studies of vital statistics and many hospital studies have consisted in efforts to reconstruct diagnoses made in the past to fit present-day conceptions of this disease.

Denny (*17*), in a study of deaths from heart disease in Brookline, Mass., during the period from 1900–1935, reclassified the deaths recorded as heart disease on the basis of the current provisions of the International List of Causes of Death, thus providing rubrics for deaths considered by him as due to "angina pectoris" and "diseases of the coronary arteries." Among 489 men considered to have died of "angina pectoris and coronary disease" during this period, 67 percent were among the business and professional classes, while among 704 men whose deaths were attributed to "myocarditis" and "other diseases of the heart" (titles 93 and 95 of the International List of Causes of Death), only 48.7 percent were business and professional men.

This does not appear to be conclusive evidence of a predisposition to death from coronary disease among business and professional groups because the study was based on a reconstruction of diagnoses made long ago and hence susceptible to arbitrary selection, because the term myocarditis is too indefinite and has been the source of much abuse in the past (*18*), and because no effort was made to study the component age groups. Furthermore, it does not necessarily follow that because "myocarditis and other diseases of the heart" are less frequently assigned as causes of death among business and professional groups, "angina pectoris" and "coronary diseases" actually occur more frequently.

Levy, Bruenn and Kurtz (*19*), on the other hand, in a study based on the number of cases of coronary disease found at autopsy among all cases examined post mortem at the Presbyterian Hospital in New York City from 1910 to 1931, found that coronary disease occurred slightly more often among foremen and skilled workers than among other occupational groups, but that, in general, occupation did not appear to play an important role in the occurrence of arterial damage. Consideration was not given to the age at death among the various occupational groups. Furthermore, it is extremely

difficult to obtain a proper classification of skilled versus unskilled workers, a matter which will be subsequently discussed in detail.

Recently, Gordon, Bland, and White (*20*) studied the incidence and degree of significant coronary disease among 600 private patients examined post mortem as compared with 2,800 ward patients examined post mortem at the Massachusetts General Hospital from 1925 to 1937. In the age period between 40 and 60 years they found that 24 percent of the private patients examined post mortem had acute coronary occlusion or myocardial infarction as compared with 12.2 percent among ward patients. This study suggests a higher incidence of acute coronary occlusion among persons in the well-to-do economic classes, provided the two groups examined post mortem were reasonably comparable.

During 1936–37 the United States Public Health Service, through the cooperation of the Works Progress Administration, conducted a National Health Survey. In Philadelphia 122,270 persons, or about 6 percent of the population, were enumerated. This group may be assumed to be a roughly representative sample of the population from the viewpoint of age, race, sex, and economic status.

From both the public health and the clinical point of view the most important age period during which persons employed in gainful occupations die from acute coronary occlusion is from 35 to 64 years. In this 30-year age period there were 18,417 white males enumerated in Philadelphia during the National Health Survey. Of these, 17,759 persons indicated that they were engaged in some occupation or were seeking work. The remainder were retired, disabled, or had no known form of employment.

The system of coding the various occupations into occupational groups used in the National Health Survey was that developed by Dr. Alba M. Edwards, of the United States Bureau of the Census. Except in a few minor details it is similar to that described in the Alphabetical Index of Occupations by Industries and Social-Economic Groups (*21*). White males between 35 and 64 years of age are divided into broad occupational groups, with the following percentages in each group:

1. Professional men, including physicians, dentists, architects, attorneys, professional types of engineers, Army and Navy officers, and the like—5.4 percent.

2. Proprietors, managers, and officials, including merchants, dealers, trained executives, contractors, brokers, government officials, and similar occupations—15.4 percent.

3. Clerks and salesmen, including ticket agents, telegraph operators, mail carriers, insurance agents, bookkeepers, credit men, commercial collectors, and the like—16.6 percent.

4. Workers, including foremen, skilled workers, semiskilled workers, apprentices, policemen, firemen, nonprofessional engineers, waiters, servants, helpers, laborers (except farm laborers), and similar occupations—59.1 percent.

5. Retired persons, invalids, persons without known occupational status; also a few farmers and farm helpers—3.5 percent.

Referring to group 4, it was not considered advisable to subdivide this group into skilled, semiskilled, and unskilled workers because physicians filling out death certificates often fail to differentiate between the various occupations as closely as do trained enumerators. For example, a doctor might list a carpenter's helper on the death certificate as a carpenter, which goes into the skilled category, whereas on a census or health survey a carpenter's helper would be classed in the unskilled category. This would also apply to other kinds of helpers, assistants, and apprentices.

It was also possible to determine the number and percentage of white males enumerated in each occupational group in the 10-year age periods between 35 and 64 years. According to the United States Census of 1930 there were 299,990 white males in Philadelphia between 35 and 64 years of age. By applying the occupational status code according to 10-year age periods between 35 and 64 years in the sample of the population enumerated during the National Health Survey to the total white male population of these ages, it was possible to obtain a fairly accurate estimate of the number of persons in each occupational group on the basis of 10-year age periods. By grouping the deaths from acute coronary occlusion in each 10-year period according to the same occupational code, it is possible to estimate the age-specific mortality per 100,000 persons in the 4 broad occupational groups.

TABLE 14.—*Estimated number of white males between 35 and 64 years of age in each occupational group by 10-year age periods, number of deaths from acute coronary occlusion among white males between 35 and 64 years of age by 10-year age periods acccrding to occupation, and the estimated mean annual age-specific mortality rates per 100,000 persons in each occupational group in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937 (deaths listed on a 5-year basis)*

Occupational group	Age period											
	35–44 years			45–54 years			55–64 years			Total, 35–64 years		
	Estimated population	Number of deaths	Estimated mean annual death rate	Estimated population	Number of deaths	Estimated mean annual death rate	Estimated population	Number of deaths	Estimated mean annual death rate	Estimated population	Number of deaths	Estimated mean annual death rate
Professional men	7,684	18	47	5,145	28	109	3,284	78	475	16,113	124	154
Proprietors, managers, and officials	18,462	34	37	17,144	129	150	10,463	159	304	46,069	322	140
Clerks and salesmen	25,915	47	36	14,780	109	147	9,139	163	357	49,834	319	128
Workers, all classes	81,641	118	29	59,677	371	124	36,083	456	253	177,401	945	107
Total	133,702	217	32	96,746	637	132	58,969	856	290	289,417	1,710	118

In table 14 are shown the estimated white male population in each occupational group by 10-year periods between 35 and 64 years of

age, the number of deaths from acute coronary occlusion during the quinquennium under study, and the estimated mean annual age-specific mortality rates per 100,000 persons in each occupational group by 10-year age periods and for the 30-year period between 35 and 64 years of age. Only persons whose occupations are included in the 4 major groups were considered. For this reason, the tabulation was based on 289,417 persons instead of 299,990 persons, the white male population of Philadelphia between 35 and 64 years of age according to the 1930 census. The remainder consisted of retired persons, invalids, persons without known occupational status, and a few farmers.

As seen in table 14, in the age period 35–64 years the death rate from acute coronary occlusion was 154 per 100,000 professional men, 140 per 100,000 proprietors, managers, and officials, 128 per 100,000 clerks and salesmen, and only 107 per 100,000 workers.

In the 35–44-year age period the death rate was highest among professional men (47 per 100,000 persons in that occupational group) and lowest among workers, who had a mean annual occupational-specific mortality rate of only 29 per 100,000. Owing to the relatively small number of deaths these figures are not very significant. In the 45–54-year age period the mortality rate was highest among proprietors, managers, and officials (150 per 100,000). It was next highest among clerks and salesmen (147 per 100,000 persons in that occupational group). Among workers the mean annual mortality rate was 124 per 100,000 persons in that group, while among professional men it was only 109 per 100,000 persons. In the 55–64-year age group the mean annual age-specific mortality rate was 475 per 100,000 professional men, much higher than in the other groups. It was next highest among clerks and salesmen (357 per 100,000 persons). Among proprietors, managers, and officials it was 304 per 100,000 persons in that group. Among workers it was only 253 per 100,000 persons in that occupational group, a little over half the estimated mortality rate among professional men.

As a check on the accuracy of the estimated number of persons in the various occupational groups, a study was made of the white male population of Philadelphia by occupational groups as determined by the United States Census of 1930. According to the census returns for that year the distribution of gainfully employed white males of *all ages* was:

Occupational group	*Number of white males*	*Percent of total*
Professional men	28, 293	5. 0
Proprietors, managers, and officials	67, 937	12. 0
Clerks and salesmen	117, 333	20. 7
Workers (all classes)	353, 990	62. 4

While there is no information available on the age distribution within the various occupational groups in Philadelphia, this information is available for the entire United States. Reference is made to table 6 on page 22 and table 15 on page 40 of the Social-Economic Grouping of the Gainful Workers of the United States (*22*). By applying these tables it was possible to make another estimate of the number of professional men, proprietors, managers, and officials (excluding farmers), clerks and salesmen, and workers (all classes) in the white male population between 35 and 64 years of age. It is probably not as accurate as the estimate based on the sample of the population obtained in the National Health Survey because the age distribution in the occupational groups may have been different in Philadelphia from that of the entire United States, because of minor differences in coding, and because of the effects of the depression.

On this basis, however, there were 14,883 professional men, 46,333 proprietors, managers, and officials, 50,219 clerks and salesmen, and 163,897 workers (all classes) among the white male population between 35 and 64 years of age. These figures do not differ greatly from the estimates given in table 14. The age distribution was also quite similar. Based on these figures the mean annual occupational age-specific mortality rates among white males between 35 and 64 years of age during the period under study were approximately the same as those in table 14, being 167 per 100,000 professional men, 139 per 100,000 proprietors, managers, and officials, 127 per 100,000 clerks and salesmen, and 115 per 100,000 workers (all classes). Owing to minor differences in coding the various occupational groups, these estimates are not considered as accurate as those given in table 14.

Caution is urged in interpreting the apparently higher mortality rates among professional men and among proprietors, managers, and officials for the following reasons:

(1) The standards of diagnosis may have been higher for these groups than for clerks, salesmen, and workers. This is suggested by the fact that there was a somewhat smaller percentage increase in the reported mortality among professional men than among the other groups. Compared to the number of deaths reported in 1933, there was an increase of 118 percent among professional men between 35 and 64 years of age in 1937, while among proprietors, managers, and officials the increase was 166 percent, among clerks and salesmen 164 percent, and among workers 185 percent. On the other hand, it is possible that the increase was not actually as great in the professional group.

(2) Among white males between 35 and 64 years of age there were 2,072 deaths attributed to acute coronary occlusion. In addition to the 1,710 deaths classified according to occupational group in table 14, there were 11 among farmers, 114 among persons listed as retired,

and 237 among persons without known occupations. It is probable that among those with unknown occupation the greater proportion were workers. This, however, is offset by the probability that a relatively greater proportion of the decedents whose occupations were listed as retired formerly belonged to the business and professional groups.

In the opinion of the writer, the figures in table 14 strongly suggest that the mortality from acute coronary occlusion is highest among men in the business and professional groups. This is particularly so among professional men between 55 and 64 years of age, when the estimated mortality was considerably higher than among other groups, especially among workers. Furthermore, the mortality was not highest among professional men in the 45–54-year age period. This fact tends to lessen the likelihood that better diagnosis was the only factor. It should be borne in mind, however, that the diagnosis of this condition probably has not become sufficiently stabilized to afford comparable accuracy among all groups. These results, therefore, should be regarded as far from conclusive. This study should be repeated at the end of another 5-year period.

It was not considered advisable to extend this study to include white males over 65 years of age, since the occupation of such persons is very often described on the death certificates as "retired," without qualification concerning their former mode of employment. There were so few Negro males in the business and professional groups that it was not possible to make a comparison. Among females of both races the occupation on death certificates is generally listed as "at home," "housewife," or "housework," regardless of previous occupations. Very few are designated as "domestics." For this reason it is almost impossible to determine the real occupational status of females from information on death certificates. The term "housewife" connotes little more than marital status. A housewife may occupy any position from executive to domestic.

IS ACUTE CORONARY OCCLUSION INCREASING?

During the quinquennium under study, the number of deaths reported as due to acute coronary occlusion increased from 672 to 1,521 per annum. Is this increase genuine? To what extent is it fictitious? What factors, other than an actual increase, may be invoked to explain why more deaths were reported each year as due to this cause? Have general practitioners of medicine become sufficiently adept in the diagnosis of acute coronary occlusion to permit many definite conclusions? These and many other questions present themselves.

It is doubtful whether any of these questions can be answered categorically at this time. It is well to take stock, even though it is not

possible to make a complete inventory of all of the factors involved. In attempting to arrive at some conclusion, certain possible considerations will be reviewed for the purpose of elimination. Having disposed of certain factors which might serve to explain this increase, the remaining possibilities will be considered in greater detail:

1. The increase in mortality from acute coronary occlusion is not due to an increased reporting of deaths among persons past 70 years of age. By and large, the reported mortality corresponds quite closely to the age distribution of diagnoses proved by necropsy examinations. Furthermore, despite the great increase in the total number of deaths, the age distribution remained practically the same during these 5 years.

2. Conversely, the increase is not due to relatively more deaths occurring during the age periods prior to 60 years of age. The age distribution and mean age at death remained about the same during the period under study. Theoretically, it might be possible for an actual increase in correct diagnoses under about 65 years of age to be counterbalanced by incorrect diagnoses past that age, so that the age distribution remained the same. This, however, does not seem likely. It would presuppose that most of the younger patients were attended by more competent diagnosticians than the older patients.

3. Although the aging of the population had an influence on the increased mortality from this cause, this factor was not sufficient to account for an increase of 126 percent during a 5-year period. Deaths from all causes, and deaths from such diseases as cancer and other tumors, diabetes mellitus, and heart disease in general during the age decades in which deaths from acute coronary occlusion occurred did not show such a precipitous increase. Furthermore, acute coronary occlusion did not appear to be a problem primarily of the aged.

