Public Health Reports

Vol. 54 • JULY 7, 1939 • *No. 27*

PREVALENCE OF POLIOMYELITIS

The incidence of poliomyelitis in the United States declined during the week ended July 1, 1939, a total of 80 cases being reported by the State health officers as compared with 83 cases for the preceding week. The figure for the current week is much below the estimated expectancy of 158 cases based on the 5-year median.

Of the 83 cases reported, South Carolina with 29 cases, California with 16 cases, and Texas with 9 cases accounted for 67 percent of the total.

A summary of poliomyelitis incidence for the 4-week period May 21–June 17, 1939, is presented in the following article and accompanying table, and the current reported incidence by States is published each week in the Public Health Reports in the first table under the section headed "Prevalence of Disease."

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

May 21–June 17, 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 June 17, 1939, the number reported for the corresponding period in 1938, and the median number for the years 1934–38.

DISEASES ABOVE MEDIAN PREVALENCE

Poliomyelitis.—The incidence of poliomyelitis in South Carolina, to which attention was called in a previous summary,[1] has remained at approximately the same level for a period of 7 weeks ended June 17,

[1] Public Health Reports, June 9, 1939, p. 969.

with an average of 23 cases per week. Arizona reported 16 cases for the current period, as compared with none for the corresponding period in 1938, California 36 cases as against 6 last year, and Georgia 10 as against 2 cases. Three-fourths of the total number of cases reported (217) occurred in the 4 States mentioned. In other regions of the country the incidence was below the normal seasonal expectancy; and, although a rise in the number of cases of this disease is to be expected at this season of the year, the increases over the preceding 4-week period were small.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period May 21–June 17, 1939, the number for the corresponding period in 1938, and the median number of cases reported for the corresponding period 1934–38 [1]

Division	Diphtheria			Influenza [2]			Measles [3]			Meningococcus meningitis		
	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median
United States [1]	1,022	1,260	1,487	3,236	2,120	2,120	48,249	79,893	79,893	140	220	363
New England	12	29	48	6	15	12	8,099	3,096	5,341	10	7	15
Middle Atlantic	208	237	311	36	29	40	8,382	24,521	18,292	58	54	64
East North Central	213	239	313	304	111	314	4,797	29,576	27,981	15	31	54
West North Central	60	78	114	138	63	157	3,225	5,235	5,285	3	12	28
South Atlantic	173	171	206	1,396	345	451	6,366	8,391	4,157	15	36	95
East South Central	71	98	98	230	135	137	810	1,429	1,429	15	40	40
West South Central	137	165	204	705	864	704	2,637	1,424	1,424	12	13	26
Mountain	43	103	57	221	122	122	1,991	2,838	2,838	7	4	8
Pacific	105	140	132	200	436	309	11,942	3,383	4,395	5	23	19

Division	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median	Current period	1938	5-year median
United States [1]	217	87	164	10,046	12,685	17,305	1,057	1,366	812	875	1,023	981
New England	2	2	5	767	1,772	1,377	0	0	0	28	17	24
Middle Atlantic	10	9	13	2,816	3,802	4,989	46	0	0	71	87	78
East North Central	9	12	13	3,904	3,799	6,567	230	239	111	102	87	91
West North Central	4	3	7	808	1,165	1,925	331	505	412	40	29	66
South Atlantic	119	12	12	386	518	566	22	11	4	204	276	262
East South Central	6	21	8	219	162	192	147	46	5	87	165	140
West South Central	10	20	20	171	315	267	114	198	62	167	243	207
Mountain	19	2	3	321	352	387	39	126	109	29	62	43
Pacific	38	6	24	654	809	952	128	241	103	147	57	55

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

Smallpox.—Tennessee, with an average of only 1 case per year during this period in the past 6 years, reported 139 cases of smallpox for the 4 weeks ended June 17. New York reported 45 cases, which breaks the past 6-year record of no cases reported for this period in that State. Georgia, with 18 cases, placed the incidence in the South Atlantic region on the highest level in that region since 1931. The high incidence of smallpox, which has been mostly confined to the Western and North Central regions, appears now to be spreading into

other regions. For the country as a whole the current incidence (1,057 cases) was only about 75 percent of the 1938 figure for this period, but it was about 30 percent in excess of the 1934–38 average incidence; with the exception of last year the current incidence is the highest for this period since 1931.

Influenza.—Although the number of cases of influenza declined about 70 percent during the current period, the total number of cases reported (3,236) was about 50 percent above the average seasonal incidence. The South Atlantic region appeared to be largely responsible for the excess incidence; in that region the number of cases was more than three times the preceding 5-year average number of cases for this period. In other regions the incidence compared very favorably with the experience of preceding years, the North Atlantic, North Central, and Pacific regions reporting a lower incidence, the West South Central approximately the same number of cases, and the East South Central and Mountain only slight increases over the 1934–38 median figure for the corresponding period.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—For the 4 weeks ended June 17 there were 1,022 cases of diphtheria reported, as compared with 1,260, 1,367, and 1,487 for the corresponding period in 1938, 1937, and 1936, respectively. The current incidence is the lowest recorded for this period in the 11 years for which these data are available. The country in general shared in this favorable situation, each geographic section reporting a very definite decline in the number of cases from the 1934–38 average incidence for this period.

Measles.—The number of cases (48,249) of measles reported for the current period was about 60 percent of the number reported for this period in 1938, which figure (79,893) also represents the preceding 5-year average incidence. The disease was unusually prevalent in the New England, South Atlantic, West South Central, and Pacific regions, while the Middle Atlantic, North Central, East South Central, and Mountain regions reported a relatively low incidence. Since the beginning of the current year the incidence in the Pacific region has been the highest on record; for the current period the number of cases (approximately 12,000) was about two and one-half times the normal seasonal incidence.

Scarlet fever.—The incidence of scarlet fever (10,046 cases) was the lowest in recent years. The East South Central region reported a 30-percent increase over last year's figure for this period, as well as a slight increase over the 1934–38 average incidence, and in the Mountain region the incidence was only slightly below the seasonal expectancy, but all other regions reported very definite decreases from the average incidence for this period.

Meningococcus meningitis.—The meningococcus meningitis incidence continued at a relatively low level in all sections of the country. The number of cases (140) reported for the 4 weeks ended June 17 was about 60 percent of the number reported for this period in 1938, and about 40 percent of the average figure (363 cases) for the period. For the years 1932–34, the only 3 years within the past 11 years in which the incidence has been exceptionally low, the average number of cases for this period was approximately 200 cases, which shows the very low incidence of this disease that has prevailed since the beginning of the current year.

Typhoid fever.—For the 4 weeks ended June 17, the State of Washington reported 113 cases of typhoid fever, as compared with 9, 3, and 8 cases for the corresponding period in 1938, 1937, and 1936, respectively. Ohio, with 40 cases as against 26 last year, and Illinois, with 33 cases as against 19, raised the incidence in the East North Central region slightly above the normal seasonal expectancy. In other regions the incidence either stood at about the normal seasonal level or was definitely lower than in recent years. For the country as a whole the number of cases reported (875) was about 10 percent below the preceding 5-year average number of cases for this period.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ended June 17, based on data received from the Bureau of the Census, was 10.6 per 1,000 inhabitants (annual basis). The current rate is the lowest for this period since 1933; the average rate for this period in the years 1934–38 was 11.1.

PROVISIONAL MORTALITY RATES FOR THE FIRST QUARTER OF 1939

The mortality rates in this report are based upon preliminary data for 38 States, the District of Columbia, Alaska, and Hawaii for the first 3 months of 1939. Comparative data for 29 States and the District of Columbia are presented for the corresponding period of 1938.

This report is made possible through a cooperative arrangement with the respective States, which voluntarily furnish provisional quarterly and annual tabulations of current birth and death records. The reports are compiled and published by the United States Public Health Service.

Because of lack of uniformity in the method of classifying deaths according to cause, and because a certain number of certificates were not filed in time to be included, these data are preliminary and may differ in some instances from the final figures subsequently published by the Bureau of the Census.

In the past, these preliminary reports have provided an early and accurate index of the trend in mortality for the country as a whole. Some deviation from the final figures for individual States is to be expected, because of the provisional nature of the information. It is believed, however, that the trend of mortality within each State is correctly represented. Comparisons of specific causes of death among different States are subject to error because of differences in tabulation procedure and completeness of reporting. Comparisons of this nature should be made only from the final figures published by the Bureau of the Census.

Reports for the first quarter of 1939 reveal that the unusually favorable record of 1938 has not been maintained. Mortality from all causes, 12.2 per 1,000 estimated population, during this quarter was 7 percent greater than that for the corresponding period of 1938, but, nevertheless, it was still about 5 percent less than the rate for 1937. The increase was fairly widespread; 23 of the 30 States for which comparative data are available reported an increased death rate, while only 2 reported a lower rate. The rise in the mortality rate was not due to an unusually high rate from any specific disease, although a minor outbreak of influenza late in the winter resulted in some increase in mortality, but rather to generally increased mortality from a large number of diseases.

Increased death rates were reported for nearly all of the important diseases, cancer, diabetes, cerebral hemorrhage, heart diseases, influenza, and pneumonia, diseases of the digestive system, nephritis, and for accidents. For all except the first four, however, the rates were less than those reported in 1937. The death rate from automobile accidents, as well as that from all accidents, showed only a very slight increase over 1938. The increase in the total death rate was also reflected in the infant mortality rate, which was about 6 percent higher than during the first quarter of 1938; however, it was still 16 percent below the rate for 1937.

The principal communicable diseases of early childhood, measles, scarlet fever, whooping cough, and diphtheria, took fewer lives than during the first quarter of 1938, and, except for measles, caused fewer deaths than in 1937. Also encouraging was the continued decline in the mortality rate from tuberculosis, although the decrease, 2 percent, was not as great as that for the previous year, 12 percent. The death rate from complications of pregnancy and childbirth showed a decrease of 9 percent over 1938, thus indicating that the declining mortality rate from these causes is continuing in 1939.

The birth rate for these States showed no change from 1938, but since the death rate was higher, the crude rate of natural increase, 4.1 per 1,000 population, was 15 percent less than that for the previous year.

Provisional mortality from certain causes in the first 3 months of 1939, with comparative provisional data for the corresponding period in preceding years