4. The increase cannot be attributed to any great extent to the aging of the foreign-born population, over and above the aging of the general population. Deaths attributed to acute coronary occlusion from the entire city among the native-born white population increased from 362 in 1933 to 873 in 1937, an increase of 141 percent. Among deaths occurring in hospitals the increase was from 116 in 1933 to 224 in 1937, an increase of 93 percent. Among the foreign-born white population, deaths reported as due to acute coronary occlusion in the city as a whole increased from 224 in 1933 to 520 in 1937, an increase of 132 percent; while deaths in hospitals increased from 65 in 1933 to 132 in 1937, or 103 percent. Throughout the city as a whole there was a smaller increase in deaths among the foreign-born white population. Of deaths in hospitals, the increase was slightly greater among the foreign-born white population, probably owing to greater use of hospital facilities by these persons. There is nothing to suggest that aging of the foreign-born population, most of whom came to this

country before 1914 and are now of the age during which this disease occurs most frequently, was sufficient to account for the increase in mortality from acute coronary occlusion.

This leaves two important considerations: Improved diagnosis and the possibility of an actual increase in mortality from this cause. Of these factors, increased recognition is by far the more outstanding. In the entire annals of medical history, it is doubtful whether there was ever a disease which has been better publicized than acute coronary occlusion during the past 15 years. At first it was regarded as a rather rare condition; later as a diagnosis which could only be made by specially qualified experts; now it is being made by nearly every general practitioner.

A person has only to look at the appropriate sections of the indexes of medical literature since 1927 to comprehend the size and rapidity of growth of the literature on this condition. All of the textbooks dealing with this subject, with the possible exception of some of the loose-leaf systems of medicine, have been written since 1929. During this period the diagnosis has been exploited from almost every conceivable point of view—in medical periodicals, local, State, and national meetings, in post-graduate courses, in hospital staff meetings and clinico-pathological conferences, and in various types of clinics.

By the beginning of the year 1933, the clinical diagnosis of acute coronary occlusion with myocardial infarction had become well established as a result of the pioneer efforts of Herrick (*1, 23*), Levine (*3*), Conner and Holt (*24*), Parkinson and Bedford (*25*), Pardee (*26*), and others too numerous to mention. Although the clinical diagnosis of this condition was somewhat improved by better recognition of atypical cases, it is doubtful whether many profound advances were made in the recognition of purely clinical signs and symptoms, except by way of differential diagnosis, during the period from January 1, 1933, to December 31, 1937.

This period was characterized, however, by a better appreciation of this disease by the mass of general practitioners. There is nearly always a considerable lag between discovery and general acceptance. It was only in the year 1931 that an accurate account of acute coronary occlusion was first given in a textbook on heart disease. Time is required for the leaven to work. Each year more physicians recognize that what they have been diagnosing as angina pectoris is often coronary thrombosis. This is reflected in mortality returns.

Even under hospital conditions there was considerable improvement in diagnosis during this period. Philadelphia is the home of electrocardiographic diagnosis by chest leads. In January 1932, Wolferth and Wood (*27*) published their first paper on the diagnosis of acute coronary occlusion by this method. Although by the beginning of 1933 this valuable diagnostic adjunct was being used in a number of

hospitals in Philadelphia, it had not gained general acceptance. It is doubtful whether many experienced cardiologists had seen a sufficient number of cases for accurate appraisal. During the period under study, many elaborations and refinements to this procedure were made. It was not until 1938 that the recommendations of a committee of the American Heart Association (*28*) concerning the standardization of chest leads were published.

Furthermore, there has been an extension in the use of the electrocardiograph in recent years. Whereas formerly its use was largely limited to hospitals, better and cheaper electrocardiographs are now available for the study of this condition in the home.

It must be remembered that most of the fatal cases are not examined with the electrocardiograph. Nearly one-half of the deaths from acute coronary occlusion occur in the home, while over one-third are coroner's cases. The electrocardiograph is a valuable instrument for localizing the site of a myocardial infarction as an aid to prognosis, and for determining the stage of the healing process in certain cases. It is also of value in detecting small occlusions, or previous occlusions in the face of negative or doubtful physical findings. Most severe attacks, however, can be diagnosed by a competent physician on the basis of the clinical history, and, to a lesser extent, on physical findings.

There was also considerable improvement during the 5 years in the differential diagnosis of acute coronary occlusion from such conditions as pulmonary embolisms, dissecting aneurysms of the aorta, acute gall bladder diseases, perforated peptic ulcers, and other conditions. Of these, a better differentiation between acute coronary occlusion and acute cor pulmonale was probably the most important from the standpoint of mortality reporting.

Even in the field of necropsy diagnosis there was probably an appreciable degree of improvement. Although the necropsy table remains the final court of clinical appeal, even a supreme court may amplify or reverse its decisions. With better electrocardiographic diagnoses it seems quite likely that greater efforts were made to locate small but important myocardial infarctions, especially on the posterior surface of the heart.

Closely related to the question of better diagnosis is the problem of fads in diagnosis. Denny (*17*) recently remarked that once a physician starts making the diagnosis of acute coronary occlusion, he ceases reporting deaths as due to angina pectoris. Examination of death certificates in Philadelphia confirms this observation. This probably explains in part the increase in reported mortality from acute coronary occlusion in Philadelphia; it does not entirely explain it. Although compiled by a different method from that in which the deaths are reported in this series, reference is made to deaths officially recorded by the local office of vital statistics as due to angina pectoris

and diseases of the coronary arteries. In 1933 there were 212 deaths attributed to angina pectoris and 314 deaths attributed to diseases of the coronary arteries. In 1937 there were 193 deaths attributed to angina pectoris and 606 deaths attributed to diseases of the coronary arteries. Although the recorded mortality from diseases of the coronary arteries had increased nearly 100 percent, the decline in mortality from angina pectoris was less than 10 percent. It is doubtful that changes in diagnostic terms have constituted a very important factor in the increased mortality reported as due to acute coronary occlusion in Philadelphia during these 5 years.

Improvement in diagnosis has undoubtedly been an important factor in the increase in deaths attributed to this cause during the period under study. Of less importance has been the aging of the population, both native- and foreign-born. Even after allowance has been made for these factors, an increase of 126 percent during a period of 5 years should not be dismissed summarily.

In hospitals approved for interneship by the American Medical Association (see table 1), there was an increase of 70 percent during these 5 years, while admissions for all causes increased only 9.5 percent. In a selected group of 11 hospitals, including 6 affiliated with medical schools, the Philadelphia General Hospital, and 4 other hospitals, 3 of which are used for teaching purposes, whose cardiologists have been especially interested in this problem, the number of deaths from acute coronary occlusion among regular admissions increased from 85 in 1933 to 137 in 1937, or over 60 percent. During this period there was no increase in admissions for all causes. Can it be stated with justification that the diagnostic acumen of members of the medical staffs of these hospitals increased to that extent during so short a period? Is there sufficient ground to explain this increase almost entirely on the basis of better diagnosis?

Although there were numerous refinements in electrocardiographic technique during this period, the diagnosis of acute coronary occlusion is usually based on the clinical picture as seen at the bedside, or on a history of previous attacks. While there has been a spread of knowledge concerning this disease to the mass of practitioners seeing patients in the home, by the beginning of 1933 medical staffs of large metropolitan hospitals, especially teaching institutions, usually had a very definite conception of this disease. Improvement in diagnosis in hospital practice has consisted largely in a better recognition of the fact that coronary occlusive phenomena occur more frequently during the course of so-called degenerative cardiovascular diseases. Many of the cases now diagnosed but formerly not diagnosed do not result fatally, as witnessed by a tendency toward lower case fatality rates in recent years. Furthermore, improvement in diagnosis is being offset to a certain extent by counter-improvement in differential diagnosis.

It is doubtful whether the increased mortality in hospitals can be explained on the basis that more patients were admitted to hospitals because of greater interest in this disease. According to table 1, 17.0 percent of the deaths in 1933 occurred among patients regularly admitted to hospitals, as compared with 12.8 percent in 1937. Furthermore, there has been an extension in the use of such facilities as portable electrocardiographs and oxygen tents so that patients can be treated more efficiently in the homes. It must also be borne in mind that acute coronary occlusion is so sudden in its onset and the shock so great that the possibilities of selecting the place of treatment are often quite limited.

TABLE 15.—*Comparison of number of deaths, by years, attributed to acute coronary occlusion among white persons and Negroes reported from all sources and in 26 civilian hospitals approved for interneship by the American Medical Association in Philadelphia from Jan. 1, 1933, to Dec. 31, 1937, with the percentage increase in 1937 over 1933*

Year	Deaths reported from all sources		Deaths in hospitals	
	White	Colored	White	Colored
1933	622	50	103	11
1934	767	36	92	4
1935	950	46	148	11
1936	1,086	38	137	3
1937	1,460	61	183	11
Total	4,885	231	663	40
Percent increase in 1937 over 1933	135	22	78	0

Another feature which is difficult to explain solely on the basis of improved diagnosis is why the reported mortality from acute coronary occlusion increased so much more among white persons than among Negroes. According to table 15, there was an increase of 137 percent in the reported mortality from all sources among white persons in 1937 over 1933, while among Negroes the increase was only 22 percent. In deaths from this cause occurring among regular admissions to 26 civilian hospitals approved for interneship by the American Medical Association there was an increase of 78 percent among white persons but no increase among Negroes. Furthermore, the incidence of deaths in hospitals among both white and colored showed a tendency to fluctuate from year to year. Even among white persons the increase in mortality was not a gradual one. Furthermore, the mortality rate among white persons increased from 36 per 100,000 population in 1933 to 84 per 100,000 population in 1937. Among Negroes the increase was only from 25 per 100,000 population in 1933 to 28 per 100,000 in 1937 (see table 8).

The improvement in diagnosis, especially in hospitals, should have applied equally to both races. In Philadelphia, with the possible

exception of one hospital, the two races are treated on the same wards and on the same services in all of the large hospitals. Although the *incidence* of this disease appears to be lower among Negroes, the *percentage increase* should have been approximately the same as among white persons if improvement in diagnosis were the only factor. Since there was no increase among Negroes during this 5-year period, while deaths among white persons in hospitals increased 78 percent, it is difficult to avoid the impression that part of this increase may be due to an actual increase in mortality from this disease among white persons.

As mentioned previously, the increase in deaths from this cause among native-born white persons is about the same as that among foreign-born white persons. Although there has been practically no immigration of Negroes to this country during the present generation, there was a large migration of Negroes to Philadelphia from the South during the years immediately following the World War. The Negro population in this city increased 69 percent between 1920 and 1930. Many of these Negroes have now reached the age during which acute coronary occlusion occurs. The lack of increase cannot be explained on the basis of a more stable population.

The previous discussion should not be interpreted as indicating that there has been an actual increase of 78 percent in deaths from acute coronary occlusion among white persons in hospitals, or over 100 percent in the city as a whole. The outstanding factor in the increase in reported mortality is improvement in diagnosis. However, the writer raises the question of whether *all* of the mortality from acute coronary occlusion among white persons may be attributed to this cause.

For the present, the possibility of a certain amount of actual increase in acute coronary occlusion should be viewed with an open mind. It is possible that there are factors, besides the aging of the population and improvement in diagnosis, which may be responsible for an increase in this condition among the urban population in particular. These factors may operate either to predispose to coronary atherosclerosis or to result in the occlusion of coronary arteries previously diseased. Until more is known concerning this extremely intricate phenomenon which is responsible for so many deaths among middle-aged persons, the increase in deaths reported from so many different sources should not be dismissed summarily as due to the aging of the population, to improved standards of clinical diagnosis, or to statistical artifacts due to changes in terminology.

SUMMARY AND CONCLUSIONS

1. In a 5-year period marked by many advances in the clinical diagnosis of acute coronary occlusion, the total mortality attributed

to this cause increased over 125 percent in Philadelphia. The mortality among patients regularly admitted to hospitals approved for interneship by the American Medical Association increased 70 percent, while coroner's cases attributed to this cause increased 108 percent. Deaths in other hospitals and in the homes increased 167 percent.

2. The diagnosis of this condition cannot be regarded as stabilized at this time.

3. Despite the great increase in the number of deaths reported as due to acute coronary occlusion, the age distribution by decades and the mean age at death remained practically the same for each year during the period under study. The age distribution of deaths attributed to this cause corresponds quite closely to that of necropsied cases in this and other series, and, in general, to the age distributions described in the literature. The increase in deaths from acute coronary occlusion cannot be attributed to a tendency to report deaths among very old persons as due to this cause.

4. Of the 5,116 deaths attributed to acute coronary occlusion in Philadelphia during this 5-year period, 703, or 13.7 percent, were reported as occurring among regularly admitted patients to 26 civilian hospitals approved for interneship by the American Medical Association, 1,868, or 36.5 percent, were coroner's cases, while 2,545, or 49.8 percent, were reported from other hospitals and by general practitioners. Of the deaths occurring in hospitals, in 197 instances the diagnosis was confirmed by necropsy examination.

5. The ratio of males to females was approximately 2 to 1 among deaths reported from all sources; among deaths in 26 civilian hospitals approved for interneship by the American Medical Association this ratio was 1.7 to 1; among deaths reported from other hospitals and by general practitioners the ratio of males to females was 1.5 to 1. The ratio of deaths among males as compared with females is lower than that reported by most writers. This may be due to fewer sources of error in the selection of cases. In the series as a whole it is probably due to more deaths among females past 70 years of age.

6. The mean age at death among all cases was 61.2 years. Among deaths occurring among regularly admitted patients to 26 civilian hospitals approved for interneship by the American Medical Association, the mean age at death was 59.5 years. Among diagnoses confirmed by post-mortem examination, the mean age at death was 59.8 years. The mean age at death among coroner's cases was 58.1 years. The mean age of deaths occurring in the homes and in other hospitals was 63.9 years.

7. The mean age at death and age distribution by age decades indicated that deaths among white females occur at older ages by several years than among white males.

8. Although acute coronary occlusion occurs less frequently among Negroes than among white persons, deaths from this cause among Negroes occur several years younger. The mean age at death of cases reported from all sources among white persons was 61.6 years, while among Negroes it was only 52.3 years. Similar differences were found among deaths in hospitals, cases studied post mortem, coroner's cases, and deaths reported by general practitioners. As regards differences between the sexes, the mean age at death, age distribution, and ratio of males to females was more nearly equal among Negroes than among white persons.

9. With the exception of the coroner's cases the maximum number of deaths in all of the groups studied occurred in the 60–69-year age decade.

10. Among the coroner's cases the peak incidence occurred in the 50–59-year age decade among all cases. Among females, it occurred in the 60–69-year age period. There were over eight times as many deaths among white males among the coroner's cases in the 40–49-year age decade as among white females. In the 50–59-year age decade over four times as many deaths were reported by the coroner among white males as among white females. Only 7.0 percent of the coroner's cases were among Negroes. The problem of sudden death from heart disease, especially among white males, should be made the subject of well-planned research projected over a number of years.

11. The mortality from acute coronary occlusion appeared to be somewhat higher among Jewish persons than among white Gentiles. This may be due to the large number of Russian Jews and may not prevail among the native born. The age and sex distribution among Jews was not greatly different from that encountered in non-Jewish white persons. The ratio of deaths among Jewish males as compared with Jewish females in the 40–49-year age period was over 5 to 1.

12. Although the monthly distribution of deaths from acute coronary occlusion did not show as marked seasonal variations as obtain in mortality from certain infectious diseases, considerably fewer deaths were reported during the warm months. Only 53 percent as many deaths occurred in August as in December.

13. The mean annual mortality rate from acute coronary occlusion for the 5-year period was 52 per 100,000 population. The mortality rate increased from 34 to 78 per 100,000 during the 5-year period. The mean annual mortality rate among white persons was 56 per 100,000 population; among white males it was 76; and among white females it was 37 per 100,000 population. The mean annual mortality rate among Negroes was 21 per 100,000 population. Among Negro males it was 23, while among Negro females it was 19 per 100,000.

14. The mortality rate among white persons increased from 36 per 100,000 population in 1933 to 84 per 100,000 population in 1937. Among Negroes the increase was only from 25 per 100,000 in 1933 to 28 per 100,000 in 1937.

15. Acute coronary occlusion is not primarily a problem among persons past 70 years of age. The age-specific mortality rises very gradually as compared with heart disease in general. Acute coronary occlusion accounted for only 5.5 percent of the total mortality from heart disease among persons past 80 years of age as compared with 26.0 percent in the 50–59-year age decade. The chief importance of this problem from both a clinical and a public health point of view consists in the number of deaths among persons in the 40–69-year age period.

16. The highest death rates occurred among persons born in Russia and in Austria-Hungary.

17. Among white males between 35 and 64 years of age the estimated mean annual mortality rate from this cause was 154 per 100,000 professional men, 140 per 100,000 proprietors, managers, and officials, 128 per 100,000 clerks and salesmen, and 107 per 100,000 workers, including skilled and unskilled workers and foremen. Among professional men it was especially high in the 55–64-year age period. In view of the possibility that diagnostic standards were not the same among these four groups, caution is urged in the interpretation of these results.

18. The increase in reported mortality from acute coronary occlusion during this period cannot be attributed to any great extent to the aging of the population in general or to the aging of the foreign-born population in particular.

19. Improvement in diagnosis was quite probably the outstanding factor responsible for the increase in reported mortality from acute coronary occlusion during the quinquennium under study.

20. The reported mortality from acute coronary occlusion increased 70 percent during this period in hospitals approved for interneship by the American Medical Association. Among a group of hospitals used for teaching purposes, affiliated with medical schools, or in hospitals where cardiologists are known to have been keenly interested in this problem, the number of deaths attributed to this cause was 60 percent greater in 1937 than in 1933. Doubt is expressed whether this increase can be attributed solely to better diagnostic standards.

21. During the period under study there was an increase of 78 percent in the mortality from acute coronary occlusion among white persons regularly admitted to 26 civilian hospitals approved for interneship by the American Medical Association, while there was no increase in mortality from this cause among colored persons. Among deaths attributed to this cause from all sources there was an increase

of 135 percent among white persons as compared with 22 percent among Negroes. Since both white and colored patients in the hospitals of Philadelphia are subjected to similar criteria and standards of diagnosis, it is difficult to escape the conclusion that there may have been a certain amount of actual increase in mortality from acute coronary occlusion among white persons in Philadelphia during the 5 years under investigation.

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REFERENCES

(1) Herrick, J. B.: Clinical features of sudden obstruction of the coronary arteries. J. Am. Med. Assoc., **59**: 2015 (1912).
(2) Levy, Robert L.: Diseases of the coronary arteries and cardiac pain. Macmillan, New York, 1936.
(3) Levine, S. A.: Coronary thrombosis: Its various clinical features. Williams and Wilkins, Baltimore, 1929.
(4) Saphir, O., Priest, W. S., Hamburger, W. W., and Katz, Louis N.: Coronary arteriosclerosis, coronary thrombosis and the resulting myocardial changes. Am. Heart J., **10**: 567 and 762 (1935).
(5) Appelbaum, Emanuel, and Nicolson, Gertrude H. B.: Occlusive diseases of the coronary arteries; an analysis of the pathological anatomy in 168 cases with electrocardiographic correlation in 36 of these. Am. Heart J., **10**: 662 (1935).
(6) Meakins, J. C. and Eakin, W. W.: Coronary thrombosis: A clinical and pathological study. Canad. Med. Assoc. J., **26**: 18 (1932).
(7) Johnston, Christopher: Racial differences in the incidence of coronary sclerosis. Am. Heart J., **12**: 162 (1936).
(8) Hedley, O. F.: A study of 450 fatal cases of heart disease occurring in Washington (D. C.) hospitals during 1932, with special reference to etiology, race, and sex. Pub. Health Rep., **50**: 1127 (1935).
(9) Hedley, O. F.: Mortality from rheumatic heart disease in Philadelphia during 1936. Pub. Health Rep., **52**: 1907 (1937).
(10) Martland, H. S.: Syphilis of the aorta and heart. Am. Heart J., **6**: 1 (1930).
(11) Wolff, L., and White, P. D.: Acute coronary occlusion. Boston Med. and Surg. J., **195**: 13 (1926).
(12) Wood, F. C., and Hedley, O. F.: The seasonal incidence of acute coronary occlusion in Philadelphia. Med. Clin. North America, **19**: 151 (1935).
(13) Mullins, William L., Age incidence and mortality in acute coronary occlusion: A review of 400 cases. Pennsylvania Med. J., **39**: 322 (1936).
(14) Master, A. M., Dack S., and Jaffe, H. L.: Factors and events associated with onset of coronary artery thrombosis. J. Am. Med. Assoc., **109**: 546 (1937).
(15) Bean, W. B., and Mills, Clarence A.: Coronary occlusion, heart failure, and environmental temperatures. Am. Heart J., **16**: 701 (1938).
(16) Fifteenth Census of the United States. Population. Vol. 11, page 497 (1930) A. Department of Commerce. Bureau of the Census. Government Printing Office, Washington (1933).

(17) Denny, Francis P.: The increase in coronary disease and its cause. New England J. Med., 214: 769 (1936).
(18) Hedley, O. F.: A critical analysis of heart disease mortality. J. Am. Med. Assoc., 105: 1405 (1935).
(19) Levy, Robert L., Bruenn, H. G., and Kurtz, Dorothy: Facts on disease of the coronary arteries based on a survey of clinical and pathological records of 762 cases. Am. J. Med. Sci., 187: 376 (1934).
(20) Gordon, William H., Bland, Edward F., and White, P. D.: Coronary artery disease analysed post mortem. Am. Heart J., 17: 10 (1939).
(21) Edwards, Alba M.: Alphabetical index of occupations by industries and social-economic group (1937). U. S. Department of Commerce, Bureau of the Census. Government Printing Office, Washington (1937).
(22) Edwards, Alba M.: Social-economic grouping of the gainful workers of the United States. U. S. Department of Commerce, Bureau of the Census. Government Printing Office, Washington (1938).
(23) Herrick, J. B.: Thrombosis of the coronary arteries. J. Am. Med. Assoc., 72: 387 (1919).
(24) Conner, L. A., and Holt, E.: The subsequent course and prognosis in coronary thrombosis. An analysis of 287 cases. Am. Heart J., 5: 705 (1930).
(25) Parkinson, J., and Bedford, D. E.: Cardiac infarction and coronary thrombosis. Lancet, 1: 4 (1928).
(26) Pardee, H. E. B.: An electrocardiographic sign of coronary artery occlusion. Arch. Int. Med., 26: 244 (1920).
(27) Wolferth, C. C., and Wood, F. C.: The electrocardiographic diagnosis of coronary occlusion by the use of chest leads. Am. J. Med. Sci., 183: 30 (1932).
(28) Standardization of precordial leads. Joint recommendations of the American Heart Association and the Cardiac Society of Great Britain and Ireland. Am. Heart J., 15: 107 (1938).

SMALLPOX VACCINATION: A COMPARISON OF VACCINES AND TECHNIQUES [1]

By Ralph V. Ellis, M. D., and Ruth E. Boynton, M. D.

During the 142 years since Jenner announced a method of prophylaxis against smallpox, the first essential change concerning vaccine virus preparations was the introduction of purely animal vaccine, which began to supersede arm-to-arm vaccination in the 1870's. Considering the inevitable contamination of the raw product when prepared on the skin of the calf or the child, it is more than probable that in the earlier period vaccine virus preparations contained various species of micro-organisms as contaminants, and that occasionally these may have included pathogens. With the development of the science of bacteriology, technical improvements were made, with the result that pathogenic contaminants are now successfully eliminated. However, the preparation of uniformly bacteria-free calf lymph virus is as yet attainable with difficulty, and the desire has long been widespread for a method of preparation resulting in a vaccine of uniform potency and entirely free from contaminating micro-organisms. Artificial cultivation has been regarded as the method offering greatest probability of fulfilling these requirements.

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Artificial cultivation of vaccine virus was first accomplished by Steinhardt, Israeli, and Lambert by a simple glass technique 25 years ago (*1*). Carrel and Rivers (*2*) modified the Steinhardt technique and adapted it for the cultivation of the virus on a larger scale. In Rivers' final modification (*3*) an emulsion of minced chick embryo in Tyrode's solution was used as a culture medium. The seed for the cultures was virus No. 611 of the Bureau of Laboratories of the New York City Department of Health. After cultivation had been assured, the emulsion was dried, and the virus retained its potency for a long period of time if stored at icebox temperature.

A second method of artificial cultivation has been introduced by Goodpasture (*4*). The successful infection of the chorioallantoic membrane of the chick embryo with fowl pox by Woodruff and Goodpasture led them, in collaboration with Buddingh, to the discovery that the membrane was also susceptible to infection with vaccine virus. Briefly, the Goodpasture technique is as follows: The hen's egg is incubated 14 days. After disinfection, a window is cut in the shell, the chorioallantoic membrane is exposed and inoculated with a sterile dermal strain of vaccine virus, and the window is replaced and sealed in position. Incubation is continued 4 days; the shell is then reopened and the pock-infected allantois is removed, ground in a mortar, and suspended in 50 percent neutral glycerol with normal saline. By either of these methods the problem of bacterial contamination is apparently solved.

From time to time desires have also been expressed for an attenuated vaccine of uniform potency, on the assumption that vaccination would become more generally acceptable if the severity of the reaction and the size of the resulting scar were lessened. Some have voiced a desire for a method which would result in immunity without the scar which has become the accepted sign of immunity. Others have decried this as undesirable, insisting upon the importance of objective evidence. Furthermore, it cannot be denied that an attitude of resistance to vaccination against smallpox embraces a considerable portion of the population. This attitude does not appear to obtain to the same extent in regard to active immunization against diphtheria. In addition, organized effort, widespread in occurrence, is continually attempting to marshal active resistance against all immunization procedures. This appears to be more successful in arousing sentiment against smallpox vaccination than is the case with other immunizations. The public is notoriously susceptible to innovations, and it is not an unreasonable possibility that the introduction of a new method of vaccination against smallpox, involving the elimination of the scar, might go far in overcoming prejudices which have so long existed. It would at least be worthy of trial.

The claim for immunity to smallpox induced without pustulation and scar was made as early as 1866 by Chauveau (*5*), who injected ordinary vaccine material subcutaneously. Nobl (*6*), Knoepfelmacher (*7*), Goodal (*8*), Spearman (*9*), and Kuhle (*10*) were favorably impressed with the method. Janson (*11*) reported failure to produce complete immunity by this method, and Tedeschi (*12*) reported that immunity could be produced in cows by the subcutaneous method but not in man. Nobl (*6*), in commenting on the unfavorable reports of these two workers, attributed Janson's failure to too early use of revaccination as a test of such immunity, and stated that Tedeschi's negative results may have been due to a belief that the appearance of the vesicle was his criterion of success. Knoepfelmacher (*7*) also claimed to have produced immunity in 7 out of 14 cases by subcutaneous injection of undiluted, heat-killed vaccine. Immunization without pustulation and scar is also claimed by Leiner and Kundratitz (*13*) and by Czapski (*14*) by intracutaneous administration of diluted calf lymph vaccine.

On the other hand, Wright (*15*), Twyman, (*16*), Berney (*17*), Toomey (*18*), and many others who have used and stress the advantages of the intracutaneous technique, do not mention the absence of vesiculation and scar as one of these. In fact, it is apparent that reaction of vesiculation and scar was the rule, and that such reaction was the criterion of success so far as primary takes are concerned. These men, however, used the calf lymph virus in various dilutions. The possibility of immunization without the typical Jennerian reaction is either overlooked or ignored by nearly all of these observers. However, Toomey describes the frequent occurrence of atypical or pseudo reactions consisting in most cases of a hard, glassy papule, rather deep-seated, which in some cases ruptured, resulting in a minute scar at the point of rupture. In other cases rupture did not occur. Two hundred and eighty-one such cases were recorded in a series of 2,852 primaries. He states further that 263 of the 281 atypical reactions showed reactions of immunity on revaccination.

Roberts (*19*), who employed the intracutaneous as well as the subcutaneous technique in the vaccination of 266 children ranging from 6 months to 5 years of age, states that very few scars were noted, although complete data in this regard are not reported. Approximately 40 percent of his cases were revaccinated at periods ranging from 1 month to 4 years following the initial vaccination, and immune reactions were obtained in the majority and a few accelerated takes. Vaccine obtained from the New York City Health Department was used in dilutions ranging from 1:2,000 to 1:20. He considers lack of scarring as one of the advantages of the intracutaneous technique.