State and period	All causes, rate per 1,000 population (annual basis)	Births (exclusive of stillbirths) per 1,000 population (annual basis)	Rate per 1,000 live births		Death rate per 100,000 population (annual basis)																			
			Total infant mortality	Maternal mortality	Typhoid fever (1, 2)	Measles (7)	Scarlet fever (8)	Whooping cough (9)	Diphtheria (10)	Influenza (11)	Acute poliomyelitis and polioencephalitis (16)	Encephalitis, epidemic or lethargic (17)	Epidemic cerebrospinal meningitis (18)	Tuberculosis, all forms (23-32)	Cancer, all forms (45-53)	Diabetes (59)	Cerebral hemorrhage, apoplexy (82a, b)	Diseases of the heart (90-96)	Pneumonia, all forms (107-109)	Diseases of the digestive system (115-129)	Diarrhea and enteritis under 2 years (119)	Nephritis (130-132)	All accidents (170-195, 201-214)	Automobile accidents (200, 208, 210)
30 States: [1]																								
1939	12.2	16.3	54	4.0	0.6	1.8	1.2	2.4	1.7	32.1	0.2	0.5	0.7	50.1	122.4	29.9	97.9	332.4	107.6	58.1	4.0	80.1	66.4	22.1
1938	11.5	16.3	51	4.4	.6	4.0	1.7	3.2	2.1	20.0	.3	.6	1.2	51.0	117.3	26.0	90.3	296.1	103.9	57.8	4.4	79.0	66.0	22.0
1937	12.9	15.1	64	5.5	.8	.8	2.3	3.1	2.0	69.0	.3	.7	2.6	57.9	112.3	27.9	94.8	309.5	151.0	60.8	4.4	82.2	74.8	27.1
Industrial policyholders, Metropolitan Life Insurance Co., ages 1 and over: [2]																								
1939	8.8	----	----	----	.4	1.0	1.2	2.3	1.6	18.7	----	----	----	47.4	101.9	30.1	66.0	[3] 183.0	78.4	----	[4] 4.9	[5] 58.3	44.2	16.6
1938	8.5	----	----	----	.8	2.9	1.9	1.7	2.2	13.4	----	----	----	47.7	94.9	26.9	64.1	[3] 169.4	78.6	----	[4] 5.6	[5] 58.2	46.7	18.6
1937	9.8	----	----	----	.5	1.0	2.5	3.2	2.2	46.8	----	----	----	56.9	95.9	30.2	66.8	[3] 185.4	122.0	----	[4] 5.2	[5] 66.2	51.4	20.4
Alabama:																								
1939	10.8	20.9	68	5.8	1.4	2.9	.7	3.9	2.6	65.1	.6	.4	1.2	52.8	58.7	13.8	76.6	175.4	121.2	45.1	5.0	73.7	64.8	19.4
1938	11.5	21.9	66	6.7	.7	7.1	1.1	4.4	3.8	55.6	.3	.6	4.0	56.2	57.2	15.4	75.7	176.4	129.9	51.1	5.4	83.6	68.3	20.0
1937	12.2	21.4	78	5.6	1.1	(6)	(6)	5.3	2.7	114.7	.4	.3	5.3	65.0	59.3	12.7	74.1	165.4	146.8	50.0	6.6	85.3	73.3	20.3
Alaska:																								
1939	13.9	25.6	44	2.4	(6)	(6)	(6)	6.2	6.2	(6)	(6)		6.2	(6)	324.4	68.6	6.2	49.9	205.9	180.9	25.0	(6)	12.5	137.3
1938	30.9	40.4	74	(6)	(6)	6.4	6.4	173.8	(6)	77.2	(6)		(6)	740.3	109.4	(6)	225.3	302.6	366.9	64.4	(6)	19.3	316.4	(6)
1937	17.5	24.0	155	11.3	(6)	(6)	(6)	60.8	(6)	74.4	(6)		(6)	331.2	81.1	6.8	74.4	338.0	169.0	40.6	(6)	20.3	141.9	(6)
California:																								
1939	13.3	15.5	45	3.0	.6	1.6	.6	.7	1.1	6.1	.2	.1	.9	66.9	154.7	29.1	99.6	416.8	78.3	78.1	4.6	86.3	90.1	39.4
1938	13.1	15.6	47	3.0	1.2	1.4	.6	2.3	1.9	7.6	.2	.4	1.2	71.7	144.0	26.3	96.6	389.0	89.8	75.4	7.4	88.7	89.9	38.4
1937	16.3	14.3	58	4.8	.7	.6	1.2	4.4	1.9	69.3	.1	.6	3.6	91.3	144.0	32.6	101.6	478.3	160.4	88.8	5.3	96.1	111.0	49.8
Colorado:																								
1939	13.0	18.9	69	5.2	1.1	1.5	1.9	6.4	4.1	50.7	(6)	.4	1.1	54.5	113.1	19.5	105.6	275.5	154.9	63.9	6.4	92.8	71.8	20.7
1938	12.4	18.6	59	5.3	1.1	4.9	1.5	1.9	5.3	23.4	1.1	.4	1.1	64.1	117.0	17.4	98.5	249.0	144.1	75.4	6.0	87.9	75.4	22.6
1937	16.4	17.9	79	7.2	.8	(6)	3.0	8.3	3.8	153.7	1.5	.8	2.6	75.0	121.6	18.9	95.8	253.7	351.4	81.0	6.8	86.0	70.0	21.6

Connecticut:																								
1939	11.3	13.2	37	2.8	(6)	.7	.5	1.9	1.2	10.4	(6)	(6)	(6)	38.7	146.5	29.9	96.9	280.1	86.0	49.4	2.3	86.0	55.7	15.5
1938	11.2	13.3	35	2.8	(6)	(6) .7	.7	.5	1.4	7.7	(6)	.2	.5	37.6	141.3	32.5	93.0	276.3	89.5	52.8	4.0	91.8	62.0	17.4
1937	11.9	12.0	50	3.8	.5	1.6	1.4	2.8	.9	35.7	(6)	.5	1.2	37.5	134.8	39.4	97.0	285.9	127.2	54.9	2.8	92.9	64.4	21.3
Delaware:																								
1939	13.7	17.6	49	4.4	(6)	(6)	(6)	4.6	3.1	24.7	(6)	1.5	(6)	70.9	117.2	40.1	120.3	417.9	145.0	54.0	6.2	128.0	67.8	29.3
1938	13.2	15.9	61	3.9	1.5	3.1	(6)	10.8	1.5	27.9	(6)	(6)	1.5	38.7	103.8	34.1	142.4	400.9	114.5	34.1	(6)	108.4	68.1	21.7
1937	16.0	16.3	80	3.8	3.1	4.7	(6)	6.2	1.5	66.8	(6)	(6)	3.1	48.2	97.9	35.7	116.5	453.7	166.3	54.4	10.9	132.1	82.4	34.2
District of Columbia:																								
1939	14.3	21.1	49	5.4	.6	(6)	(6)	1.9	.6	21.4	(6)	.6	1.3	70.6	160.8	32.2	93.3	403.7	110.4	79.5	7.6	111.0	75.7	18.3
1938	13.7	20.3	52	4.4	(6)	.6	1.9	.6	1.9	8.9	(6)	1.3	2.6	69.6	131.6	24.9	93.9	355.1	152.0	70.2	5.1	118.2	67.1	21.1
1937	17.2	19.5	76	6.0	.6	2.6	1.3	5.8	5.2	52.4	(6)	.6	7.8	102.2	146.8	37.5	116.4	381.6	249.7	75.0	5.2	106.7	88.6	33.0
Florida: [7]																								
1939	13.6	17.1	65	5.9	.7	.7	.4	2.9	1.4	38.8	(6)	.4	1.1	56.1	97.5	26.3	116.2	307.9	83.1	83.4	11.1	102.1	116.5	47.5
1938	14.3	16.9	52	7.8	1.8	.7	.4	2.2	1.8	41.6	(6)	.7	1.1	58.4	107.3	27.7	117.9	326.6	111.0	78.8	5.5	110.2	95.3	42.7
1937	13.3	16.9	59	7.7	2.2	.4	(6)	2.2	3.0	65.2	(6)	.4	5.6	61.1	87.8	24.8	107.4	261.9	88.5	94.5	15.2	100.8	121.1	54.8
Georgia:																								
1939	9.3	17.5	68	7.1	.9	3.4	.4	3.1	1.3	41.9	.3	.1	.6	42.6	50.9	12.2	80.6	159.4	98.9	40.6	4.0	85.9	47.0	18.4
1938	10.4	18.1	68	7.3	.5	6.9	.6	4.4	1.7	47.4	.4	.5	.9	51.6	54.8	13.4	83.2	166.8	124.2	50.0	5.1	104.6	58.9	22.8
1937	10.7	17.7	69	7.5	1.7	.3	.4	2.4	2.6	94.8	.5	.4	1.8	47.6	50.0	11.6	83.7	157.6	120.2	47.3	4.6	94.9	63.5	25.1
Hawaii:																								
1939	6.9	16.6	62	5.2	3.5	(6)	(6)	2.6	2.6	4.3	(6)	(6)	(6)	56.4	52.1	18.2	37.3	129.4	57.3	55.6	10.4	57.3	43.4	11.3
1938	7.2	19.9	56	3.1	1.8	(6)	.9	8.0	.9	5.3	1.8	(6)	.9	59.3	63.8	11.5	50.5	116.9	71.7	61.1	17.7	68.2	39.0	9.7
1937	9.5	20.4	82	5.3	3.6	102.3	(6)	.9	1.8	9.1	(6)	(6)	.9	79.7	62.5	19.0	47.1	121.9	109.5	75.1	19.0	65.2	53.4	16.3
Idaho:																								
1939	10.4	21.1	58	2.3	4.0	.8	.8	2.4	.8	39.4	(6)	.8	4.0	19.3	94.8	26.5	88.3	277.9	106.0	54.6	2.4	36.1	75.5	25.7
1938	9.4	20.9	45	5.0	2.4	1.6	.8	3.2	(6)	21.9	(6)	1.6	1.6	21.9	67.3	5.7	68.1	181.7	118.4	60.0	1.6	39.7	62.4	24.3
1937	12.0	20.3	57	3.6	.8	6.6	2.5	1.6	(6)	118.4	(6)	.8	2.5	28.0	93.0	11.5	78.1	188.4	128.3	67.4	5.8	37.0	65.8	20.6
Illinois:																								
1939	12.6	13.9	45	3.5	.3	.1	1.8	2.9	2.1	35.6	.2	.3	.2	48.9	140.6	33.3	83.0	400.6	103.7	58.4	1.8	112.5	59.3	22.2
1938	11.5	14.3	44	3.6	.3	7.1	3.2	.6	1.6	10.5	.2	.4	.4	46.1	132.6	31.0	78.6	341.5	92.8	61.6	3.0	100.0	62.3	22.6
1937	12.7	13.2	57	5.1	.2	.1	3.7	2.6	2.5	51.5	.4	.5	2.5	52.4	127.1	31.8	80.4	355.3	123.8	71.5	2.8	110.3	87.1	29.2
Indiana:																								
1939	12.8	15.7	49	4.2	.3	.2	2.4	1.0	1.9	69.2	(6)	.7	.6	44.2	112.5	20.8	146.3	275.9	138.7	(6)	2.8	67.8	61.8	23.4
1938	11.6	15.8	44	4.6	.2	6.3	3.0	2.0	3.1	22.8	.5	.9	1.2	42.7	114.0	17.3	129.8	256.6	103.8	(6)	3.0	64.7	65.1	26.3
1937	13.2	14.5	64	3.4	.1	.9	5.6	5.0	1.6	90.6	.5	.5	2.8	54.9	103.4	18.8	127.5	288.7	181.9	(6)	3.9	70.0	73.3	32.7
Iowa:																								
1939	11.8	(6)	(6)	(6)	.5	2.2	2.1	1.7	.5	64.4	(6)	.8	.8	19.3	119.9	34.7	121.6	319.6	97.8	58.7	2.2	62.7	62.5	14.8
1938	10.4	16.7	40	4.1	.5	1.1	4.1	3.8	1.0	23.4	.3	.6	.8	21.4	133.1	23.8	112.6	257.0	100.1	59.1	3.3	66.1	59.6	15.7
1937	11.4	14.7	54	6.7	(6)	.2	7.0	1.7	.3	96.5	.6	1.3	1.1	22.1	124.1	25.9	117.0	244.9	110.9	52.8	3.0	68.8	69.1	15.7
Kansas: [7]																								
1939	10.4	13.6	50	4.9	1.0	(6)	1.0	.7	1.6	27.4	(6)	2.0	.7	22.8	108.6	33.7	82.2	263.6	82.2	52.1	3.0	104.6	92.1	17.2
1938	10.3	14.3	50	3.7	.3	2.0	2.7	2.3	.7	21.9	(6)	.7	.7	26.6	106.2	25.6	86.6	242.6	97.9	58.7	2.0	106.5	90.3	22.9
1937	13.2	13.6	66	5.6	(6)	(6)	8.0	1.7	2.3	116.8	.3	1.0	2.0	31.9	110.9	23.9	127.1	285.4	148.4	59.4	2.3	97.2	108.2	19.9
Maine:																								
1939	14.0	16.8	67	4.2	2.4	.9	.5	4.7	6.6	32.5	(6)	(6)	.5	33.5	149.0	29.7	135.8	422.5	113.6	49.0	4.7	81.1	52.8	12.3
1938	12.9	17.9	50	4.5	1.9	3.8	(6)	3.8	1.4	23.6	.5	(6)	(6)	33.1	145.1	27.9	122.4	345.5	105.9	56.7	7.1	96.0	58.6	14.7
1937	14.4	16.7	69	6.5	1.4	.5	.5	1.9	.5	100.9	(6)	(6)	1.4	30.3	146.9	23.7	126.5	393.7	161.1	58.3	4.7	101.9	46.4	12.3

See footnotes at end of table.