Successful vaccination without a scar, through intracutaneous administration of artificially cultivated virus, is a possibility claimed

by Rivers (*20*). In a communication addressed to Dr. H. S. Diehl on November 28, 1934, in connection with material furnished him for use at the Students' Health Service of the University of Minnesota, after giving instructions as to technique, Rivers states: "The takes will appear 5 to 8 days after inoculation and will consist of red, indurated areas that will disappear *leaving no scar*. Reactions in immune individuals appear very promptly in 24 to 48 hours, and are characterized by redness and itching."

The case for virus prepared by the Goodpasture technique, in addition to the elimination of extraneous bacterial contaminants, rests upon the claims for a virus of uniform potency and attenuated virulence. The vaccination reactions are described as definitely milder than with calf vaccine, but without other important alteration in the clinical course of development of the lesions. The standard technique of vaccination is recommended. There have been no reports of its use by the intracutaneous technique.

During the physical examinations given routinely to students entering the University of Minnesota in the winter quarter of 1934 and the spring quarter of 1935, under the direction of one of our colleagues (*21*) vaccine prepared by the method of Rivers was used. For the winter quarter he reported 98 immune reactions and 5 primary takes. Of the latter he states that 3 were severe. In commenting on results in the spring quarter he states: "Of 11 who had no previous vaccination, 3 gave reactions interpreted as primary takes, and in these redness did not begin until the seventh to tenth day. Eight gave reactions interpreted as immune or questionable or unsatisfactory primary takes." No mention is made of vesicular or pustular reactions or scars.

In the fall of 1935 approximately 3,000 vaccinations were done with the vaccine prepared by Rivers and used according to his instructions. Since the procedure was not supposed to result in pustule formation and scar, the vaccinations were done on the flexor surface of the forearm, as Rivers recommended. The first of these inoculations was done on Monday, and by Saturday of the same week several cases returned to the Health Service presenting typical Jennerian reactions on the forearm. Numerous similar reactions were encountered by the following week. It appeared inevitable that these should result in scars; therefore, it seemed advisable to follow up as many of these cases as possible. Notices were sent within a few weeks following vaccination to these students for whom a primary take had been recorded. Responses included 60 boys and 30 girls. Scars were present in 43 (71.7 percent) of the former and 20 (66.6 percent) of the latter. The size of the scars ranged from 2 to 10 mm., with an average of 5 mm. Constitutional reactions were reported by approximately one-fifth of the group, necessitating bed rest in many

instances. All in all, there appeared nothing unusual about these reactions except for the site of inoculation. Had the inoculation been made upon the brachium the reaction would have been accepted as a commonplace.

The following year (1936) we decided to continue the use of Rivers' minced chick embryo vaccine in comparison with others, but to use the skin over the region of the deltoid insertion for all inoculations. During the fall quarter entrance examinations we employed 3 kinds of vaccine and 2 techniques. Calf virus and chick embryo virus (Goodpasture method) were administered by the multiple puncture technique. A drop of the virus was placed on the skin and exactly 3 punctures through it were made in all cases. Rivers' minced chick embryo vaccine and a diluted commercial chick embryo vaccine prepared by the Goodpasture method were employed intracutaneously. When judged by the percentage of primary takes on the previously unvaccinated group the chick embryo vaccine (Goodpasture method) appeared to be much less efficient than the calf lymph when both were administered by multiple puncture. Of 81 individuals vaccinated with the former, 13 (16 percent) developed primary takes, whereas 89 (36 percent) of 249 individuals developed primary takes with calf lymph. For this reason the use of chick embryo vaccine by the multiple puncture technique was not continued in 1937. Since the Rivers' minced chick embryo vaccine did not show any superiority to the diluted commercial chick embryo vaccine (Goodpasture method) when both were used intracutaneously, the former was discontinued in 1937 in favor of the latter, because of greater availability. Calf virus by the multiple puncture method and commercial chick embryo virus intracutaneously were therefore employed on alternate days during the entrance physical examinations of 1937.

Reactions were interpreted in the same manner in all cases without regard to method of vaccination or kind of vaccine used. Reactions were interpreted in accordance with the descriptions of Leake (*22*): "With a reaction of immunity which indicates full protection against smallpox at the time of vaccination, the broadest redness is reached and passed in 8 to 72 hours after vaccination. This redness is accompanied by a slight elevation of the skin, which can be felt by passing the little finger lightly over the vaccinated area, and often there is slight itching at the site. With the *accelerated* or modified vaccination, called vaccinoid, which indicates partial immunity, previously, the broadest redness is reached and passed in 3 to 7 days after vaccination. With a typical primary vaccinia, indicating absence of immunity to smallpox prior to this vaccination, the zone of redness, or areola, rather narrow from the third to the seventh day, begins a sudden spread about 7 days after vaccination and reaches its broadest

diameter in 8 to 14 days after vaccination, disintegrating and disappearing soon thereafter. * * * Definite vesicles are formed in vaccinia reactions, and usually in vaccinoids (accelerated reactions) but not usually with the reaction of immunity." Rivers also states, in directions for interpretation of reactions to his vaccine, that reactions appear in immune individuals in 24 to 48 hours and a primary take in 5 to 8 days.

Since it was not feasible in dealing with thousands of students to make daily observations, it seemed to us that two readings should suffice for the application of the above-mentioned criteria. We therefore made the first reading at 48 hours to observe the immune reactions. A second reading was done at 7 days. If a reaction which had been recorded as immune had developed vesiculation in the interval, it was interpreted as an accelerated reaction. If a reaction of redness with edema and induration was present at 7 days, when none had been present at 48 hours, it was interpreted as a primary take whether vesiculation had occurred or not. According to Rivers, no vesiculation should occur with his vaccine used intracutaneously; and although Leake specifies vesiculation for the primary take, this is not universally present by the seventh day. A follow-up during the next few weeks of those reactions recorded as primary take was also done for the purpose of discovering the presence or absence and the size of the scar.

VACCINATION HISTORY

Since vaccinations are done routinely on all new students, it would be impossible to compare results of the various vaccines used without a history in relation to previous vaccinations or smallpox in each case. The numbers of new students for the years 1936 and 1937 were 3,467 and 3,599, respectively. Of the former 83.6 percent and of the latter 83.2 percent presented scars of previous successful vaccinations. (See table 1.) The lapse of time since the previous vaccination should also be known. However, after interviewing several hundred students in this regard, it was evident that reliable histories as to time could not be obtained from the majority of them. Minnesota has no law requiring vaccination for school attendance, and it is probable that vaccination is stimulated largely through the occurrence of epidemics of smallpox. Since the last extensive epidemic occurred in 1924–25, it is probable that a major portion of the vaccinations for these groups of students was done either at or before that time. This would give a probable interval of 12 or more years since the previous successful vaccination for the majority of the vaccinated students.

TABLE 1.—*Vaccination history*

	1936		1937	
	Number	Percent	Number	Percent
Previous successful vaccination	2,899	83.6	2,996	83.2
No previous successful vaccination	538	15.5	561	15.6
Doubtful or unknown	30	.9	42	1.2
Total	3,467		3,599	

EFFICIENCY OF THE VACCINE

For the previously successfully vaccinated group, when judged by the percentage of primary takes obtained, calf lymph was twice as efficient as the commercial chick vaccine (Goodpasture) used intracutaneously, and approximately eight times as efficient when both were used by the multiple puncture method. The Rivers' chick vaccine was least efficient of all. (See table 2.)

TABLE 2.—*Results of vaccinations on previously successfully vaccinated group* [1]

[I=Intracutaneously. P=By multiple puncture]

Vaccine	Number of cases	Failures		Total reacting, number	Type of reaction					
		Number	Percent		"Immune"		Accelerated		Primary	
					Number	Percent	Number	Percent	Number	Percent
Rivers' chick (I)	473	2	0.4	471	461	97.8	9	2.0	1	0.2
Commercial chick (I)	2,209	209	9.5	2,000	1,940	97.0	30	1.5	30	1.5
Commercial chick (P)	457	11	2.4	446	435	97.5	9	2.0	2	.4
Calf (P)	2,756	206	7.5	2,550	2,367	92.8	102	4.0	81	3.2

[1] Combined figures for the school years 1936-37 and 1937-38.

For the unvaccinated group the Goodpasture chick vaccine (intracutaneous) was greatest in efficiency in 1936, with 44 percent of primary takes. Calf lymph and the Goodpasture chick vaccine by the multiple puncture method gave 36 percent and 16 percent, respectively, for the same year, while the Rivers' chick vaccine produced only 10 percent of primary takes. However, in 1937 the calf vaccine gave 28 percent of primary takes as against 18.5 percent for the Goodpasture chick vaccine intracutaneously. When the results of the 2 years are combined, calf lymph by multiple puncture appears slightly superior to the commercial chick vaccine (Goodpasture method), used intracutaneously, and much superior when the latter vaccine is used by the multiple puncture (table 3).

According to the criteria of Leake (*22*), the potency of these vaccines is low. He states that a vaccine of full potency should give more than 50 percent of vaccinoid reactions in unselected persons who have been

vaccinated or who have had smallpox 10 to 40 years previously, and immune reactions or typical vaccinias in the remainder. He also states: "A more prolonged test that the vaccine is of full potency is that when properly applied it gives 100 percent of vaccinias (typical primary successes) in every application on at least 100 previously unvaccinated individuals." When viewed also in this light, the vaccines used were low in potency.

TABLE 3.—*Results of vaccination on group not previously successfully vaccinated*[1]

[I=Intracutaneously. P=By multiple puncture]

Vaccine	Number of cases	Failures		Total reacting, number	Type of reaction					
		Number	Percent		"Immune"		Accelerated		Primary	
					Number	Percent	Number	Percent	Number	Percent
Rivers' chick (I)	80	13	16.2	67	57	85.1	2	3.0	8	11.9
Commercial chick (I)	457	110	24.1	347	224	64.5	6	1.7	117	33.7
Commercial chick (P)	81	24	29.6	57	44	77.2	0	------	13	22.8
Calf (P)	481	88	18.3	393	226	57.5	13	3.3	154	39.2

[1] Combined figures for the school years 1936-37 and 1937-38.

Goodpasture and Buddingh (*4*), in 1935, reported that of 35 non-immune persons vaccinated with a dermal strain cultivated by the method of Goodpasture, 29 developed typical vaccinal reactions, 3 accelerated reactions, 1 was negative, and for 2 more complete information is omitted. This is a much higher efficiency than we have obtained from vaccine of the same type from a commercial source.

VACCINATION SCARS

There can be little doubt that the large, unsightly scars formerly so common were responsible in part for a certain amount of prejudice against vaccination, particularly among women. The vaccine and technique of vaccination producing the least amount of unsightly scars, other things being equal, would appear to be most desirable. A comparison of results of scars in this study, however, gives little weight to the choice of either vaccine or method, for the scars in no case were large. The largest scar recorded was 13 mm., and resulted from the intracutaneous injection of (Goodpasture) chick vaccine. The average scar by this method was 5.6 mm. In the case of the Rivers' chick vaccine, which was supposed to produce immunity without scar, the average scar was 5.1 mm., but scars up to 10 mm. resulted in some cases. Since scarring was not anticipated with the Rivers' vaccine it was surprising to find on follow-up of 95 cases recorded as primary takes only 29 (30 percent) without scars and 66 (70 percent) with scars. It was more surprising, however, to find a similar result with the other types of vaccines. With calf lymph

virus, out of 166 cases recorded as primary takes for 1936 and 1937, 35 cases, or 21 percent, presented no scars at follow-up. With diluted commercial chick vaccine given intracutaneously in 102 cases recorded as primary takes, 18 cases, or 18 percent, had no scars. (See table 4.) It will be recalled that primary takes were recorded for cases presenting a reaction of redness and edema on the seventh day when no reaction had been observed at the 48-hour reading.

TABLE 4.—*Vaccination scars in primary takes* [1]

[I=Intracutaneously. P=By multiple puncture]

Vaccine	Cases recorded	Cases examined		Size in mm.										Minimum	Maximum	Average
				None		2-4		5-7		8-10		11-13				
		Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent			
Rivers' chick (I)	(?)[2]	95	----	29	31	17	18	43	45	6	6	0	----	3	10	5.1
Commercial chick (I)	147	101	70	18	18	24	24	46	45	11	11	2	2.0	2	13	5.6
Commercial chick (P)	15	9	60	1	11	4	44	3	33	1	11	0	----	2	9	4.8
Calf (P)	235	166	70	35	21	40	24	58	35	32	19	1	.6	2	11	5.8

[1] Combined figures for the school years 1936-37 and 1937-38, also Rivers' vaccine, 1935-36.
[2] The records for 1935 were not preserved.

SEVERITY OF REACTION IN PRIMARY TAKES

In carrying out routine vaccination procedures it is certainly desirable that no more disability result than is consistent with the development of immunity. In the self-supporting student a number of days of work may be lost through enforced disuse of the arm at the height of the local reaction. In addition, absence from classes may also result from the necessity of bed rest in the case of severe constitutional reactions.

Since it was not feasible to make daily observations, no attempt was made to classify local reactions on the basis of appearance during the course of vaccinia. A classification of local reactions on a subjective basis, as reported by the student, appears in table 5.

TABLE 5.—*Local reaction to primary take* [1]

[I=Intracutaneously. P=By multiple puncture]

Vaccine	Cases	Mild		Moderate		Severe		None	
		Number	Percent	Number	Percent	Number	Percent	Number	Percent
Rivers' chick (I)	95	20	21	24	25	16	17	35	37
Commercial chick (I)	101	35	35	24	23	5	5	37	37
Commercial chick (P)	9	5	50	2	25	0	----	2	25
Calf (P)	166	63	38	37	22	10	6	56	34

[1] Combined figures for the school years 1936-37 and 1937-38, also Rivers' vaccine, 1935-36.

Severe reactions include only those who stated that complete disuse of the arm for purposes of work resulted. It will be seen that from this standpoint the greatest amount of disability resulted from the Rivers' chick vaccine, amounting to 17 percent of the primary takes. The location of the vaccinations in 90 of these cases in 1935 was the flexor surface of the forearm, and may have influenced the results. The number of primary takes with this vaccine in 1936 was too small to be significant (5 cases); however, none reported disability. The site of vaccinia in these 5 cases was the brachium at the deltoid insertion. It will be noted that the amount of disability from local reactions for the calf vaccine was not greatly different by multiple puncture and the commercial chick vaccine (intracutaneously).

Although the group is small (8 cases), the local reaction with the commercial chick vaccine (Goodpasture) by multiple puncture was mild in all cases. Our impression thus confirms the statement of Goodpasture in this regard, who reported comparative results with chick and calf virus. He states that the appearance of the areola in the lesions from the calf vaccine was much more fiery and the induration of the surrounding tissues was much more extensive and deep-seated than they were in the cases given the chick vaccine. There was a marked difference in the amount of glandular involvement. Little or no enlargement of the axillary lymph nodes was noted in those vaccinated with the chick vaccine as compared with those vaccinated with the calf vaccine. It is our impression, however, that the glandular involvement is proportional to the size of the local lesion. The same lot of commercial chick vaccine in 1936 was used both intracutaneously and by multiple puncture. Those done with the intracutaneous technique resulted in lesions comparable to those done with calf vaccine, and approximately the same percentage of cases reported severe local reactions. All these cases seen presented axillary gland involvement, whereas those done with the chick vaccine by multiple puncture had little or no glandular enlargement. The adenitis, in fact, appears to be the most important factor where enforced disuse of the arm is concerned.

THE CONSTITUTIONAL REACTION

Constitutional reactions, characterized by symptoms more or less common to all microbic invasions, were occasionally encountered. These included fever, malaise, headache, backache, generalized muscular aching and soreness, and occasionally nausea and vomiting. All cases of primary take with evidence of the local lesion were questioned as to symptoms, and as to whether the reaction was of such severity as to necessitate bed rest. Table 6 is a tabulation of these findings, as reported by the students. Since loss of time from work or

school is the most important consideration, discussion is here limited to those who found it necessary to remain in bed. In a comparison of the vaccines from this standpoint it is noted that the Rivers' chick

TABLE 6.—*Constitutional reaction to primary take* [1]

[I=Intracutaneously. P=By multiple puncture]

Vaccine	Cases	Fever		Toxic		Bed		None	
		Number	Percent	Number	Percent	Number	Percent	Number	Percent
Rivers' chick (I)	95	22	23	7	7	15	16	73	77
Commercial chick (I)	101	11	11	17	17	6	6	84	83
Commercial chick (P)	9	2	25	0		0		7	75
Calf (P)	166	22	13	30	18	13	8	136	82

[1] Combined figures for the school years 1936-37 and 1937-38, also Rivers' vaccine, 1935-36.

vaccine in 1935 produced the greatest amount of disability. Calf vaccine in 1936 produced reactions confining every tenth case to bed, while in 1937 only about half as many were so affected. Of the cases vaccinated with commercial chick vaccine intradermally the disability was about half as great as with calf vaccine in 1936, and about the same for 1937. By multiple puncture with the commercial chick vaccine in the small number observed no disability resulted.

IMMUNE REACTIONS IN THE NONIMMUNE GROUP

A puzzling feature observed in this study was a high incidence of immune reactions in the group of individuals presenting no scar as evidence of previous vaccination and without a history of smallpox. The immune reaction in this group of individuals was encountered with all types of vaccines used and with both the intracutaneous and multiple puncture technique. The highest incidence of the immune reaction in the supposedly nonimmune group, amounting to 85 percent, was obtained with Rivers' chick vaccine; the lowest with the calf vaccine, amounting to 57 percent of cases. (See table 3.)

That this result was not due entirely to error in history or in reading the reaction is shown by follow-up of a considerable number of these cases in both 1936 and 1937. Of the 551 cases of supposedly nonimmunes for which "immune reactions" were recorded, we were able to interview 270 (table 7). Of these, 50 presented scars of previous vaccinations missed at the first examination. Fifty-three others gave a history of having had smallpox. Two cases interpreted as immune reactions at the first examination terminated as primary takes. After correction of these errors there were 165 persons, amounting to 61.1 percent of those re-examined, for whom "immune reactions" were recorded, but without any satisfactory basis for an acquired immunity.

TABLE 7.—*Follow-up of "immune reactions" in supposedly nonimmunes* [1]

	Number	Percent
Total number of cases recorded	551	
Cases re-examined	270	100.0
Previous successful vaccination	50	18.5
History of smallpox	53	19.6
Primary take at entrance (recorded immune)	2	.7
No successful vaccination; no history of smallpox	165	61.1

[1] Combined figures for the school years 1936-37 and 1937-38.

One hundred and fifty-two of these cases were revaccinated, all with calf lymph vaccine by the multiple puncture method with 25 to 30 punctures in each case. Thirteen cases refused revaccination. In table 8 is shown the type of vaccine which produced the "immune reaction" at the first vaccination and the results of revaccination with calf lymph vaccine. Of 14 cases recorded as immune to Rivers chick vaccine, 10, or 71 percent, gave "immune reactions" to calf lymph, while 4 gave no reaction. Of 41 cases giving "immune reactions" to the commercial chick vaccine intracutaneously, 26, or 63 percent, gave "immune reactions" to the calf vaccine also. Of 42 cases giving "immune reactions" to calf vaccine at entrance, 31 cases, or 74 percent, gave "immune reactions" at revaccination with the same kind of vaccine but from a different lot.

TABLE 8.—*Results of revaccination with calf lymph of nonimmune group giving immune reactions on entrance*

[I=Intracutaneously. P=By multiple puncture]

Reaction on revaccination	Vaccine used at entrance								Total	
	Rivers' chick (I)		Commercial chick (I)		Commercial chick (P)		Calf (P)			
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Immune	10	71	26	63	4	100	31	74	71	70
Accelerated	0	-------	3	7	-------	-------	2	5	5	5
Primary	0	-------	8	20	-------	-------	7	16	15	15
None	4	29	4	10	-------	-------	2	5	10	10
Total read	14	100	41	100	4	100	42	100	101	100

The groups vaccinated with the commercial chick vaccine and calf lymph are greatest in numbers. No marked difference was noted in the results obtained on revaccination in these two groups. Sixty-three percent of the former and 74 percent of the latter, none of whom had a history of smallpox, nor evidence of a typical Jennerian reaction, again presented "immune reactions."

Few of these individuals would consent to further attempts to produce typical vaccinia. The average number of trials per case at

this time was 4.6. Repeated vaccinations were done for a few cases, from 2 to 10 times, with immune reactions in all cases.

This illustrates the fact that immunity can be judged from the type of reaction only if vaccine of full potency is used (*22*). Susceptibility to vaccinia and to smallpox is generally believed to be universal. There is no history of smallpox in any case, and a history of chickenpox is by no means universally present. There is no evidence that a typical Jennerian reaction has ever been obtained in any of these individuals, some of whom state that they have attempted to obtain this result as many as 15 times.

In answer to the question, "Does a red, slightly raised area observed at the site of vaccination 48 hours after vaccination is performed necessarily indicate that the person vaccinated was immune to smallpox?", Leake (*22*) states that there are three other possibilities:

(1) The most frequent, he states, is that the vaccine used was so reduced in potency that, although able to give the reaction described above, it did not go on to the production of a more marked reaction as would a vaccine of full strength if used on the same person. It is obvious that this explanation does not explain the immediate reaction in a nonimmunized person. The early reaction presumably indicates a previous sensitization to the virus of smallpox, or to the cultivated vaccine virus. Leake further states (*22*) that the fact that reactions otherwise indistinguishable from reactions of immunity may be given by heated vaccine indicates that at least part of the visible phenomenon which we call the reaction of immunity is due to this inert material and to that extent may be called a reaction of sensitivity.

(2) A second possibility involves the accelerated or vaccinoid reaction, in that an early reaction interpreted as an immune reaction at 48 hours may present a maximum of reaction at 7 days characteristic of the accelerated reaction. Even this, Leake states, indicates some immunity, however. He also states (*22*) that, "Even true vaccinias not infrequently show an early reaction, especially if there have been previous unsuccessful attempts at vaccination." Gins and Hackenthal (*24*) found the vaccine virus on the tonsils of children 3 to 5 days after cutaneous vaccination and in various organs of animals 2 days after such vaccination. This would indicate distribution of virus through the body, whatever the method of vaccination.

(3) A third alternative to immunity as a possibility of error is that of reaction to trauma. Leake states that early reactions are more clearly apparent with the multiple pressure method than with other noninjection methods, on account of the absence of injury to the true skin and the consequent absence of an obscuring, traumatic reaction. To obviate falsely reading such reaction as a reaction of immunity he states further that it is necessary to treat another site as a control, with exactly the same degree of trauma but without applying the

vaccine, or preferably to use the method of multiple puncture which leaves no traumatic reaction after 6 hours to obscure faint reactions of immunity. If this be true, then it appears obvious from table 3 that the immediate reactions observed in this supposedly nonimmune group cannot be explained on the basis of trauma, for a greater proportion of these resulted from the use of the multiple puncture method with chick vaccine than from the chick vaccine intracutaneously. The former gave 77 percent, the latter 64 percent, and the calf lymph, 57 percent. Furthermore, the proportions reported as negative reactions to the calf vaccine by multiple puncture and to the chick vaccine intracutaneously were not greatly different, being 18 percent and 24 percent, respectively.

SUMMARY

(1) The results of the vaccination against smallpox of 9,086 students at the University of Minnesota during the years 1936–38 are reported.

(2) Eighty-three percent of students entering the University of Minnesota have been successfully vaccinated prior to admission.

(3) Three kinds of vaccine were used, two cultured artificially by the methods of Rivers and Goodpasture, respectively, and one prepared by the usual calf method. The Rivers' vaccine was employed intracutaneously. The vaccine by the Goodpasture methods was employed both intracutaneously and by the multipuncture technique. The calf vaccine was used only by multipressure technique.

(4) The calf lymph virus with multiple puncture technique gave a higher percentage of primary takes than the two types of chick virus when either the multiple puncture or intracutaneous method of administration was used. However, the results with the Goodpasture chick vaccine when used intracutaneously were not grossly inferior to those obtained with calf lymph.

(5) Severe local and constitutional reactions following vaccination were slightly less frequent with the chick vaccine (Goodpasture) than with the calf lymph vaccine. Severe local and constitutional reactions were reported more frequently with Rivers' vaccine given intracutaneously than with any other type of vaccine.

(6) There was little difference in the size of the scar produced in the primary takes with the various kinds of vaccine or methods of vaccination.

(7) Immune reactions were encountered in over 61 percent of individuals who had never been vaccinated successfully and who had never had smallpox. A majority of these individuals had been vaccinated many times.

REFERENCES

(1) Steinhardt, Israeli, and Lambert: J. Inf. Dis., **13**: 294 (1913).

(2) Carrel, A., and Rivers, T. M.: Compte Rend. Soc. Biol., **96**: 848 (April 1927).

(3) Rivers, T. M.: J. Exp. Med., **54**: 453 (October 1931).

(4) Goodpasture, Ernest W., and Buddingh, John: Am. J. Hyg., 21: 319 (March 1935).
(5) Chauveau, M.: Bull. de l'Acad. méd., 31: 1111 (1865–66); Rev. mensuelle de Méd. et de Chir., p. 240, 1877.
(6) Nobl, G.: Wien. klin. Wchnschr., 19: 975 (1906).
(7) Knoepfelmacher, W.: Wien. Med. Wchnschr., 46: 2198 (1906); 79:247 (1929); Ztchr. f. Exp. Path. u. Therap., 4: 880 (1907).
(8) Goodal, J. R.: Am. J. Med. Sci., 158: 721 (November 1919).
(9) Spearman, F. S.: Colorado Med., 19: 13 (1922).
(10) Kuhle: Monatschr. f. Kinderh., 30: 390 (1925).
(11) Janson, C.: Hygiea (Stockholm), 1: 683 (1888).
(12) Tedeschi, V.: Trieste Tr. d. Soc., d. tiographi, 1901. Abstr. Zentralbl. f. Bakteriol., 31: 349 (1902).
(13) Leiner, C., and Kundratitz, K.: Ztschr. f. Kinderh., 30: 205 (1921).
(14) Czapski: Münch. Med. Wchnschr., 71: 284 (1924).
(15) Wright, L. T.: J. Am. Med. Assoc., 71: 654 (Aug. 24, 1918).
(16) Twyman, T.: J. Missouri Med. Assoc., 19: 353 (August 1922).
(17) Berney, D. E.: Atl. Med. J., 27: 713 (August 1924).
(18) Toomey, J. A., and Hauver, R. B.: Am. J. Dis. Child., 35: 186 (1928).
(19) Roberts, B. E.: J. Prev. Med., 6: 453 (November 1932).
(20) Rivers, T. M., and Ward, S. M.: J. Exp. Med., 62: 549 (1935).
(21) Radl, R. B.: Unpublished.
(22) Leake, J. P.: Questions and answers on smallpox vaccination. Reprint No. 1137 from the Public Health Reports, Washington: Government Printing Office, 1937.
(23) Gins, H. A., Hackenthal, H., and Kamentzawa, N.: Centralbl. f. Bakteriol. (Abt. I, Hft. 6–8, Beihft.) 110: 115 (1929).

INFLUENCE OF CASTRATION ON THE INDUCTION OF SUBCUTANEOUS TUMORS IN MICE OF THE C_3H STRAIN BY 1:2:5:6-DIBENZANTHRACENE

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Contradictory findings have been reported regarding the influence of the sex glands and sex hormones on the development of tumors in animals treated with tar or with the carcinogenic hydrocarbons.

Boyland and Warren (*1*) found no significant difference in the percentage of tumors between normal and castrated or between male and female mice of either the CBA or the Simpson albino strain, injected subcutaneously either once or twice with 0.25 cc. of lard containing 1 mg. of methylcholanthrene.

Tavares and Morais (*2*) painted the ears of 25 rabbits with tar twice weekly for 7 months, starting 7 to 8 days after double ovariectomy, and similarly painted 25 normal rabbits. They found that castration inhibited the appearance and evolution of tar tumors, but its action was not sufficiently intense to outweigh the individual susceptibilities of all rabbits, and tumor nodules were not constantly more numerous or larger in the normal rabbits.