Provisional mortality from certain causes in the first 3 months of 1939, with comparative provisional data for the corresponding period in preceding years—Continued

State and period	All causes, rate per 1,000 population (annual basis)	Births (exclusive of stillbirths) per 1,000 population (annual basis)	Rate per 1,000 live births: Total infant mortality	Rate per 1,000 live births: Maternal mortality	Death rate per 100,000 population (annual basis): Typhoid fever (1, 2)	Measles (7)	Scarlet fever (8)	Whooping cough (9)	Diphtheria (10)	Influenza (11)	Acute poliomyelitis and polioencephalitis (16)	Encephalitis, epidemic or lethargic (17)	Epidemic cerebrospinal meningitis (18)	Tuberculosis, all forms (23–32)	Cancer, all forms (45–53)	Diabetes (59)	Cerebral hemorrhage, apoplexy (82a, b)	Diseases of the heart (90–95)	Pneumonia, all forms (107–109)	Diseases of the digestive system (115–129)	Diarrhea and enteritis under 2 years (119)	Nephritis (130–132)	All accidents (176–195, 201–214)	Automobile accidents (206, 208, 210)
Maryland:																								
1939	14.4	17.6	55	2.7	0.5	2.6	0.5	1.4	2.4	23.0	[6]	0.7	1.2	80.9	138.1	32.6	122.6	398.9	136.5	52.2	4.8	153.2	73.3	18.7
1938	13.4	17.7	56	2.9	.2	.7	1.2	4.6	1.0	16.1	[6]	1.9	1.2	81.4	129.8	32.8	101.9	366.8	135.1	56.1	3.6	139.0	67.4	21.9
1937	15.4	16.6	72	4.4	.7	4.3	1.2	7.0	2.4	47.8	.5	2.2	5.1	84.8	127.8	30.2	128.3	366.9	194.4	57.2	6.0	163.5	84.5	31.9
Michigan:																								
1939	12.4	18.4	53	4.0	.3	1.3	2.5	1.8	1.0	40.6	[6]	.2	.4	40.6	123.4	33.2	101.6	352.6	111.5	62.1	4.4	67.2	67.6	20.9
1938	10.9	18.6	48	3.8	.9	3.1	3.6	1.5	1.1	11.7	[6]	.5	.5	39.1	116.6	28.5	96.0	306.9	80.6	59.1	5.3	59.9	62.2	21.1
1937	12.4	17.4	61	4.1	.2	[6]	5.5	3.7	1.2	50.2	[6]	.2	1.2	44.2	116.7	28.8	95.2	307.4	186.4	64.8	4.1	65.8	73.5	29.4
Minnesota:[7]																								
1939	10.5	18.0	46	2.6	.2	6.5	.7	.5	1.2	15.7	[6]	.5	.5	32.8	139.2	28.6	101.6	270.1	84.3	60.2	3.2	45.9	56.1	14.1
1938	10.0	17.4	41	3.9	.2	[6]	2.6	2.3	.2	15.3	.7	.2	.7	26.2	141.5	27.9	93.9	251.4	93.2	54.5	2.3	49.2	55.7	19.0
1937	12.4	16.5	58	4.5	.2	.5	2.6	1.4	.9	104.0	[6]	.9	1.9	35.0	139.3	27.5	102.9	292.5	154.0	56.0	.7	46.9	63.7	14.2
Missouri:																								
1939	12.0	17.0	52	3.4	1.1	.4	1.8	2.1	2.7	30.1	.5	.6	1.0	48.9	126.9	27.3	98.6	272.0	130.0	59.2	5.7	116.4	66.3	20.9
1938	12.3	15.9	57	4.5	2.0	9.6	4.2	7.2	4.6	30.0	.7	.4	.8	51.8	122.6	24.9	91.9	287.8	141.2	57.3	4.2	107.1	72.5	26.6
1937	14.1	12.3	83	8.0	3.7	[6]	3.7	3.2	3.0	90.7	.4	.9	2.0	61.9	121.4	26.5	101.8	291.5	210.8	60.1	5.1	114.2	86.0	31.1
Montana:																								
1939	11.7	18.3	66	4.8	2.2	8.8	1.5	8.1	.7	35.4	[6]	2.2	.7	39.1	112.8	16.2	94.4	258.1	118.7	65.6	5.2	62.7	88.5	19.2
1938	10.7	19.3	45	5.0	.7	1.5	2.2	5.2	1.5	33.5	[6]	[6]	1.5	46.1	84.8	24.6	93.8	224.7	116.8	55.8	3.7	76.5	77.4	19.3
1937	14.1	18.5	69	3.7	3.0	.8	6.8	1.5	3.8	181.3	[6]	1.5	3.0	45.1	101.6	21.1	113.6	218.2	205.4	60.9	.8	72.2	79.0	15.0
Nebraska:[7]																								
1939	9.7	16.0	36	2.8	1.4	1.4	.9	.5	.9	25.8	[6]	1.4	.5	15.4	107.8	27.2	95.6	159.4	99.2	56.2	1.8	68.8	62.5	13.6
1938	9.0	16.4	40	3.3	.9	[6]	.5	2.3	1.4	20.0	[6]	[6]	.9	12.7	117.0	25.4	78.5	233.6	84.8	45.8	2.7	58.5	50.3	15.9
1937	13.4	15.9	74	5.4	.5	[6]	5.9	3.6	1.4	190.0	.5	[6]	.9	23.6	116.6	37.6	99.8	272.6	161.0	56.5	3.2	75.7	64.4	18.6
Nevada:																								
1939	12.6	18.8	55	2.1	[6]	3.9	[6]	[6]	[6]	15.7	[6]	[6]	[6]	55.1	129.9	3.9	86.6	334.7	165.4	39.4	[6]	70.9	78.7	15.7
1938	12.2	17.0	56	4.7	4.0	[6]	[6]	[6]	4.0	4.0	[6]	[6]	[6]	87.5	99.4	15.9	71.6	302.2	163.0	31.7	[6]	79.5	107.4	31.8
1937	14.5	12.2	75	3.2	[6]	[6]	4.0	[6]	[6]	28.1	[6]	[6]	[6]	72.2	100.4	4.0	100.4	273.0	212.8	44.2	8.0	80.2	136.5	35.1

New Jersey:																							
1939	11.5	12.9	47	3.5	.2	(6)	1.3	2.1	.7	13.9	.2	.7	.6	44.7	132.0	38.1	91.7	389.0	87.7	55.9	3.3	78.2	48.0	16.5
1938	10.7	12.9	43	3.3	.1	1.2	.4	1.3	1.3	8.1	.1	.7	1.0	45.6	122.7	30.8	85.0	343.6	89.8	57.8	3.4	80.1	51.2	18.5
1937	11.7	12.2	51	4.4	.3	1.8	.4	1.7	.8	26.3	.1	.8	2.0	50.7	119.1	35.1	82.9	362.3	119.7	54.6	3.4	80.0	63.3	25.9
New Mexico:																								
1939	16.6	35.3	125	7.0	1.0	1.9	1.9	9.5	9.5	64.7	1.0	(3)	2.9	100.9	64.7	10.5	49.5	155.2	227.5	77.1	10.5	72.4	89.5	45.7
1938	13.6	32.6	94	4.7	3.8	23.1	(6)	26.9	4.8	32.7	1.0	(6)	1.0	94.2	59.6	3.8	55.7	156.6	148.0	72.1	14.4	58.6	87.5	34.6
New York:																								
1939	12.9	14.6	46	3.2	.2	.5	.8	1.1	.3	8.9	(5)	.3	.7	52.6	158.7	45.2	74.1	439.8	102.3	61.5	5.3	82.1	58.4	13.5
1938	12.3	14.4	45	4.0	.2	.2	.7	1.2	.5	6.9	.1	.7	1.1	52.3	156.8	38.7	72.4	399.8	99.1	61.7	5.2	81.3	61.4	17.3
1937	13.9	14.2	54	4.4	.3	.5	1.4	1.5	.9	29.1	(5)	.7	2.1	64.5	154.1	44.3	87.0	413.8	160.5	71.9	6.4	86.4	68.4	20.3
North Carolina:																								
1939	9.6	21.3	65	5.5	.6	2.8	.7	7.2	4.6	34.4	.1	.3	.5	53.4	58.3	5.6	87.4	166.9	102.6	46.7	6.1	86.9	58.5	21.7
1938	10.3	22.4	63	6.3	.7	10.0	.6	4.7	6.3	29.4	.2	.7	1.6	54.0	54.2	11.6	86.5	174.1	128.0	49.9	6.0	95.6	62.3	21.3
1937	10.4	22.2	70	5.8	1.0	.9	.6	2.6	4.9	58.0	.3	.9	1.2	61.8	52.4	11.4	84.5	168.3	131.0	44.6	5.3	88.3	69.1	26.1
North Dakota:																								
1939	8.2	19.4	59	3.8	.6	6.3	2.9	2.9	.6	30.3	(6)	1.1	1.1	22.3	87.4	29.1	71.4	189.6	78.3	45.7	8.0	45.7	29.1	7.4
1938	7.3	18.9	44	1.2	(6)	1.1	1.7	12.0	.6	10.3	(6)	(6)	1.2	20.6	84.2	22.9	59.0	146.6	66.4	49.8	5.2	46.4	42.4	10.9
1937	9.6	19.5	69	6.2	(6)	(6)	1.7	(6)	1.7	68.9	(6)	.6	4.0	30.4	90.8	24.7	87.3	181.5	113.7	53.4	12.1	41.4	53.4	9.8
Ohio:																								
1939	13.1	14.4	55	4.0	.3	.2	2.0	1.3	1.5	40.1	.1	.9	.4	46.7	134.2	33.6	127.6	351.9	112.7	61.8	3.4	90.2	76.7	23.8
1938	12.0	16.1	49	3.5	.4	5.1	2.1	2.0	1.6	20.2	.2	.4	1.1	48.8	127.6	28.2	112.0	300.7	96.0	57.3	3.5	83.1	79.8	23.3
1937	13.8	14.8	61	6.0	.6	.7	3.1	4.0	1.9	78.6	.2	1.1	2.2	55.6	112.2	32.4	124.0	325.3	148.0	62.5	4.3	88.5	94.7	37.8
Oklahoma:																								
1939	9.5	17.0	63	4.1	2.2	6.0	1.6	1.9	3.3	43.7	.5	.6	.2	42.1	69.7	15.7	82.9	165.2	118.3	42.9	4.1	61.3	100.8	21.8
1938	9.0	18.5	49	4.7	2.2	1.1	1.4	9.8	7.0	34.6	1.7	.2	1.7	54.1	75.4	14.4	75.4	126.8	96.1	50.8	6.0	66.6	64.4	17.4
1937	10.6	16.1	72	7.2	1.9	1.0	2.1	1.6	3.3	121.6	1.0	.3	4.5	55.7	70.8	15.1	73.5	153.3	150.1	53.0	2.2	71.1	61.9	22.6
Oregon:																								
1939	12.2	15.1	46	2.1	.4	(6)	.4	.8	(6)	14.8	.4	.8	.8	28.8	135.7	31.1	131.0	307.6	76.6	35.0	.4	136.5	77.0	26.1
1938	12.2	15.7	38	3.8	(6)	(6)	1.6	1.6	1.2	16.5	.8	2.7	1.2	28.2	131.3	25.1	107.4	299.0	81.9	45.5	1.2	120.3	70.9	17.6
1937	14.5	14.8	49	4.3	.8	.4	2.4	3.2	.4	98.7	(5)	.8	.8	39.5	126.8	28.4	114.9	351.5	122.4	63.6	.4	126.8	74.6	24.1
Pennsylvania:[7]																								
1939	11.9	15.7	53	3.5	.8	.2	.9	2.0	1.2	18.0	.1	.6	.9	41.1	121.9	39.4	93.4	364.9	82.9	53.0	4.9	94.5	49.8	12.8
1938	12.0	16.3	53	3.6	.5	4.9	2.0	1.6	1.8	20.4	(5)	.8	.9	42.1	118.1	37.4	89.6	347.4	98.0	55.5	4.5	85.3	60.6	16.8
1937	13.7	15.5	65	5.3	.4	.4	2.7	3.8	1.8	92.5	.1	.9	2.2	54.2	114.1	37.9	87.9	360.1	148.9	55.7	4.0	98.3	61.9	19.4
South Carolina:[7]																								
1939	8.8	16.1	79	7.7	2.6	.3	.7	7.2	1.6	41.8	.7	.3	1.0	37.2	39.5	12.7	94.3	157.4	98.3	13.7	1.3	77.0	55.5	22.5
1938	9.8	16.4	86	7.4	1.6	7.2	1.6	8.5	1.3	68.6	.3	.3	1.0	36.8	43.3	11.5	82.4	173.0	121.4	12.1	.3	85.0	50.2	21.3
1937	8.9	14.6	107	9.9	1.6	1.0	(6)	1.0	4.9	65.4	(6)	.6	1.6	37.8	40.4	9.2	80.2	150.6	110.8	11.8	3.9	69.1	42.4	21.4
Tennessee:																								
1939	9.5	14.2	64	5.7	.7	2.1	.4	3.6	2.2	56.7	.6	.1	1.1	71.8	63.2	11.6	76.4	169.4	107.9	52.0	3.3	56.0	51.6	16.0
1938	9.5	14.7	63	6.9	.7	10.7	.6	6.2	3.5	46.9	.7	.6	2.5	74.4	64.0	10.8	79.7	152.6	121.0	47.2	2.6	59.7	57.4	19.4
1937	11.2	14.2	78	9.2	1.1	1.1	1.0	3.8	4.9	117.2	.3	.7	4.0	85.5	61.7	12.9	81.6	164.9	164.7	47.8	2.6	60.6	63.5	22.6
Texas: 1939	9.6	16.4	71	5.6	1.6	1.4	.6	3.1	3.4	51.4	.4	.1	.5	58.4	68.0	12.9	67.8	181.6	115.7	(6)	9.1	62.8	57.5	20.9
Utah:																								
1939	9.2	24.4	49	3.2	(5)	(6)	(6)	(6)	(5)	14.0	(5)	.8	2.3	21.8	94.2	14.8	42.8	269.3	72.4	66.2	.8	69.3	57.6	20.2
1938	9.5	24.3	52	3.2	(5)	3.1	3.1	4.7	(5)	21.0	(5)	(5)	4.7	21.0	84.2	22.6	50.7	245.7	88.9	60.0	3.9	56.9	81.9	34.3
1937	10.9	21.7	45	5.0	(5)	.8	3.1	3.9	.8	67.2	.8	1.6	3.1	21.9	93.0	22.7	67.5	275.8	96.1	73.4	3.1	57.0	74.2	27.4