Moore and Melchionna (*3*) injected 3:4 benzpyrene (5 percent in lard) into the prostates of white rats, of which 20 were castrated at the time of the injection. The earliest tumor in an intact rat was observed on the one hundred and seventeenth day, and in a castrated rat on the eighty-third day. Six of the intact series were castrated after the establishment by biopsy of the presence of a tumor; 35 days

later these rats were killed and the tumor showed no difference in structure from the biopsy specimen. Of 20 castrated rats 13 developed carcinoma of the prostate and 1 sarcoma of the prostate; of 18 intact rats, 13 developed carcinoma of the prostate and 1 sarcoma of the prostate. Four castrated rats received injections of 3:4 benzpyrene, and after the eighty-sixth day daily injections of "male sex hormone" (Oreton, Schering). All showed well-developed carcinomas and three of them sarcomas also. This high incidence of sarcoma, in comparison with the data previously mentioned, suggests the need for further experiments. Schockaert found that oestradiol (20 international units of the benzoate in oil subcutaneously weekly) caused tar cancer to develop more rapidly in 150 mice than in 150 controls receiving oil only, 25 percent of cancers as against 4 percent after 4 months, and 65 percent as against 27 percent after 5½ months.

Gilmour applied 3:4-benzpyrene (0.3 percent in benzene) 20 times to the interscapular skin of mice of mixed strain; in one-half of these oestrone (0.01 percent) also was applied to the rump, both paintings taking place twice weekly until the end of the experiment. Some of the results showed that oestrone enhanced the carcinogenic action of the hydrocarbon both as to time of induction and number of tumors. In the oestrone-treated animals (series A), the first papilloma appeared 4 weeks earlier than in those painted with benzpyrene alone (series B), and 14 mice of series A bore papillomas when the first ones appeared in series B; the first malignant tumor appeared in series A after 8 weeks and in series B after 14 weeks, when there were already 7 malignant tumors in series A. Thus the action of oestrone enhanced the carcinogenic response of the skin to benzpyrene both as to time of induction and number of tumors. Flaks and Ber (*4*) applied methylcholanthrene in benzol to the skin of mice 3 times a week and gave half the mice injections of "Sterandryl" (testosterone propionate) starting a week before the first application of methylcholanthrene. In the first experiment, with 118 mice, Sterandryl somewhat delayed but did not prevent the appearance of tumors. In the second experiment the dosage of Sterandryl was increased and the effect was more pronounced, though only 20 mice were used; after 150 days 9 out of 10 control mice had tumors, of which 4 were carcinomas, while only 3 out of 9 mice injected with the hormone had very small warts. Perry and Ginzton observed a wide variety of neoplastic lesions in many different locations in skin and viscera of female mice following painting with a solution of 1:2:5:6-dibenzanthracene combined with theelin (oestrone).

Thus Boyland and Warren demonstrated no significant difference in the development of induced tumors as a result of castration. This is in contrast to Tavares and Morais (*2*) as well as Moore and Melchionna (*3*), who did find a difference although of questionable sig-

nificance. The injection of sex hormones was found to exert an influence by making the animals more susceptible to induced tumors by Schockaert (*5*) and Gilmour (*6*), Moore and Melchionna (*3*), and Perry and Ginzton (*7*). However, Flaks and Ber (*4*) reported that similar treatment made the animals more resistant.

These conflicting observations are based upon experiments employing animals of several species, treated with different carcinogenic compounds in varying dosage applied to different areas and tissues of the body. Failure to maintain uniform conditions as to compound, dosage, site and method of administration, age at castration, and genetic constitution of the test animal may partly account for these discrepancies. With these points in mind the present study was undertaken to examine the effects of castration in early life on the development of tumors induced in pure strain mice of both sexes injected with a constant amount of 1:2:5:6-dibenzanthracene.

MATERIALS AND METHODS

The mice employed in this study were of the inbred C_3H strain obtained from the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine. The mice were maintained on a diet of dog chow exclusively except for the first several days after being received at this laboratory, during which time their diet was supplemented with bread and milk. Water was allowed freely at all times. One hundred and twenty-one mice between the ages of 6 and 8 weeks were injected subcutaneously into the right chest wall, in the axillary region, with 0.2 cc. of a filtered lard solution containing 0.8 mg. of 1:2:5:6-dibenzanthracene. The 1:2:5:6-dibenzanthracene was obtained from the Eastman Kodak Co. and was used without further purification. Forty-nine male and thirty-three female mice were castrated a few hours to 2 weeks prior to the subcutaneous injection of lard solution of 1:2:5:6-dibenzanthracene. The remaining mice, consisting of 21 males and 18 females, were set aside as intact controls following the injection.

The operation of castration was performed in the male by delivering each testicle through a lateral scrotal incision and cutting through the spermatic cord. The stump of the cord was dropped back through the incision into the scrotum. Usually there was no indication for employing ligatures or for suturing the incision. In the case of the female each ovary was delivered through a single midline abdominal incision and a cut made through the terminal horn of the uterus. Thus each ovary together with the corresponding fallopian tube and adjacent segment of the uterine horn was removed. The abdominal incision was approximated with interrupted linen sutures. The animals were examined weekly by palpation and the time of the

appearance of the tumor was charted. Most of the lesions, including all the doubtful ones, were examined microscopically.

Time of appearance of induced tumors in castrated and intact male and female mice of C_3H strain following subcutaneous injection of 0.8 mg. of 1:2:5:6-dibenzanthracene dissolved in 0.2 cc. of lard

Time in weeks after inoculation	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	33	Total number with tumor	Average time in weeks	Number dying without tumor	Number killed without tumor
Sex, and number of mice	Number of tumors																					
Male:																						
49 castrates	2	2	2	1	1	1	6	6	6	3	3	2	1	2	0	1	---	---	39	17.1	8	2
21 controls	1	0	0	0	1	0	2	4	2	1	0	3	1	0	2	0	1	---	18	18.8	2	1
Female:																						
33 castrates	0	0	1	0	0	5	1	1	3	3	0	3	6	3	3	1	---	---	30	19.7	3	----
18 controls	0	0	0	0	0	0	0	1	2	3	4	4	1	0	0	0	1	1	17	20.9	1	----

EXPERIMENTAL OBSERVATIONS

The results of the experiment are presented in the accompanying table. During the course of the experiment the following animals died without tumor: Eight male castrates at 4, 7, 9, 9, 9, 10, 12, and 16 weeks after inoculation, respectively; three female castrates at 1, 2, and 6 weeks after inoculation, respectively; two male controls at 13 and 14 weeks after inoculation, respectively; and one female control at 16 weeks after inoculation. All these animals died without developing frank tumor. At the site of inoculation of the hydrocarbon in these mice there were frequently hyperplasia of the nearby tissue cells and chronic inflammatory changes. The causes of death were thymic tumor, lymphoma, abscess of abdominal wall, retroperitoneal abscess, chronic bronchitis, and pneumonia. Three other mice, two male castrates and one male control, did not develop tumors and were killed at the end of 36, 42, and 41 weeks, respectively. In two of these animals careful search of the axillary region failed to reveal the presence of the injected lard.

As set forth in the table, in the castrated mice the tumors arose in the males at the average time of 17.1 weeks following inoculation of the lard solution of 1:2:5:6-dibenzanthracene, and in the females at the average time of 19.7 weeks. Tumors appeared in the control mice at an average time of 18.8 weeks in the males and 20.9 weeks in the females. These results in the control mice approximate those reported by Andervont (*8*) in which strain C_3H mice were used for injections of a lard solution of the same amount of 1:2:5:6-dibenzanthracene. It is of interest to record that Andervont's mice were all bred in this laboratory while the animals recorded herein were obtained from the Roscoe B. Jackson Memorial Laboratory. These sublines of the strains have been separate for approximately 8 years.

The earliest tumors came up at 10 weeks following inoculation, one each in a male control and in a male castrate mouse; in the case of the females the earliest tumor appeared in a castrate at the twelfth week following inoculation and in a control at the seventeenth week. Comparing the two groups of tumor-bearing castrates and controls, the growths arose in the male castrate mice 1.7 weeks earlier on the average than in the male controls and similarly 1.2 weeks earlier in the female castrates than in the female controls. These differences are slightly less than those between the average time of tumor development in the males (2.6 weeks) on the one hand and the females (2.1 weeks) on the other hand in the control and castrate groups, respectively. In neither case are the differences in the average time at which tumors appeared of sufficient magnitude to regard them of particular importance, although it is interesting that the slight differences between the castrates and controls observed with males and females are in the same direction.

It is worthy of note, however, that at the end of 16 weeks there were 7 tumors in the female castrates and none in the female controls; in the same length of time there were 15 tumors in the male castrates as compared with 4 in the controls of the same sex. Based upon the number of animals alive in each group at the time of appearance of the first tumor, this would mean that 38 percent of the male castrates and 23 percent of the female castrates had developed tumors as compared with 21 percent and 0 percent, respectively, of the intact controls.

The results of this experiment are comparable to those of Boyland and Warren who also used highly inbred mice. But these authors also used a relatively large amount of a more powerful carcinogen (2 mg. methylcholanthrene). Whether the differences might have been brought out more strikingly by employing a weaker dose of the carcinogen it is impossible to state at present.

CONCLUSION

Castration decreased slightly, but not apparently to a significant extent, the average time of development of tumors in male and female mice of the C_3H strain injected subcutaneously into the right axillary region with 0.8 mg. of 1:2:5:6-dibenzanthracene in 0.2 cc. of lard. However, up to the end of the sixteenth week a higher percentage of tumors appeared in the castrates of each sex.

REFERENCES

(*1*) Boyland, E., and Warren, F. L.: Induction of tumours by methylcholanthrene in 2 strains of mice. J. Path. and Bact., **45**:171 (1937).

(*2*) Tavares, A., and Morais, E.: L'influence de la castration ovarienne sur le développement et la croissance des tumeurs du goudron. Compt. rend. Soc. de biol., **125**:179 (1937).

(3) Moore, R. A., and Melchionna, R. H.: Production of tumors of prostate of white rat with 1:2-benzpyrene. Am. J. Cancer, 30:731 (1937).
(4) Flaks, J., and Ber, A.: Action anticancérigène de l'hormone male. Compt. rend. Soc. de biol., 128:506 (1938).
(5) Schockaert, J. A.: Grossesse, cancer et folliculine. Bruxelles-mèd., 15:1010 (1935).
(6) Gilmour, M. D.: Investigation into influence of oestrone on growth and genesis of malignant cells. J. Path. and Bact., 45:179 (1937).
(7) Perry, I. H. and Ginzton, L. L.: Development of tumors in female mice treated with 1:2:5:6-dibenzanthracene and theelin. Am. J. Cancer, 29:680 (1937).
(8) Andervont, H. B.: The incidence of induced subcutaneous and pulmonary tumors and spontaneous mammary tumors in hybrid mice. Pub. Health Rep., 53:1665 (1938). See also table III *in*—
Fieser, L. F.: Carcinogenic activity, structure, and chemical reactivity of polynuclear aromatic hydrocarbons. Am. J. Cancer, 34: 37 (1938).

DEATHS DURING WEEK ENDED MAY 20, 1939

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended May 20, 1939	Corresponding week, 1938
Data from 88 large cities of the United States:		
Total deaths	8,009	[1] 8,174
Average for 3 prior years	[2] 8,283	
Total deaths, first 20 weeks of year	183,380	175,188
Deaths under 1 year of age	462	[1] 529
Average for 3 prior years	[2] 541	
Deaths under 1 year of age, first 20 weeks of year	10,713	10,823
Data from industrial insurance companies:		
Policies in force	67,365,626	68,326,308
Number of death claims	15,291	12,459
Death claims per 1,000 policies in force, annual rate	11.8	9.5
Death claims per 1,000 policies, first 20 weeks of year, annual rate	11.7	9.9

[1] Data for 87 cities.
[2] Data for 86 cities.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

Cases of certain diseases reported by telegraph by State health officers for the week ended May 27, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (----) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

Division and State	Diphtheria				Influenza				Measles			
	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median
NEW ENG.												
Maine	0	0	0	1	----	----	----	2	640	106	156	156
New Hampshire	0	0	0	0	----	----	----	----	0	0	3	12
Vermont	0	0	0	0	----	----	----	----	2,252	168	140	49
Massachusetts	2	2	3	7	----	----	----	----	1,109	943	392	697
Rhode Island	0	0	0	1	----	----	----	----	1,015	133	----	59
Connecticut	3	1	7	2	3	1	1	1	2,428	818	71	181
MID. ATL.												
New York [2]	8	21	22	29	[1]6	[1]8	[1]2	[1]5	873	2,181	3,445	2,904
New Jersey	8	7	10	10	6	5	9	6	57	48	708	708
Pennsylvania	12	23	35	36	----	----	----	----	72	141	2,216	2,216
E. NO. CEN.												
Ohio	8	11	5	9	----	----	----	6	52	67	1,362	1,241
Indiana	12	8	5	12	4	3	----	15	19	13	159	270
Illinois	24	36	36	36	35	54	6	10	29	44	1,089	1,089
Michigan [2]	17	16	1	9	6	6	2	2	705	667	3,080	375
Wisconsin	0	0	1	4	62	35	22	19	1,380	785	2,511	1,694
W. NO. CEN.												
Minnesota	0	0	1	1	10	5	1	1	492	254	359	359
Iowa [2]	6	3	2	2	8	4	----	1	419	207	308	231
Missouri	12	9	7	16	----	----	13	25	28	22	192	192
North Dakota	7	1	2	1	15	2	5	----	796	109	76	32
South Dakota	15	2	0	1	60	8	----	----	1,578	210	----	4
Nebraska	8	2	1	1	11	3	1	----	1,351	354	294	185
Kansas	14	5	2	3	----	----	5	1	271	97	401	401

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended May 27, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median
SO. ATL.												
Delaware	0	0	1	0					256	13	8	12
Maryland [2 3]	9	3	5	7	9	3	1	5	509	165	84	216
Dist. of Col.	24	3	11	11					2,555	316	19	66
Virginia [3]	6	3	11	10	49	26			888	474	325	465
West Virginia	24	9	3	5	46	17	28	28	19	7	504	187
North Carolina	13	9	6	9	4	3	3	4	1,045	715	1,402	298
South Carolina [4]	5	2	9	4	666	244	59	104	33	12	118	68
Georgia [4]	13	8	1	5	90	54			120	72	294	26
Florida	18	6	7	4	81	27	3	1			110	39
E. SO. CEN.												
Kentucky	16	9	7	7	16	9	4	9	61	35	148	268
Tennessee [4]	4	2	5	6	21	12	17	16	71	40	87	87
Alabama [4]	2	1	7	11	55	31	28	14	262	149	226	119
Mississippi [3]	15	6	4	4								
W. SO. CEN.												
Arkansas	10	4	5	5	57	23	38	38	176	71	143	69
Louisiana [4]	10	4	5	12	17	7	5	5	264	109	13	24
Oklahoma	2	1	3	4	34	17	18	31	352	175	95	65
Texas [4]	19	23	25	27	148	179	163	137	399	482	79	216
MOUNTAIN												
Montana [3]	9	1	0	2	94	10		7	2,172	232	84	84
Idaho [3]	20	2	0	0	31	3	8	3	806	79	5	14
Wyoming [3 5]	0	0	0	0					1,549	71	26	26
Colorado [3 5]	39	8	14	6	58	12			1,112	231	266	266
New Mexico	0	0	4	4	12	1	3	2	124	10	76	74
Arizona	25	2	0	0	491	40	40	30	258	21	16	22
Utah [3 5]	0	0	2	0	30	3			725	73	389	46
PACIFIC												
Washington	9	3	1	3					4,009	1,300	37	62
Oregon [3]	0	0	7	1	139	28	30	21	413	83	36	39
California	20	24	22	25	25	31	20	32	1,874	2,285	564	1,119
Total	11	280	305	373	43	914	535	572	590	14,587	22,116	22,116
21 weeks	17	9,031	10,693	11,339	329	146,309	41,434	100,109	555	288,402	660,788	558,589

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median
NEW ENG.												
Maine	0	0	0	0	0	0	0	0	84	14	31	18
New Hampshire	0	0	0	0	0	0	0	0	41	4	7	8
Vermont	0	0	0	0	0	0	0	0	67	5	16	16
Massachusetts	1.2	1	0	2	0	0	0	2	185	157	323	234
Rhode Island	0	0	0	0	0	0	0	0	46	6	16	20
Connecticut	9	3	0	0	0	0	0	0	151	51	98	98
MID. ATL.												
New York [3]	2	5	4	5	0.4	1	1	1	195	486	643	758
New Jersey	1.2	1	0	3	0	0	0	0	263	221	97	181
Pennsylvania	3	6	8	8	0	0	0	1	139	274	391	573

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended May 27, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median
E. NO. CEN.												
Ohio	0.8	1	4	9	0	0	1	1	231	300	241	390
Indiana	0	0	0	3	0	0	0	0	140	94	28	90
Illinois	0	0	0	7	0.7	1	1	1	239	364	331	512
Michigan [3]	1.1	1	1	3	1.1	1	0	0	465	440	381	381
Wisconsin	4	2	1	1	0	0	0	1	283	161	155	289
W. NO. CEN.												
Minnesota	0	0	0	1	0	0	1	0	142	73	79	130
Iowa [3]	0	0	0	1	0	0	1	0	124	61	88	88
Missouri	0	0	0	2	0	0	0	0	71	55	14	71
North Dakota	0	0	0	0	0	0	0	0	51	7	6	32
South Dakota	0	0	0	0	0	0	0	0	60	8	7	11
Nebraska	0	0	0	0	0	0	0	0	95	25	14	47
Kansas	2.8	1	1	1	0	0	0	0	148	53	84	84
SO. ATL.												
Delaware	0	0	0	0	0	0	0	0	79	4	6	7
Maryland [2]	0	0	0	4	0	0	0	0	80	26	68	56
District of Columbia	0	0	0	0	0	0	0	0	105	13	15	15
Virginia [2]	0	0	1	6	0	0	0	0	9	5	17	23
West Virginia	2.7	1	1	1	2.7	1	0	0	65	24	25	55
North Carolina	1.5	1	1	2	0	0	0	1	26	18	10	16
South Carolina [4]	0	0	1	0	60	22	0	0	16	6	4	4
Georgia [4]	0	0	0	0	5	3	1	0	10	6	10	7
Florida	0	0	1	0	3	1	1	0	21	7	2	2
E. SO. CEN.												
Kentucky	1.7	1	2	2	1.7	1	1	0	42	24	12	29
Tennessee [4]	0	0	2	2	0	0	0	0	44	25	7	9
Alabama [4]	4	2	6	4	0	0	2	1	9	5	11	5
Mississippi [3]	2.5	1	1	1	2.5	1	0	0	3	1	2	5
W. SO. CEN.												
Arkansas	5	2	0	0	0	0	0	0	10	4	3	5
Louisiana [4]	0	0	1	1	2.4	1	4	0	24	10	11	8
Oklahoma	2	1	0	1	0	0	0	0	32	16	14	14
Texas [4]	1.7	2	1	4	0	0	2	0	17	21	68	49
MOUNTAIN												
Montana [3]	0	0	0	0	9	1	0	0	131	14	21	21
Idaho [3]	0	0	0	0	0	0	0	0	41	4	4	1
Wyoming [3] [5]	22	1	0	0	0	0	0	0	87	4	5	13
Colorado [3] [5]	5	1	1	1	0	0	0	0	212	44	45	45
New Mexico	0	0	0	1	0	0	0	0	124	10	10	11
Arizona	0	0	0	0	25	2	0	0	135	11	5	11
Utah [2] [3]	0	0	0	0	0	0	0	0	149	15	20	20
PACIFIC												
Washington	0	0	0	1	0	0	0	0	111	36	17	55
Oregon [3]	5	1	2	0	0	0	0	0	45	9	28	28
California	0	0	7	7	1.6	2	2	5	109	133	202	202
Total	1.4	35	47	120	1.5	38	18	22	133	3,354	3,692	5,438
21 weeks	1.9	1,003	1,639	2,995	0.9	451	408	432	192	101,249	117,582	140,683

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended May 27, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases	1934–38, median	May 27, 1939, rate	May 27, 1939, cases	May 28, 1938, cases
NEW ENG.											
Maine	0	0	0	0	0	0	0	2	1,074	178	68
New Hampshire	0	0	0	0	0	0	0	0	20	2	
Vermont	0	0	0	0	13	1	0	0	630	47	57
Massachusetts	0	0	0	0	0	0	1	1	112	95	86
Rhode Island	0	0	0	0	31	4	0	0	321	42	14
Connecticut	0	0	0	0	12	4	4	1	240	81	143
MID. ATL.											
New York [1]	6	15	0	0	4	10	11	6	164	410	452
New Jersey	0	0	0	0	1	1	5	2	407	342	177
Pennsylvania	0	0	0	0	4	8	11	7	118	232	157
E. NO. CEN.											
Ohio	8	11	1	1	6	8	6	11	139	181	212
Indiana	37	25	22	1	7	5	2	2	137	92	8
Illinois	7	10	7	7	3	5	4	4	163	249	169
Michigan [3]	11	10	7	0	3	3	3	3	253	239	295
Wisconsin	2	1	5	6	0	0	2	2	220	125	209
W. NO. CEN.											
Minnesota	21	11	13	10	0	0	0	1	85	44	31
Iowa [3]	83	41	31	31	12	6	2	2	47	23	37
Missouri	57	44	11	6	1	1	2	1	31	24	5
North Dakota	7	1	2	2	0	0	0	1	7	1	17
South Dakota	128	17	10	9	0	0	0	0	0	0	11
Nebraska	23	6	3	4	4	1	0	0	103	27	14
Kansas	31	11	28	28	6	2	1	1	78	28	148
SO. ATL.											
Delaware	0	0	0	0	20	1	1	1	216	11	9
Maryland [2 3]	0	0	0	0	9	3	3	6	123	40	57
District of Columbia	0	0	0	0	8	1	4	0	218	27	9
Virginia [3]	0	0	0	0	9	5	2	5	66	35	51
West Virginia	0	0	0	0	0	0	5	6	65	24	168
North Carolina	0	0	1	0	4	3	8	6	365	250	315
South Carolina [4]	0	0	0	0	16	6	6	6	167	61	62
Georgia [4]	0	0	0	1	13	8	21	17	53	32	70
Florida	0	0	0	0	21	7	8	6	87	29	33
E. SO. CEN.											
Kentucky	3	2	3	0	5	3	5	5	16	9	87
Tennessee [4]	67	38	0	0	2	1	7	5	67	38	59
Alabama [4]	4	2	0	0	2	1	9	8	123	70	44
Mississippi [3]	0	0	4	0	5	2	5	5			
W. SO. CEN.											
Arkansas	22	9	9	2	7	3	14	3	82	33	66
Louisiana [4]	2	1	1	0	19	8	4	12	63	26	2
Oklahoma	12	6	31	2	12	6	6	5	46	23	21
Texas [4]	5	6	4	5	12	14	8	9	154	186	297
MOUNTAIN											
Montana [3]	19	2	15	12	19	2	0	0	281	30	50
Idaho [3]	0	0	9	6	0	0	1	1	20	2	7
Wyoming [3 5]	0	0	1	3	6	0	0	0	0	0	1
Colorado [3 6]	10	2	6	4	10	2	2	2	385	80	28
New Mexico	0	0	0	0	0	0	1	1	494	40	19
Arizona	25	2	3	0	12	1	1	2	221	18	41
Utah [3 5]	0	0	0	0	0	0	1	0	516	52	60
PACIFIC											
Washington	12	4	28	4	6	2	0	1	68	22	133
Oregon [3]	10	2	4	4	10	2	1	2	94	19	27
California	14	17	18	15	8	10	4	5	153	187	356
Total	12	296	277	215	6	150	181	179	154	3,806	4,382
21 weeks	14	7,312	10,436	4,657	5	2,510	2,721	2,721	161	83,808	89,953

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended May 27, 1939, 24 cases as follows: New York, 1; Iowa, 2; Maryland, 6; Virginia, 1; Montana, 3; Idaho, 1; Wyoming, 2; Colorado, 3; Utah, 2; Oregon, 3.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended May 27, 1939, 51 cases as follows: South Carolina, 1; Georgia, 21; Tennessee, 2; Alabama, 11; Louisiana, 2; Texas, 14.
[5] Colorado tick fever, week ended May 27, 1939, 13 cases as follows: Wyoming, 3; Colorado, 10.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Meningitis, meningococcus	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid and paratyphoid fever
April 1939										
Arizona	1	19	824	1	112	2	3	45	18	18
Colorado	2	50	92		1,651		0	146	10	1
Dist. of Columbia	2	26	16		1,053		0	70	0	1
Hawaii Territory	0	5	11		4		2	1	0	8
Kansas		22	81	2	253		1	312	11	4
Louisiana	5	47	140	40	688	6	1	54	1	55
Maine	2	11	386		162		0	96	0	6
Montana	0	3	231	1	677		0	87	6	2
Nevada	0	0	1		421		0	6	0	0
North Carolina	5	58	141	54	2,888	24	1	108	2	16
Ohio	1	90	397	1	98	1	1	1,879	72	14
Oregon	0	8	350		325		2	102	27	17
South Dakota	1	15	85		1,545		0	101	76	3
Virginia	3	57	2,227	4	2,685	19	2	140	0	7
Washington	3	1	26		3,476	1	0	168	9	3
Wyoming	0	2			290		0	17	0	0

April 1939

	Cases
Chickenpox:	
Arizona	72
Colorado	313
District of Columbia	75
Hawaii Territory	228
Kansas	451
Louisiana	75
Maine	189
Montana	162
Nevada	1
North Carolina	372
Ohio	1,772
Oregon	182
South Dakota	51
Virginia	440
Washington	729
Wyoming	36
Colorado tick fever:	
Wyoming	4
Conjunctivitis, infectious:	
Hawaii Territory	55
Wyoming	1
Diarrhea:	
Ohio (under 2 years; enteritis included)	4
Dysentery:	
Arizona (bacillary)	41
District of Columbia (amoebic)	1
Hawaii Territory (amoebic)	1
Hawaii Territory (bacillary)	2
Kansas (amoebic)	2
Louisiana (amoebic)	12
Louisiana (bacillary)	1
Maine (amoebic)	1
Maine (bacillary)	1
North Carolina (bacillary)	1
Oregon (amoebic)	2
Virginia (amoebic)	2
Virginia (bacillary)	60
Washington (amoebic)	2
Encephalitis, epidemic or lethargic:	
Colorado	1
Kansas	1
Louisiana	1
Montana	1
Ohio	1
Oregon	1
Washington	1
German measles:	
Arizona	6
Kansas	14
Maine	12

April 1939—Continued

	Cases
German measles—Contd.	
Montana	2
North Carolina	33
Ohio	7
Washington	15
Wyoming	1
Hookworm disease:	
Hawaii Territory	13
Louisiana	142
Impetigo contagiosa:	
Hawaii Territory	18
Kansas	7
Montana	2
Oregon	57
Lead poisoning:	
Ohio	8
Leprosy:	
Hawaii Territory	1
Louisiana	2
Mumps:	
Arizona	63
Colorado	39
Hawaii Territory	254
Kansas	1,349
Maine	96
Montana	42
Nevada	10
Ohio	2,100
Oregon	135
South Dakota	67
Virginia	510
Washington	294
Wyoming	101
Ophthalmia neonatorum:	
Ohio	62
Puerperal septicemia:	
Ohio	2
Rabies in animals:	
Louisiana	12
Oregon	4
Washington	39
Rocky Mountain spotted fever:	
Colorado	2
Montana	3
Nevada	2
Oregon	13
Washington	2
Wyoming	3
Scabies:	
Kansas	11
Montana	8
Oregon	37
Septic sore throat:	
Colorado	1
Hawaii Territory	1
Kansas	12
Montana	9

April 1939—Continued

	Cases
Septic sore throat—Contd.	
North Carolina	11
Ohio	12
Oregon	10
South Dakota	3
Virginia	74
Washington	14
Wyoming	1
Tetanus:	
Hawaii Territory	1
Tick paralysis:	
Montana	2
Trachoma:	
Hawaii Territory	8
Trichinosis:	
Hawaii Territory	3
Tularaemia:	
Louisiana	12
North Carolina	2
Ohio	1
Virginia	1
Typhus fever:	
Louisiana	3
North Carolina	9
Undulant fever:	
Arizona	4
Colorado	2
Kansas	3
Louisiana	7
Maine	1
North Carolina	1
Ohio	5
Oregon	3
Virginia	2
Washington	3
Vincent's infection:	
Kansas	6
Maine	6
Oregon	32
Washington	1
Whooping cough:	
Arizona	74
Colorado	254
District of Columbia	128
Hawaii Territory	222
Kansas	111
Louisiana	72
Maine	318
Montana	17
Nevada	2
North Carolina	1,077
Ohio	596
Oregon	68
South Dakota	27
Virginia	274
Washington	85
Wyoming	7