See footnotes at end of table.

Provisional mortality from certain causes in the first 3 months of 1939, with comparative provisional data for the corresponding period in preceding years—Continued

State and period	All causes, rate per 1,000 population (annual basis)	Births (exclusive of stillbirths) per 1,000 population (annual basis)	Rate per 1,000 live births		Death rate per 100,000 population (annual basis)																	
			Total infant mortality	Maternal mortality	Typhoid fever (1, 2)	Measles (7)	Scarlet fever (8)	Whooping cough (9)	Diphtheria (10)	Influenza (11)	Acute poliomyelitis and polioencephalitis (16)	Encephalitis, epidemic or lethargic (17)	Epidemic cerebrospinal meningitis (18)	Tuberculosis, all forms (22–32)	Cancer, all forms (45–53)	Diabetes (59)	Cerebral hemorrhage, apoplexy (82a, b)	Diseases of the heart (90–95)	Pneumonia, all forms (107–109)	Diseases of the digestive system (115–129)	Diarrhea and enteritis under 2 years (119)	Nephritis (130–132)	All accidents (176–195) 201–214)	Automobile accidents 206, 208, 210)
Vermont:																								
1939	12.4	14.5	37	5.0	(6)	(6)	1.0	5.2	1.0	38.6	(6)	1.0	1.0	37.5	139.7	34.4	139.7	367.0	146.0	44.8	5.2	77.1	64.6	20.9
1938	12.3	14.8	55	3.5	(6)	10.5	(6)	4.2	(6)	24.2	(6)	(6)	(6)	45.2	121.9	30.5	120.0	339.4	134.5	65.1	2.1	87.2	49.4	12.6
1937	13.5	11.8	58	15.2	2.1	(6)	(6)	2.1	2.1	93.2	(6)	(6)	1.0	54.0	136.6	19.1	119.6	349.4	151.4	60.4	5.3	74.1	70.9	16.9
Virginia:																								
1939	11.5	17.0	77	6.6	.3	.4	.4	6.0	3.7	47.2	.1	.4	1.6	61.9	73.6	20.2	112.2	252.9	111.4	40.0	2.1	89.4	64.7	21.1
1938	11.2	17.5	73	5.6	1.2	6.2	.3	7.7	3.3	36.5	(6)	.1	2.2	68.9	71.7	18.7	94.1	245.2	112.6	40.9	3.6	85.7	59.1	21.2
1937	12.7	17.5	81	5.5	.9	2.8	.6	6.6	3.1	107.0	.6	.9	7.2	67.1	69.5	18.1	99.7	244.6	191.2	39.3	2.4	96.5	59.0	21.9
Washington:																								
1939	11.8	13.7	51	3.5	.7	.7	.5	.2	.7	15.2	1.0	.7	1.0	40.7	140.3	28.5	112.8	313.4	83.9	61.0	2.2	72.8	80.5	21.9
1938	11.6	14.1	43	4.0	.5	.5	.5	3.2	.7	22.1	.2	2.2	1.2	44.7	137.4	27.4	114.6	293.8	92.5	56.6	.5	75.3	69.9	24.0
1937	13.1	12.6	56	5.2	1.0	2.0	2.2	.2	1.2	75.1	.2	2.0	1.5	45.0	126.2	28.9	114.6	321.4	121.3	57.0	1.0	88.5	80.0	23.0
Wisconsin:[7]																								
1939	11.4	15.1	59	3.7	(6)	1.3	1.7	1.7	.6	22.2	.2	.4	.4	27.4	136.9	34.5	101.1	351.0	81.2	(8)	4.8	63.6	60.1	16.3
1938	10.6	16.1	49	2.4	(6)	.6	1.5	1.9	.8	11.6	.4	.2	.2	27.6	128.5	30.3	101.0	299.3	90.0	(8)	2.9	69.8	56.0	13.5
1937	13.2	15.4	65	4.0	.2	(6)	5.1	.6	1.3	99.8	(6)	.4	1.1	39.1	133.8	36.6	110.2	339.6	129.6	(8)	3.8	80.1	67.7	18.8
Wyoming:																								
1939	9.8	19.1	59	3.6	1.7	1.7	(6)	1.7	3.4	25.5	(6)	(6)	(6)	20.4	88.2	15.3	76.4	249.4	84.8	49.2	1.7	83.1	83.1	18.7
1938	9.3	16.9	60	7.1	(6)	(6)	(6)	29.1	1.7	27.4	(6)	(6)	1.7	18.8	97.5	15.4	61.6	215.6	104.4	71.9	8.6	47.9	85.6	34.2
1937	12.6	17.9	71	8.7	1.7	1.7	12.1	3.5	(6)	158.8	1.7	1.7	5.2	15.6	57.0	6.9	65.6	253.7	201.9	70.8	13.8	43.3	89.7	20.7

[1] Includes all States with data for the 3-month period of 1937, 1938, and 1939. The District of Columbia is included as a State. Estimated population July 1, 1939: 83,599,000.
[2] These data are taken from the April 1938 and 1939 Statistical Bulletins published by the Metropolitan Life Insurance Co. All figures are provisional and are subject to correction, since they are based on provisional estimates of lives exposed to risk (17,700,000 persons in 1938). Data do not include all diseases reported to the Public Health Service.
[3] Excludes pericarditis, acute endocarditis, acute myocarditis, coronary artery diseases, and angina pectoris.
[4] Classified as diarrhea and enteritis, age not specified.
[5] Chronic nephritis (Bright's disease) only.
[6] No deaths reported. [7] January and February. [8] Data not available. [9] Less than 0.1 per 100,000 population.

THE INDUCTION OF CARDITIS BY THE COMBINED EFFECTS OF HYPERTHYROIDISM AND INFECTION [1]

By MARK P. SCHULTZ,[2] *Surgeon, United States Public Health Service*

Some observers conclude that patients with exophthalmic goiter and animals given toxic doses of thyroid hormone suffer extensive morphological cardiac damage. Others, on the basis of similar investigations, find that hyperthyroidism induces at the most very slight injury of this kind. In some accounts belonging to the former category, it is recorded that different types of infection were present during the period of hyperthyroidism.

The effect of chronic infection upon the cardiovascular system in thyroid-treated animals was, therefore, considered worthy of investigation. It was the purpose of the experiments reported here to study the morphological changes in the heart and aorta incident to chronic, focal, hemolytic streptococcus infection in rabbits receiving thyroxin and in guinea pigs fed desiccated thyroid. In order to investigate the pathogenesis of cardiovascular lesions induced in this manner, certain attributes of infection in thyroid-treated animals as indicated by the body temperature, variations in the erythrocyte sedimentation rate, and peculiarities of antibody responses were compared with those of infected, untreated animals. Inasmuch as a state of bacterial hypersensitivity may be followed by one of immunity in the type of focal infection employed, the influence of these two phases of infection was studied individually by rendering thyroid-toxic rabbits both hypersensitive and immune to bacteria. Further, an attempt was made to analyze the influence of increased metabolic rates upon the pathology of the heart and aorta during infection by investigating the effect of treatment with dinitrophenol under similar conditions.

REVIEW OF LITERATURE

The subject of cardiac pathology in exophthalmic goiter has been extensively reviewed by the following authors: Rautmann, 1915 (*1*); Wilson, 1923 (*2*); McEachern and Rake, 1931 (*3*); Baust, 1931 (*4*); Lewis, 1932 (*5*); Weller et al., 1932 (*6*); and de Chatel and Molnar, 1933 (*7*). Most investigators studying controlled series of *uncomplicated* cases have concluded that hyperthyroidism induces only slight permanent cardiac damage. These observers, however, occasionally encountered instances of extensive heart involvement for which no cause could be assigned.

On the other hand, there are numerous pathological descriptions, frequently of single cases, of severe carditis in association with exophthalmic goiter in which mention of concurrent infection is fre-

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quently omitted. The occurrence, however, of chronic bronchitis was noted in 1 of 2 such cases reported by Goodpasture (*8*) and of paratyphoid in 1 of the 2 described by Fahr (*9*); while pneumonia, erysipelas, or acute tonsillitis were present in single instances recorded by Ceelen (*10*), Loos (*11*), and Davis (*12*). Active infection was mentioned as a complication in 3 of 28 cases of hyperthyroidism in which extensive cardiac damage was described by Fahr and Kuhle (*13*). Others have reported similar findings without stating whether infection was present or not (*14, 15, 16, 17*). These observations suggest that, although morphological cardiac damage may not be detected in uncomplicated exophthalmic goiter, it may be induced by the coincident presence of infection.

More definitely suggestive evidence is afforded by studies of hyperthyroidism in different species of laboratory animals incident to the exhibition of thyroid-gland preparations, thyroxin, or inorganic iodine compounds. Some observers find the heart essentially negative to pathological examination in such experiments (*18, 19, 20, 21, 22*), while others describe extensive lesions without recording the presence or absence of associated infection (*15, 23, 24*). Heinlein and Dieckhoff (*25*), however, reported the occurrence of extensive morphological damage, without mentioning the frequency with which it developed, in a group of cats free from infection which had been given thyroxin for a long period of time.

Nevertheless, in certain reports, the occurrence of extensive cardiac lesions in experimental hyperthyroidism apparently can be correlated with a complicating factor of infection. Zalka (*26*) administered thyroxin to guinea pigs, cats, and rabbits. In the two species first named no evidences of infection were seen, and the hearts were found to be normal with the exception of 1 of 5 cats which developed definite myocarditis. On the other hand, 4 of the 7 rabbits examined had pneumonia, and extensive heart lesions developed in this species. Menne et al. (*27*) fed desiccated thyroid to a group of rabbits of which some members were found to harbor spontaneous infections. They discovered definite cardiac lesions in 90 percent of their animals but noted that "the latter were most pronounced in the hearts of rabbits with abscesses in the pleural cavities." Hashimoto (*28*) treated rats similarly and found extensive myocardial damage in many animals. Pneumonia, however, was present in 85 percent of them. Rake and McEachern (*29*), in attempting to eliminate the factor of infection, selected animals only after preliminary periods of observation during which the rate of weight gain and fluctuations of body temperature were noted. The 44 rabbits and 17 guinea pigs to which they administered thyroxin were, therefore, presumably healthy. Cardiac lesions developed in only 5 of the guinea pigs, which were found to be suffer-

ing from bronchisepticus pneumonia; there were no signs of infection in other animals.

In these last four investigations the influence of infection in inducing myocardial damage in animals with thyroidism was differently assessed. Zalka concluded that the occurrence of pneumonia in thyroxin-treated rabbits developing myocarditis was merely fortuitous because cardiac lesions also appeared in 3 rabbits and 1 cat without any demonstrable evidence of an associated infection. Hashimoto, probably on insufficient grounds, likewise considered that pneumonia was not a determining factor. He observed that pneumonia might be acute and extensive in rats dying early in the course of thyroid feeding and only slight myocardial damage be present. In animals observed longer, on the other hand, no acute pneumonia was found, but extensive cardiac lesions developed. He states,

> It is evident, therefore, that an acute pneumonia appearing in the thyroid-fed animals has little or no part in causing the myocarditic lesions * * *. The same is true of chronic pneumonia inasmuch as no myocarditic lesion was found in the control animals, although 85 percent of them showed chronic bronchopneumonia.

These conclusions appear unjustified for two reasons: (1) Since acute pneumonia was present in animals dying early (the average survival period was only 11 days), sufficient time may not have elapsed for the development of cardiac lesions; (2) the fact that chronic pneumonia did not incite cardiac lesions in controls has little bearing upon the question of its effect in animals with hyperthyroidism. Menne et al. stated that in some thyroid-fed rabbits which they observed, "* * * infection seems to have augmented the destruction of cardiac muscle," but that "there was no way of determining the relationship of these two conditions" (hyperthyroidism and infection). Rake and McEachern considered the possibility that hyperthyroidism might increase susceptibility of the heart to damage in the presence of infection but quoted statements which would indicate that uncomplicated bronchisepticus infection of the type they encountered is capable of inducing myocardial damage such as that observed.

Obviously, therefore, even though studies of cardiac pathology in exophthalmic goiter have not demonstrated that morphological lesions are incident to an associated infection, they are not incompatible with such a conception. Similarly, although such an effect of infection in experimental hyperthyroidism has not been proved, the results of several investigators suggest that it may be susceptible of demonstration. Such a relationship has been pointed out with respect to liver damage in experimental hyperthyroidism by Haban (*30*), who found extensive lesions only when there was an associated infection.

METHODS

Animals.—The rabbits were hybrids of English, lilac, and Havana varieties. All were males within 2 weeks of the same age, weighing between 1,200 and 1,500 grams at the start of each experiment. The guinea pigs were males of mixed stock obtained from a dealer and weighed approximately 400 grams.

Infection.—The rabbits were infected with a group C hemolytic streptococcus strain (K158b) originally isolated from a spontaneously infected rabbit. Cultures, grown 18 hours at 37° C. in "streptolysin broth" (*31*), were injected subcutaneously in axillary or inguinal regions at weekly intervals. The inoculations were arranged so that the same region was reinfected at approximately monthly intervals. The dose was gradually raised from 0.1 cc. to as high as 10.0 cc. in animals surviving for long periods. When quantities larger than 0.5 cc. were given, the culture was centrifuged and resuspended in sterile physiological saline. This treatment resulted in each infected rabbit having constantly one or more abscesses in groin or axilla of approximately 2 to 5 cm. in diameter. These animals were bled fortnightly for serum or determination of the erythrocyte sedimentation rate. Their rectal temperatures were taken daily, and they were weighed three times a week.

Guinea pigs were infected with a group C hemolytic streptococcus (J20) originally isolated from a guinea pig with spontaneous lymphadenitis. Cultures were grown in the manner described above for strain K158b, while chronic infection, maintained in a manner similar to that described for rabbits, was manifest by similar abscesses. The dose of culture for guinea pigs, however, was uniformly 0.1 cc., and local lesions occasionally broke down with the discharge of yellow pus. Among the guinea pigs, spontaneous hemolytic streptococcus lymphadenitis was frequently present. Infections of this type were present in one group selected for study. Other guinea pigs were examined with care clinically and pathologically to exclude the presence of this complication.

Bacterial hypersensitization.—A strain of indifferent streptococcus (Q155) was employed. Each rabbit to be sensitized received 0.01 cc. of broth culture intracutaneously daily for 22 days and thereafter an equivalent dose twice weekly until the experiment was terminated on the sixtieth day. Cultures were diluted with physiological saline so that 0.1 cc. volume was injected. About the first, fourteenth, twenty-first, and sixtieth days, using small calipers, two diameters and the height of cutaneous lesions resulting from test inocula of 0.01 and 0.001 cc. were measured in millimeters 24 and 48 hours after the intracutaneous injections of culture. The relative degrees of cutaneous hypersensitivity are, therefore, expressed by the average diameter

and estimated height of the lesion resulting from these test doses at the stage of maximum development. The hair was removed with clippers, and areas on the hind legs were used for sensitizing injections, while freshly clipped regions on the sides were utilized for the test reactions. Specimens of blood for serum were obtained at the start of the experiment and on the fourteenth, twenty-first, and sixtieth days.

Intravenous injection of culture.—A Group A strain of hemolytic streptococcus (London MA) was utilized. Rabbits were injected on 4 succeeding days each week, with vaccine during the first 2 weeks and with living broth culture during the third to seventh weeks, inclusive. Each dose of vaccine was the equivalent of 1.0 cc. of culture; and the doses of culture were gradually raised from 0.5 cc. to 3.0 cc. The cultures were centrifuged and resuspended in sterile physiological saline, and vaccine was prepared by adding formalin in a final strength of 0.2 percent to a suspension concentrated to one-tenth the original volume in sterile physiological saline which was then allowed to remain 48 hours in the refrigerator. Specimens of blood were collected weekly from the rabbits receiving this treatment.

The administration of thyroxin.—A weighed quantity of thyroxin [3] moistened with a drop of 5 percent sodium hydroxide was rubbed into a paste and dissolved in sterile physiological saline to make a 0.1 percent solution. This was injected intravenously into rabbits thrice weekly in doses sufficient to prevent weight gain in infected animals. Although at the time of each injection all rabbits received the same quantity of thyroxin, it was necessary to vary the dosage level occasionally to obtain a continuously uniform effect. The variation in susceptibility to this hormone which necessitated change in dosage was apparently correlated with fluctuations in the atmospheric temperature; indeed, an increased susceptibility to thyroxin at higher temperatures has been demonstrated (*32*). The weekly dose per rabbit varied between 0.6 and 1.2 mg. of thyroxin.

Administration of desiccated thyroid.—Desiccated thyroid [4] was administered to guinea pigs by mouth thrice weekly in the form of a 12.5 percent suspension in water. The weekly dose, adjusted according to the criteria used for rabbits, varied between 0.3 and 0.75 gm.

Diet.—All animals were given "Purina complete" pellets, oats, hay, cabbage, and water daily. Appetite and thirst were increased in thyroid- and dinitrophenol-treated animals; and, although water was freely supplied to all animals, the food intake was regulated as follows: The approximate amount of each of the several articles of diet consumed by the controls during the preceding 24 hours was estimated, and the average amount consumed by these controls was fed to

[3] "Thyroxin Synthetic," Hoffman La Roche.

[4] "Thyroid glands desiccated," standardized to contain 0.3 percent of iodine in organic combination.

every animal. Each was offered at least 50 gm. of cabbage each day. Under these conditions, treated animals almost invariably ate all that was offered them. All were weighed three times a week.

Administration of dinitrophenol.—Rabbits were injected intravenously twice daily with a sterile aqueous solution containing 0.5 percent alpha 1-2-4 dinitrophenol in a total daily dose of 30 mg. per kg. of body weight. Individual rabbits varied greatly in their susceptibility to this drug, for 3 of 13 died after 1 or 2 days' treatment. The survivors, however, reacted in a most uniform manner and either failed to gain weight or lost slightly during the entire period of observation, even though the dosage was maintained at a uniform level. Because this diluted solution was very irritating it was necessary to avoid allowing any of it to escape into the tissues surrounding the vein injected.

Agglutinin titration.—Agglutinin titrations were performed in the usual manner; results were read after the tubes had remained in an incubator at 56° C. for 1 hour. To insure uniform results, all sera in each experiment were titrated simultaneously.

Precipitin titration.—In one experiment the sera were tested for the presence of anti-"M" precipitins. This fraction of the bacterial cell was prepared from hemolytic streptococcus group A strain "London MA"; and the precipitin reactions were performed following the method described by Lancefield (*33*).

Erythrocyte sedimentation rate.—The erythrocyte sedimentation rate in rabbit blood was determined as follows: Four parts of blood obtained from the marginal ear vein were diluted with one part of 3.8 percent sodium citrate solution. The mixture was then drawn up into a tube of 3.0 mm. internal diameter until a column 200 mm. high was obtained and the tube stood upright on plasticene. The length of the clear layer of serum was measured after the tubes had remained in a vertical position for 1 hour at room temperature.

Pathologic technique.—Animals which did not succumb were exsanguinated. In all cases all the organs and subcutaneous areas were examined in the gross with particular attention to the presence of infection, either spontaneous or induced. The heart and aorta of all animals were examined microscopically and also the lungs of all the guinea pigs. These organs were fixed in Zenker's solution, sectioned in paraffin, and stained with eosin-methylene blue and Van Gieson elastica stain. Before being embedded, each aorta was rolled so that the entire longitudinal extent was represented in each section. Several sections were made from each of the two blocks of the heart—one included the aortic valve and the other the mitral valve (occasionally the tricuspid) and portions of both ventricles.

RESULTS

EXPERIMENT 1. CHRONIC HEMOLYTIC STREPTOCOCCUS INFECTION IN RABBITS TREATED WITH THYROXIN

Sixty-nine rabbits were separated into 5 groups and treated as follows:

Group A.—Nine untreated served as controls.

Group B.—Eighteen were subjected to chronic hemolytic streptococcus infection only.

Group C.—Ten received intravenous injection of thyroxin only.

Group D.—Twenty-eight were subjected to the continued effects of chronic hemolytic streptococcus infection and intravenous thyroxin injections after the latter had been given for 3 to 140 days.

Group E.—Four were given thyroxin intravenously while chronic hemolytic streptococcus infection was present; but the latter had been maintained for 125 to 130 days before thyroxin was exhibited.

PATHOLOGIC CHANGES

Group A, controls.—The 9 control rabbits gained weight uniformly at an average rate of 6 grams per day and were apparently healthy. At autopsy no macroscopic pathological changes were evident. The hearts were normal microscopically except in two instances, where the lesions consisted of small, compact, bacteria-free collections of cells, occasionally in perivascular areas of the myocardium, but frequently subendocardial in location. These cell collections consisted chiefly of small lymphocytes with an occasional pseudoeosinophile. They were quite isolated and only one or two were present in any section of the heart. The myocardial muscle fibers showed no evidence of damage even in areas adjacent to the focal lesions.