WEEKLY REPORTS FROM CITIES

City reports for week ended May 20, 1939

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza cases	Influenza deaths	Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
Data for 90 cities:											
5-year average	152	88	38	6,497	613	2,106	17	408	28	1,361	----
Current week[1]	100	82	28	4,439	342	1,345	19	345	26	1,097	----
Maine:											
Portland	0	----	0	0	1	1	0	0	0	21	12
New Hampshire:											
Concord	0	----	0	0	1	0	0	0	0	0	10
Manchester	0	----	0	0	1	0	0	0	0	0	11
Nashua	0	----	0	0	1	0	0	0	0	0	10
Vermont:											
Barre	----	----	----	----	----	----	----	----	----	----	----
Burlington	0	----	0	0	0	0	0	0	0	1	9
Rutland	0	----	0	0	2	0	0	0	0	0	4
Massachusetts:											
Boston	0	----	4	228	6	56	0	12	0	13	208
Fall River	----	----	----	----	----	----	----	----	----	----	----
Springfield	0	----	0	21	0	1	0	0	0	0	32
Worcester	0	----	0	20	3	6	0	0	0	14	39
Rhode Island:											
Pawtucket	0	----	0	15	0	1	0	0	0	0	21
Providence	0	----	1	64	2	2	0	1	0	84	57
Connecticut:											
Bridgeport	0	----	0	9	0	6	0	1	0	0	34
Hartford	0	1	1	22	4	3	0	1	0	4	49
New Haven	0	----	0	301	2	1	0	1	0	5	40
New York:											
Buffalo	0	----	0	143	10	34	0	4	0	19	120
New York	21	9	3	218	72	235	0	75	2	107	1,425
Rochester	0	----	0	228	5	20	0	1	0	8	78
Syracuse	0	----	0	305	1	9	0	0	0	58	57
New Jersey:											
Camden	1	----	0	0	1	6	0	0	0	2	27
Newark	0	1	1	6	3	46	0	9	0	58	92
Trenton	0	----	1	0	3	24	0	2	0	1	42
Pennsylvania:											
Philadelphia	1	3	0	63	15	49	0	27	2	82	437
Pittsburgh	5	5	0	1	9	29	0	11	1	30	157
Reading	1	----	0	8	2	1	0	2	1	0	30
Scranton	0	----	----	0	----	4	0	----	1	1	----
Ohio:											
Cincinnati	2	----	0	2	7	42	0	11	2	3	132
Cleveland	3	7	1	9	8	52	0	9	0	43	162
Columbus	1	----	0	4	2	5	0	3	0	7	67
Toledo	0	2	1	36	5	18	5	4	0	22	66
Indiana:											
Anderson	0	----	0	0	0	4	0	0	0	2	7
Fort Wayne	0	----	0	0	1	0	0	0	0	0	31
Indianapolis	3	----	1	3	9	50	0	4	0	22	105
Muncie	0	----	0	0	0	4	0	0	0	0	13
South Bend	0	----	0	1	1	0	0	0	0	14	12
Terre Haute	0	----	0	0	1	0	0	0	0	0	18
Illinois:											
Alton	0	----	0	0	0	1	0	0	0	0	5
Chicago	9	3	1	26	22	238	0	40	1	76	702
Elgin	0	----	0	0	0	1	0	0	0	5	2
Moline	0	----	0	2	0	3	0	0	0	1	12
Springfield	0	----	0	1	1	0	0	0	0	4	27
Michigan:											
Detroit	7	1	0	36	12	117	0	16	0	67	250
Flint	1	----	0	18	3	17	0	0	1	0	33
Grand Rapids	0	----	0	2	0	28	0	0	0	3	30
Wisconsin:											
Kenosha	0	----	0	1	0	6	0	0	0	3	6
Madison	0	----	0	140	1	1	0	0	0	6	10
Milwaukee	0	1	1	4	4	24	0	2	1	46	108
Racine	0	----	0	2	0	5	0	0	0	2	14
Superior	0	----	0	2	1	1	0	0	0	0	9

[1] Figures for Barre, and Fall River estimated; reports not received.

City reports for week ended May 20, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0	------	0	1	0	2	0	0	0	0	20
Minneapolis	1	------	0	81	5	17	8	3	1	20	108
St. Paul	0	------	0	54	6	7	0	3	0	7	68
Iowa:											
Cedar Rapids	0	------	--------	2	-------	0	0	-------	0	0	--------
Davenport	0	------	--------	1	-------	3	4	-------	0	0	--------
Des Moines	0	------	0	0	0	25	11	0	0	0	24
Sioux City	1	------	--------	0	-------	1	0	-------	0	2	--------
Waterloo	1	------	--------	2	-------	5	0	-------	0	2	--------
Missouri:											
Kansas City	0	------	1	1	8	11	4	6	0	0	105
St. Joseph	0	------	0	0	2	0	0	1	0	0	20
St. Louis	3	------	0	3	7	24	0	6	0	19	173
North Dakota:											
Fargo	0	------	0	0	1	0	0	0	0	0	9
Grand Forks	0	------	--------	0	-------	0	0	-------	0	0	--------
Minot	0	------	0	2	0	0	0	0	0	0	7
South Dakota:											
Aberdeen	0	------	--------	201	-------	0	13	-------	0	0	--------
Nebraska:											
Lincoln	0	------	--------	52	-------	1	0	-------	0	8	--------
Omaha	1	------	0	7	4	8	3	0	0	4	68
Kansas:											
Lawrence	0	------	0	0	0	0	0	0	0	0	3
Topeka	0	------	0	1	1	3	2	0	0	1	12
Wichita	0	------	0	18	3	3	0	0	0	1	25
Delaware:											
Wilmington	1	------	0	0	2	2	0	2	1	0	28
Maryland:											
Baltimore	0	2	0	165	9	19	0	8	0	15	189
Cumberland	0	------	0	0	0	0	0	0	0	0	9
Frederick	0	------	0	0	0	0	0	0	0	0	4
Dist. of Col.:											
Washington	1	------	0	391	6	12	0	7	0	15	142
Virginia:											
Lynchburg	1	------	0	66	0	0	0	0	0	19	6
Norfolk	0	------	0	4	0	3	0	3	0	0	26
Richmond	0	------	0	367	0	3	0	1	0	0	46
Roanoke	0	------	0	0	0	0	0	1	0	0	7
West Virginia:											
Charleston	0	------	0	0	1	0	0	1	0	2	22
Huntington	6	------	--------	0	-------	0	0	-------	0	0	--------
Wheeling	0	------	0	0	2	0	0	2	0	14	24
North Carolina:											
Gastonia	0	1	--------	0	-------	0	0	-------	0	0	--------
Raleigh	0	------	0	0	0	2	0	1	0	5	13
Wilmington	0	------	0	1	0	0	0	0	0	1	8
Winston-Salem	0	------	0	5	1	0	0	2	0	0	18
South Carolina:											
Charleston	0	10	0	0	0	1	0	1	0	9	14
Greenville	0	------	0	2	3	0	1	0	0	2	14
Georgia:											
Atlanta	1	10	2	0	4	2	0	5	0	1	72
Brunswick	0	------	0	9	0	0	0	0	0	0	4
Savannah	0	10	1	0	1	0	0	2	0	13	32
Florida:											
Miami	0	------	0	0	3	0	0	3	0	10	35
Tampa	0	------	0	41	1	2	0	0	0	5	24
Kentucky:											
Ashland	0	------	0	0	0	0	0	0	0	0	6
Covington	0	------	0	1	0	4	0	2	0	0	9
Lexington	0	------	0	1	1	6	0	1	0	0	18
Louisville	0	------	0	6	7	7	0	3	0	7	60
Tennessee:											
Knoxville	0	3	0	2	1	6	0	1	0	0	23
Memphis	0	------	0	0	2	14	0	6	1	4	70
Nashville	0	------	1	1	2	9	0	1	0	5	43
Alabama:											
Birmingham	1	4	0	1	1	0	0	3	0	4	61
Mobile	0	------	0	0	1	1	0	1	0	0	26
Montgomery	0	1	--------	2	-------	0	0	--------	0	1	--------

City reports for week ended May 20, 1939—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Arkansas:											
Fort Smith	0	------	--------	6	--------	2	0	--------	0	0	--------
Little Rock	0	------	1	1	0	0	0	0	0	1	2
Louisiana:											
Lake Charles	0	------	0	3	0	3	0	0	0	0	2
New Orleans	9	------	0	20	9	11	0	6	4	33	147
Shreveport	0	------	0	8	1	1	0	3	1	1	41
Oklahoma:											
Oklahoma City	0	------	0	16	2	3	0	0	0	0	37
Tulsa	0	------	--------	36	--------	0	0	--------	0	0	--------
Texas:											
Dallas	1	2	2	27	4	1	0	2	0	3	67
Fort Worth	0	4	0	19	1	0	0	1	0	3	34
Galveston	0	------	0	0	1	0	0	1	0	0	13
Houston	4	------	2	45	6	2	0	3	3	3	69
San Antonio	1	------	0	0	3	0	0	2	0	4	59
Montana:											
Billings	1	------	0	0	0	0	0	0	0	1	9
Great Falls	0	------	0	42	1	0	0	0	0	0	10
Helena	0	------	0	0	1	0	0	0	0	0	5
Missoula	0	------	0	5	1	0	0	1	0	0	7
Idaho:											
Boise	0	------	0	3	0	0	0	1	0	0	3
Colorado:											
Colorado Springs	1	------	0	9	1	5	0	1	0	7	8
Denver	5	------	2	34	4	6	0	4	0	25	81
Pueblo	0	------	0	102	1	2	0	1	0	13	9
New Mexico:											
Albuquerque	0	------	0	0	0	5	0	4	1	5	11
Utah:											
Salt Lake City	0	------	0	8	2	7	0	1	1	18	26
Washington:											
Seattle	0	------	1	527	0	6	2	5	0	5	72
Spokane	0	------	0	136	0	1	0	0	0	0	21
Tacoma	0	------	0	14	0	0	0	0	0	3	32
Oregon:											
Portland	2	------	0	5	5	4	2	1	0	9	87
Salem	0	------	--------	1	--------	1	0	--------	0	0	--------
California:											
Los Angeles	12	11	0	341	13	45	0	14	0	26	306
Sacramento	2	------	0	123	3	3	0	2	0	0	28
San Francisco	0	2	0	42	7	13	0	4	3	5	148

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Rhode Island:				Arkansas:			
Pawtucket	1	0	0	Fort Smith	1	0	0
New York:				Little Rock	0	1	0
Buffalo	1	0	0	Louisiana:			
New York	3	1	0	New Orleans	1	0	1
Pennsylvania:				Shreveport	0	2	0
Pittsburgh	1	0	0	Texas:			
Reading	1	0	0	Houston	2	0	0
Scranton	1	0	0	Washington:			
West Virginia:				Seattle	0	1	0
Huntington	0	0	1	California:			
South Carolina:				Los Angeles	0	0	1
Charleston	0	0	16				

Encephalitis, epidemic or lethargic.—Cases: New York, 1; Davenport, 1; Topeka, 1.
Pellagra.—Cases: Washington, 1; Charleston, S. C., 2; Montgomery, 2; San Antonio, 1; Los Angeles, 1.
Typhus fever.—Cases: New York, 1; Miami, 1; Houston, 2.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended May 6, 1939.—During the week ended May 6, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			3	2	1	1			7
Chickenpox	7	2	109	104	38	23		53	336
Diphtheria	9		45	1	3	2			60
Dysentery				2					2
Influenza	47			76		56		17	196
Lethargic encephalitis						5			5
Measles	22		576	877	7	1	4	3	1,490
Mumps			[1] 35	63	28	4	5	4	139
Pneumonia	5			12		2	1	6	26
Scarlet fever	7	10	45	157	14	13	14	3	263
Smallpox					2				2
Trachoma					1			2	3
Typhoid and paratyphoid fever		3	12	3		1		1	20
Tuberculosis	20	9	6	65	7	1	4		112
Whooping cough	3	4	60	184	18	52	21	84	426

[1] "Other communicable diseases," mostly mumps.

NOTE: None of the diseases listed was reported from Prince Edward Island during the week ended May 6, 1939.

CUBA

Provinces—Notifiable diseases—4 weeks ended March 4, 1939.—During the 4 weeks ended March 4, 1939, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer		2	1	3	1		7
Chickenpox		3		2	1	5	11
Diphtheria		22	4	1	1	1	29
Leprosy		2					2
Malaria	15	11	2	12	6	44	90
Measles		1	1	2		3	7
Rabies	3						3
Tuberculosis	28	49	22	39	16	58	212
Typhoid fever	11	73	3	16	12	33	148
Undulant fever		1					1
Yaws						2	2

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for May 26, 1939, pages 906-918. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China.—During the week ended May 20, 1939, 9 cases of cholera were reported in Hong Kong, China. Under date of May 24, 1939, Japanese authorities reported several hundred cases of cholera with 154 deaths in Fatshan. It was stated that 9 cases of cholera, with 6 deaths, had been reported in Canton.

Plague

Belgian Congo—Virakwa.—During the week ended May 20, 1939, 2 cases of plague were reported in Virakwa, Belgian Congo.

Egypt—Asyut Province.—During the week ended May 20, 1939, 1 case of plague was reported in Asyut Province, Egypt.

Smallpox

Iraq—Baghdad.—During the week ended May 13, 1939, 1 imported case of smallpox was reported in Baghdad, Iraq.

Japan—Tokyo.—On April 20, 1939, 1 case of smallpox was reported in Tokyo, Japan.

Typhus Fever

Egypt.—Typhus fever has been reported in Egypt as follows: During the week ended May 20, 1939, Alexandria, 3 cases, Cairo, 2 cases; during the week ended May 13, Provinces, 200 cases.

Iraq.—During the week ended May 13, 1939, 1 case of typhus fever was reported in Baghdad, and during the week ended May 27, 7 cases were reported in Tapasura, Iraq.

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