Group B, infection only.—The 18 rabbits with chronic hemolytic streptococcus infection all developed abscesses in axillae and groins but gained weight at about the same rate as the controls, although the gain was temporarily arrested during periods of most active infection, and there was rapid antemortem loss in 5 which died. Four succumbed during the second, third, and fourth weeks of infection and one during the third month. In each of these rabbits the heart was the seat of acute, focal, purulent myocarditis and there were many circumscribed abscesses in which numerous gram positive cocci were present. Furthermore, in these animals macroscopic purulent lesions in other organs were frequently apparent. The remaining 13 members of this group were autopsied after 30 to 90 days of infection. Their organs were negative to gross examination, but in each animal there were 4 well-encapsulated, purulent abscesses in groins and axillae. Upon microscopic examination, cardiac lesions were observed in 5 of

the 13, a somewhat greater incidence than among the controls, and these lesions were somewhat more extensive and more numerous in the affected hearts. Furthermore, twice a slight degree of muscle fiber degeneration was evident adjacent to the cell collections, and in one instance small mononuclear cells were sparsely and diffusely scattered over wide areas in the myocardium. No correlation was apparent between the character or extent of the cardiac lesions and the duration of infection.

Group C, thyroxin only.—The 10 uninfected rabbits receiving thyroxin gained weight at about half the rate of the controls. There was some gain in body length evident, associated with moderate, progressive loss of subcutaneous fat. None of these animals died, and individuals were autopsied at intervals between 30 and 180 days' treatment. The only pathologic change macroscopically evident was a marked diminution or absence of fat. Four of the 10 hearts were normal on microscopic examination, but in the others the occurrence of damage could not be correlated with the duration of thyroxin administration. The microscopic cardiac lesions in 4 animals, examined after 40 to 80 days' treatment, were comparable in character and extent to those observed in the simple infected group (B). The heart of one animal after 80 days' treatment presented, in addition to round cell infiltration, a proliferation of fibroblasts in some of the small, focal, cellular accumulations, while the relative number of lymphocytes was reduced. In 3 rabbits examined between the 100th and 180th days of thyroxin injection only small patches of fibrosis were present, which corresponded in distribution and extent to the cell accumulations observed in the hearts of some members of the control (A) and the infected (B) groups, as well as to those in the hearts of 4 of the thyroxin-treated group (C) which had received injections for a shorter period of time.

Group D, infection and thyrotoxicosis.—Twenty-eight rabbits were subjected to the combined effects of chronic, focal, hemolytic streptococcus infection and the intravenous injection of thyroxin. Infection, however, was induced 3 to 140 days after the administration of thyroxin was begun. The trend of body weight was variable, but the final weight of those which survived for more than 1 month was usually about the same as at the beginning of the experiment. When infection was most active there were frequently sharp weight losses which were slowly regained in intervening periods. In the animals which succumbed after dissemination of the infection through the blood stream there was precipitous antemortem weight loss.

As indicated in table 1, 6 of the 28 rabbits in this group died with purulent focal, bacterial myocarditis, a relatively smaller number than among the members of group B with uncomplicated, chronic infection of the same type. In these animals neither the microscopic cardiac

changes nor the purulent lesions apparent in other organs on gross examination differed from those which developed in group B.

TABLE 1.—*Influence of thyroxin treatment and infection, separately and combined, upon cardiac pathology in rabbits*

Group	Treatment	Number of rabbits	Days duration of infection	Days duration of thyroxin treatment	Spontaneous death	Cardiac pathology
A	Untreated	9	None	None	None	All negative.
B	Infection only	5	14 to 70	do	5	Purulent carditis.
		13	90	do	None	All negative.
C	Thyroxin	10	None	30 to 180	do	Essentially negative.
D	Infection induced during course of thyroxin treatment.	4	10	18 to 125	All	Do.
		5	11 to 30	42 to 169	2 of 5	Severe nonpurulent carditis in over 50 percent.
		7	31 to 60	52 to 170	3 of 7	
		6	61 to 86	67 to 100	None	
		6	7 to 49	12 to 128	All	Complicated by purulent carditis.
E	Thyroxin treatment after infection established.	4	153 to 180	25 to 50	1 of 3	Essentially negative.

Of the remaining 22 rabbits in this group, 9 died, but, aside from abscesses in the groins and axillae, no purulent lesions were found in the internal organs and none was visible microscopically in the heart. On the other hand, rather extensive and characteristic microscopic, nonpurulent, cardiovascular lesions were found in 12 of the 22 animals, and in only 4 of the 19 which survived over one week were the hearts normal. The pathologic changes were as follows:

Myocardium.—The earliest changes observed are seen in rabbit No. 5 which had received thyroxin for 151 days but died 11 days after being infected. The essential lesion (fig. 7) was an edema involving the finest interstices between the muscle fibers, which were widened and occupied by material of an apparently "foamy" consistency. In these areas the capillaries were distended, presumably with serum, for they contained very few cells, and there was extensive perivascular edema. The staining of such areas with Van Gieson elastica (fig. 8) did not demonstrate the presence of any fibrillar structures in the interstices. This resembles closely the early changes in "serous myocarditis" described by Rössle (*16*) in the hearts of patients dying with Basedow's disease. Intermediate stages between this and established myocardial fibrosis, which have been demonstrated by Rössle, were not found in the material presented here.

In animals longer under the influence of thyroxin and infection there were extensive areas of myocardial fibrosis which consisted of stellate or elongated zones of proliferating fibroblasts rich in nuclear elements associated with degenerative changes in adjacent muscle fibers (fig. 9, rabbit No. 9, and fig. 10, rabbit No. 11, both infected for 30 days). The latter stained poorly with loss of internal structure and were of irregular size with indistinct outlines. In

animals still longer exposed to the influences of infection and thyrotoxicosis there were dense myocardial scars with few fibroblasts. Figure 11 (rabbit No. 15, infected for 60 days) shows perivascular scarring, while figure 12, representing an adjacent region stained with Van Gieson elastica, demonstrates the presence of adult connective tissue. Neighboring arterioles were usually thick-walled and showed considerable hypertrophy of the media (see figs. 13 and 14).

In thyroxin-treated rabbits infected for over 2 months, the areas of myocardial fibrosis were more dense (fig. 15, rabbit No. 20, infected for 86 days) and the muscle fibers remaining, while of irregular shape and distribution, no longer showed such extensive degenerative changes. In such areas multinucleated giant cells were frequently seen (fig. 16). These myocardial lesions are very similar to those which have been described in exophthalmic goiter and experimental hyperthyroidism.

Endocardium.—Lesions were rarer and less extensive in the endocardium than in the myocardium; however, they often appeared in conjunction with the latter. Apparently active processes, including fibrinoid degeneration and endocardial proliferation, were present only in rabbits which had been infected less than 50 days. The former change was rarely intense and not frequently present. A typical example appeared in the left ventricular endocardium of rabbit No. 13 which had been infected 49 days (fig. 3). In a small, sharply limited region, the endocardium was thick and possessed a fairly loose fibrillar structure with few deeply staining, homogenous, elongated nuclei. Irregular, fairly well defined areas, chiefly near the surface, were stained intensely with eosin and possessed a more homogenous, less fibrillar structure than adjacent parts. Here nuclei were more numerous, slightly larger, and not so intensely stained, many possessing eccentrically placed nucleoli. No lymphocytes, pseudoeosinophiles, or plasma cells were present. At times the missing endothelial surface was replaced by small fibrin clots. There was moderate interstitial edema of the subjacent myocardium. The appearance of perivascular lesions in this heart is described below.

Small areas of fibrinoid degeneration occurred in regions where active endocardial proliferation was apparently in progress. In the auricular endocardium of rabbit No. 7, which had been infected for 29 days, a localized area of endocardial proliferation developed in which such change was evident (fig. 4). The thickened endocardium and a small area of fibrinoid degeneration were of structure similar to that described in the preceding example. The endothelial layer, however, was intact and near the surface there was a more cellular area in which the fibrillar structure was disturbed. The nuclei in

this region were closely placed, large, irregular but not elongated, sharply outlined, and faintly but uniformly stained. In the adjacent endothelium nuclei were larger and more numerous than elsewhere, occasionally forming a double layer.

Fibrin deposits were sometimes extensive over areas denuded of endothelium. In such instances the subjacent endocardium was usually thickened and rich in nuclei, many of which sometimes appeared to be disintegrating. Such a lesion appeared in rabbit No. 6 which died after 11 days of infection (fig. 5). No bacteria were identified in such mural thrombi.

Areas of active endocardial proliferation, apparently not associated with other processes, were frequently observed. This occurred at the base of mitral valve in rabbit No. 5 which died after 11 days of infection. The considerably thickened mural endocardium was of fairly dense structure but presented no other abnormality except near the surface. A portion of this involved region is shown in figure 2. A definite endothelial layer was lacking but there was no fibrin deposit. Near the surface, deeply and uniformly stained, irregular nuclei were closely packed. No lymphocytes, pseudoeosinophiles, or plasma cells were present. Areas of myocardial edema in this heart, which have been described above, were subjacent.

In thyroxin-treated rabbits which had been subject to infection for longer than 50 days, no active endocardial lesions were seen. Occasionally regions of the endocardium, not sharply defined, were found to be considerably thickened and it is probable that these represented sites of earlier active inflammation.

Valves.—Slight valvular lesions varying little in their structure were not uncommon. The mitral valve of rabbit No. 11, which died after 43 days of infection, is a typical example (fig. 6). Subendocardial fibrinoid degeneration of slight degree was present along the superior surface with very little associated cellular reaction. There was little disturbance of structure except for an associated thickening of the endocardium and evidence of proliferation in the increased number of large, round or oval, faintly staining nuclei near the surface. No pseudoeosinophiles and no bacteria were found in the area.

Extensive valvular damage was rarely seen. However, near the base of the aortic valve in rabbit No. 20, which died after 86 days of infection, the lesion developed which is shown in figure 1.

Near the surface at the base of a thickened valve was a well-defined nodule, formed of cells with large, round or oval, deeply staining nuclei. Apparently an embolus had broken off from this area about 48 hours antemortem, when there was sudden hemiplegia due to plugging of a cerebral artery.

EXPLANATION OF PLATES[5]

FIGURE 1.—(A. M. M. 64844, rabbit No. 20. Received thyroxin for 100 days; infected for 86 days.) A somewhat nodular stromal proliferation in the aortic valve. The irregular cleft extending from the endocardial surface through the center of the lesion probably resulted from the displacement of a thrombus 48 hours before the death of the animal (see the text). Rather closely packed large, deeply staining nuclei surround an area of fibrinoid degeneration. (×175.)

FIGURE 2.—(A. M. M. 64849, rabbit No. 5. Received thyroxin for 151 days; infected 11 days.) Mural endocardial proliferation near base of mitral valve. The thickened endocardium possesses a fairly dense structure which is included here only in part. Deeply stained, irregular nuclei are closely packed near the surface but there are no infiltrating cells. (×655.)

FIGURE 3.—(A. M. M. 64870, rabbit No. 13. Received thyroxin for 170 days; infected 49 days.) Fibrinoid degeneration in the left ventricular endocardium over the interventricular septum. The endothelial surface is missing and replaced in some regions by small fibrin clots. Irregular areas in the endocardium near the surface stain rather intensely with eosin. Here, the nuclei are larger than elsewhere, more numerous, and less deeply stained. (×175.)

FIGURE 4.—(A. M. M. 64842, rabbit No. 7. Received thyroxin for 169 days; infected 29 days.) A localized area of auricular endocardial proliferation. The endocardium is thickened but the endothelial layer is intact. The fibrillar structure is disturbed in an area near the surface where large irregular, faintly stained nuclei are numerous. There is slight proliferation of the adjacent endothelium over a small area of fibrinoid degeneration. (×300.)

FIGURE 5.—(A. M. M. 64852, rabbit No. 6. Received thyroxin for 22 days; infected 11 days.) Ventricular endocardium. Nuclei are numerous in the thickened endocardium. The endothelial surface is replaced by fibrin clot. (×660.)

FIGURE 6.—(A. M. M. 64863, rabbit No. 11. Received thyroxin for 64 days; infected 30 days.) Mitral valve. There is some subendocardial fibrinoid degeneration with very little associated cellular reaction. At the base of the valve there is slight endocardial proliferation. (×175.)

FIGURE 7.—(A. M. M. 64861, rabbit No. 5. Received thyroxin for 151 days; infected 11 days.) Early "serous inflammation" in the myocardium. "Foamy" edema of the interstices between muscle fibers is present. (×650.)

FIGURE 8.—(A. M. M. 64869.) Section adjacent to that shown in figure 7, here stained with Van Gieson elastica. Fibrillar structures are not visualized in the interstices. (×650.)

FIGURE 9.—(A. M. M. 64860, rabbit No. 9. Received thyroxin for 42 days; infected 30 days.) Two areas of myocardial fibrosis in the left ventricle. In one probably younger area there is fibroblast proliferation and nuclei are numerous: in the other the connective tissue is more adult. Myocardial fibers adjacent to these regions show degenerative changes. (×100.)

FIGURE 10.—(A. M. M. 64853, rabbit No. 11. Received thyroxin for 64 days; infected 30 days.) An area of myocardial fibrosis in the left ventricle. The lesion is similar to that shown in figure 9, but there are strands of fibrosis and muscle fiber degeneration is more extensive.

FIGURE 11.—(A. M. M. 64845, rabbit No. 15. Received thyroxin for 74 days; infected 60 days.) Perivascular myocardial fibrosis. The connective tissue is of the adult type, and the walls of the vessel are apparently involved in the process. (×175.)

FIGURE 12.—(A. M. M. 64866.) Section adjacent to that shown in figure 11 here stained with Van Gieson elastica. (×185.)

FIGURE 13.—(A. M. M. 64862, rabbit No. 20. Received thyroxin for 100 days; infected 86 days.) Arterioles in myocardium. The vessels are hypertrophied and possess very muscular media. There is moderate myocardial fibrosis adjacent. (×175.)

FIGURE 14.—(A. M. M. 64865.) Section adjacent to that shown in figure 13 here stained with Van Gieson elastica. (×175.)

FIGURE 15.—(A. M. M. 64846, rabbit No. 20. Received thyroxin for 100 days; infected 86 days.) Myocardial fibrosis in the left ventricle. This probably represents a far advanced process for only a few muscle fibers remain and the connective tissue is of the adult type. (×175.)

FIGURE 16.—(A. M. M. 64864.) A high magnification of the region shown in figure 15. Multinucleated giant cells are present in the myocardial scar. (×605.)

FIGURE 17.—(A. M. M. 64851, rabbit No. 13. Received thyroxin for 170 days; infected 49 days.) Arteriole in the myocardium. Lymphocytes are diffusely scattered in the perivascular region. (×280.)

FIGURE 18.—(A. M. M. 64848, rabbit No. 5. Received thyroxin for 151 days; infected 11 days.) Arteriole in the myocardium. There is a small, irregular area of fibrinoid degeneration in the adventitia. The nuclei in this region are pyknotic and deeply stained. There is some localized endothelial proliferation but the media does not appear hypertrophic. (×355.)

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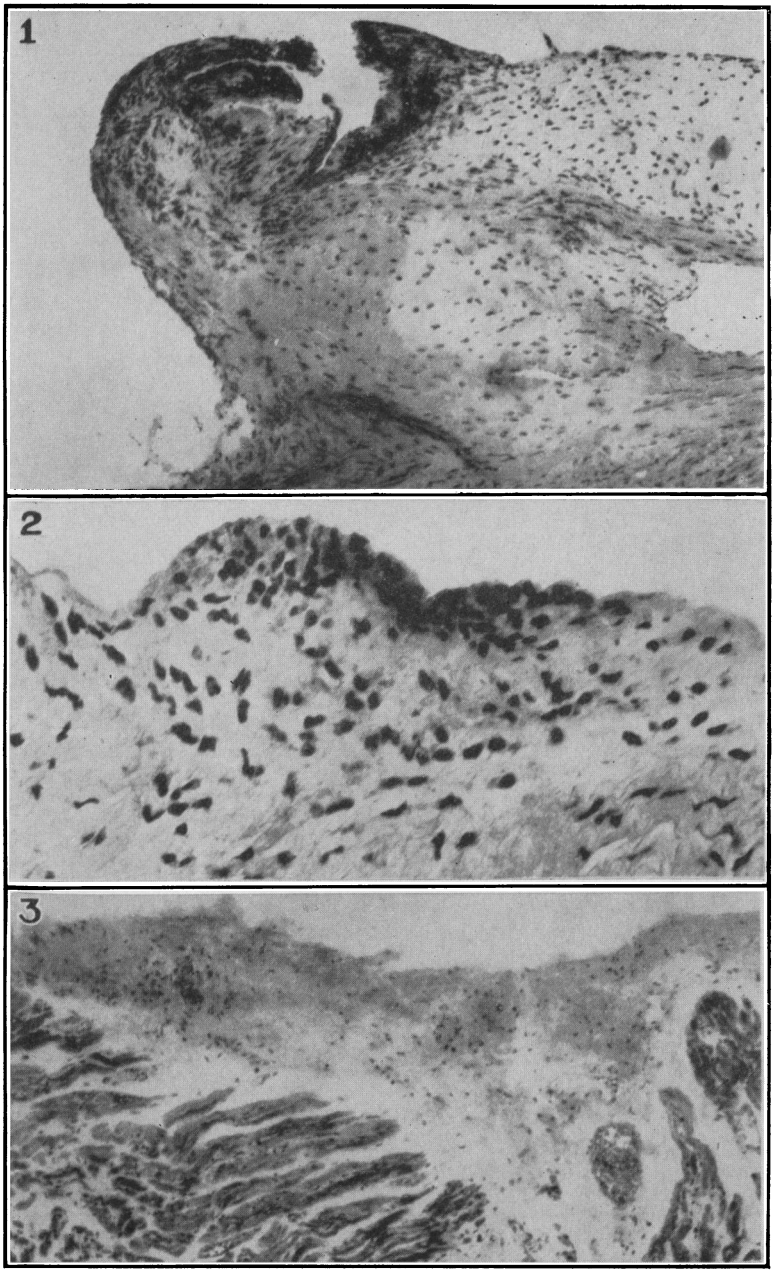
[5] The author wishes to thank the staff of the Army Medical Museum for preparing the photomicrographs from which these illustrations were reproduced. Figures here following the abbreviation, "A. M. M.," indicate the number of the original picture on file in the Museum. Unless otherwise noted, sections were stained with hematoxylin-eosin.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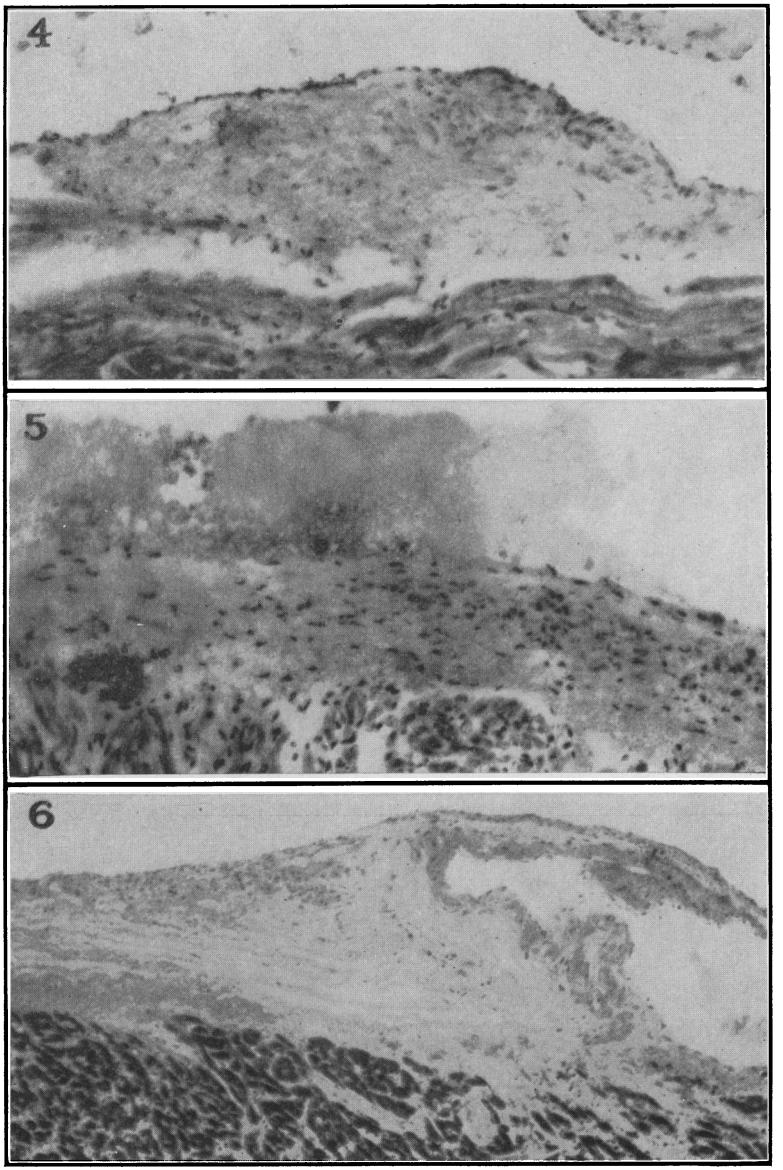
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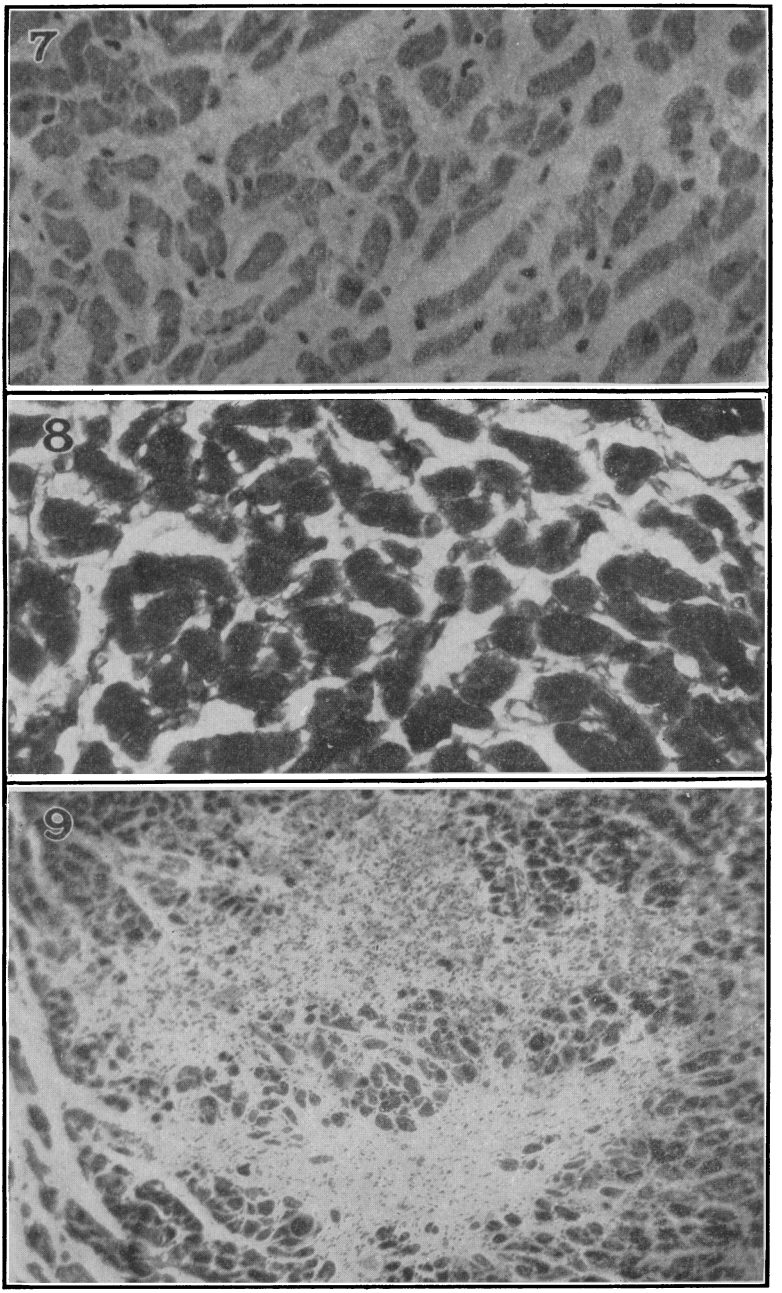
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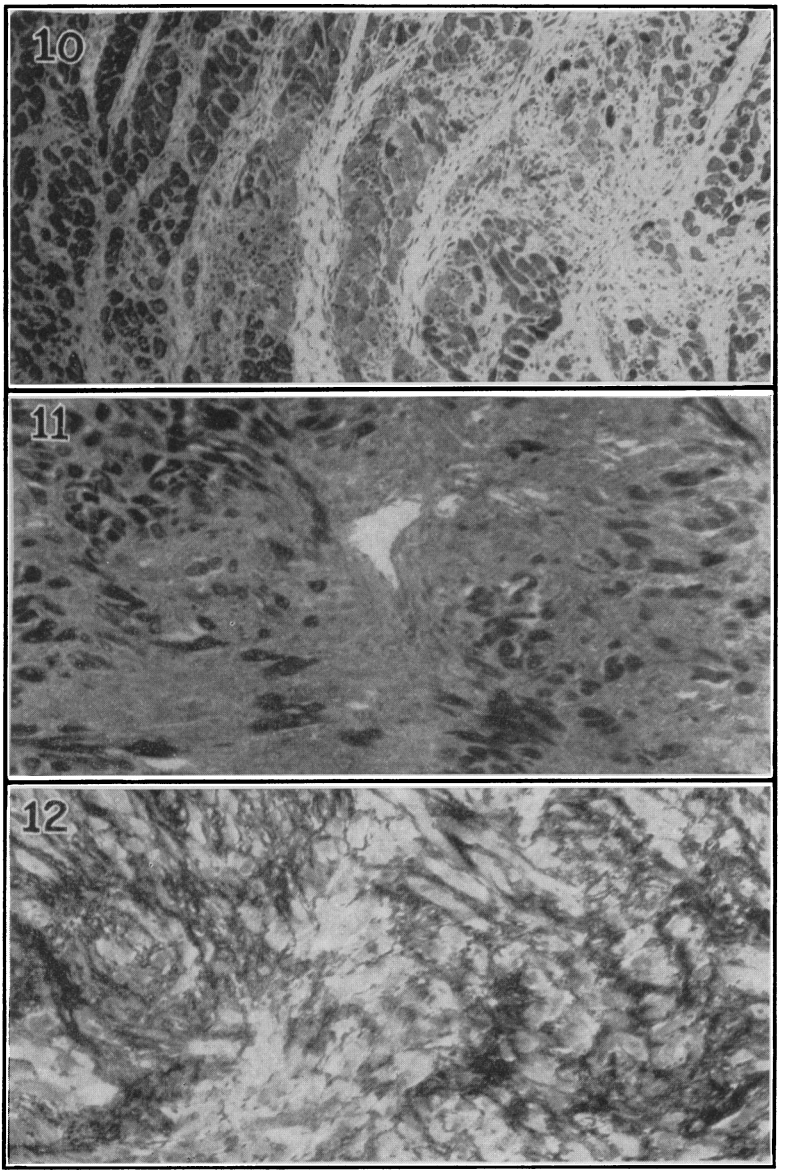
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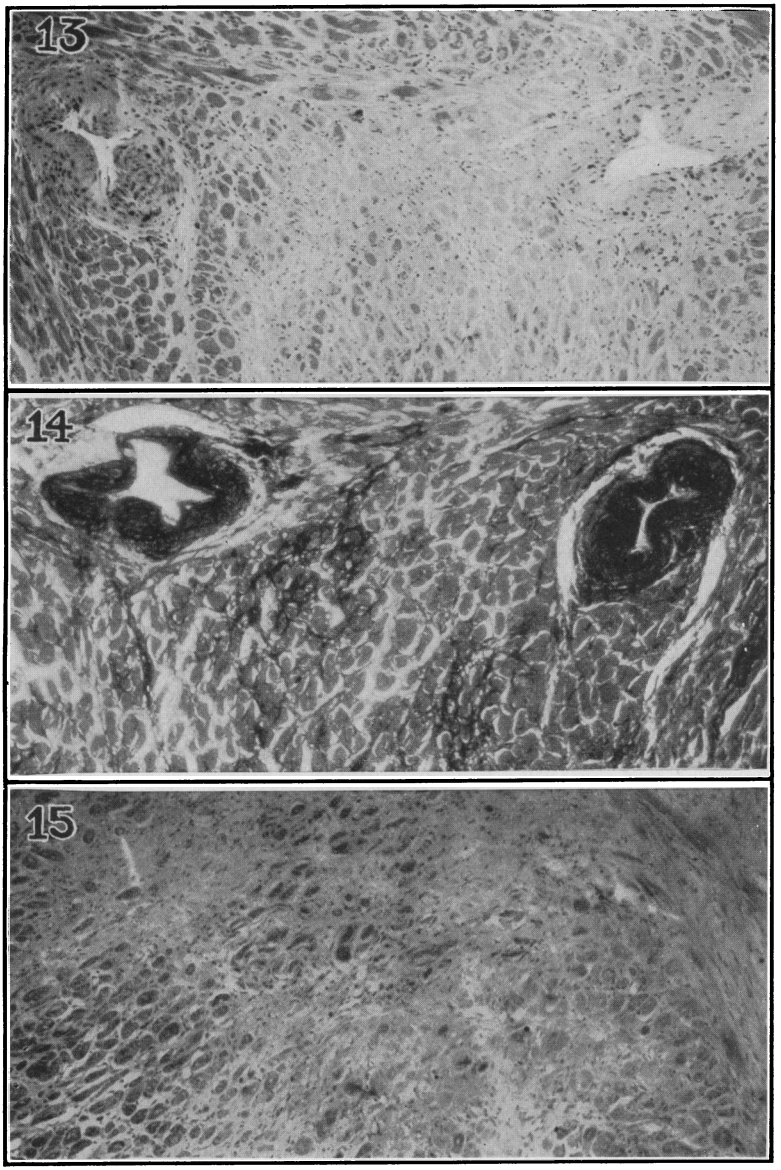
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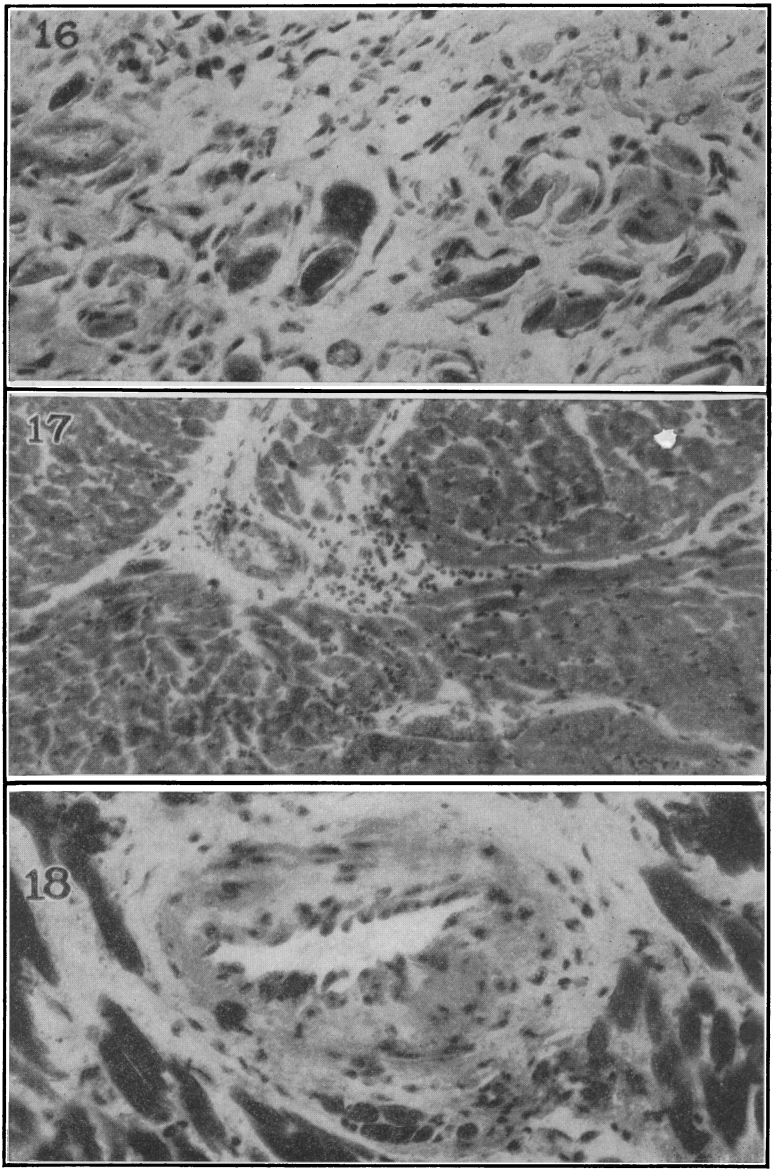
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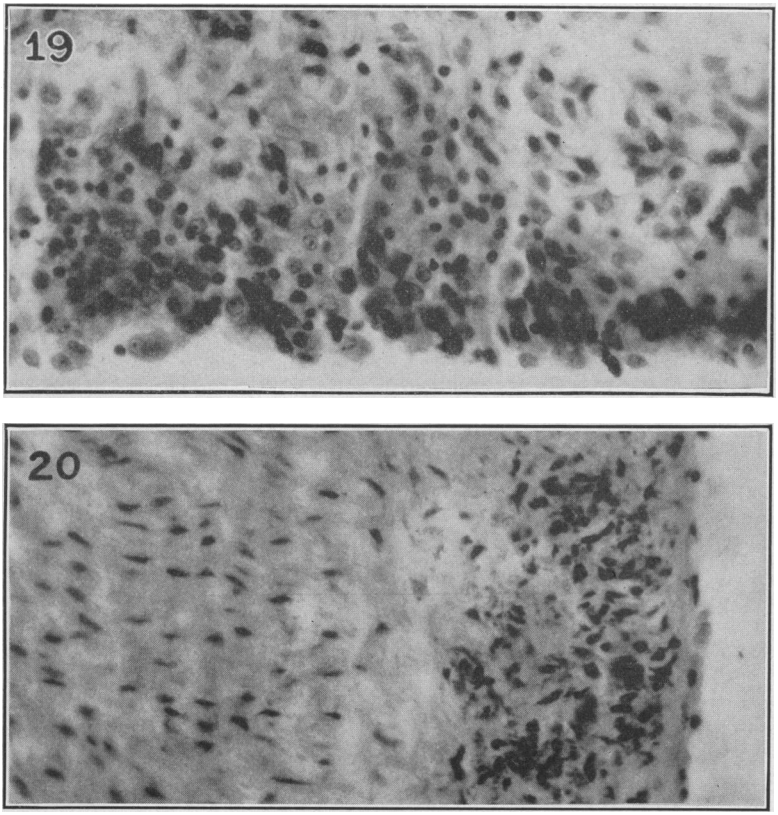


FIGURE 19.—(A. M. M. 64857, rabbit No. 7. Received thyroxin for 169 days; infected 29 days.) Internal surface of the pericardium. The membrane is thickened and near the surface lymphocytes are diffusely scattered while there are numerous round, faintly staining nuclei with conspicuous nucleoli. (×660.)

FIGURE 20.—(A. M. M. 64850, rabbit No. 8. Received thyroxin for 42 days; infected 30 days.) Disruption of structure near the internal surface of the aortic media. (×660.)

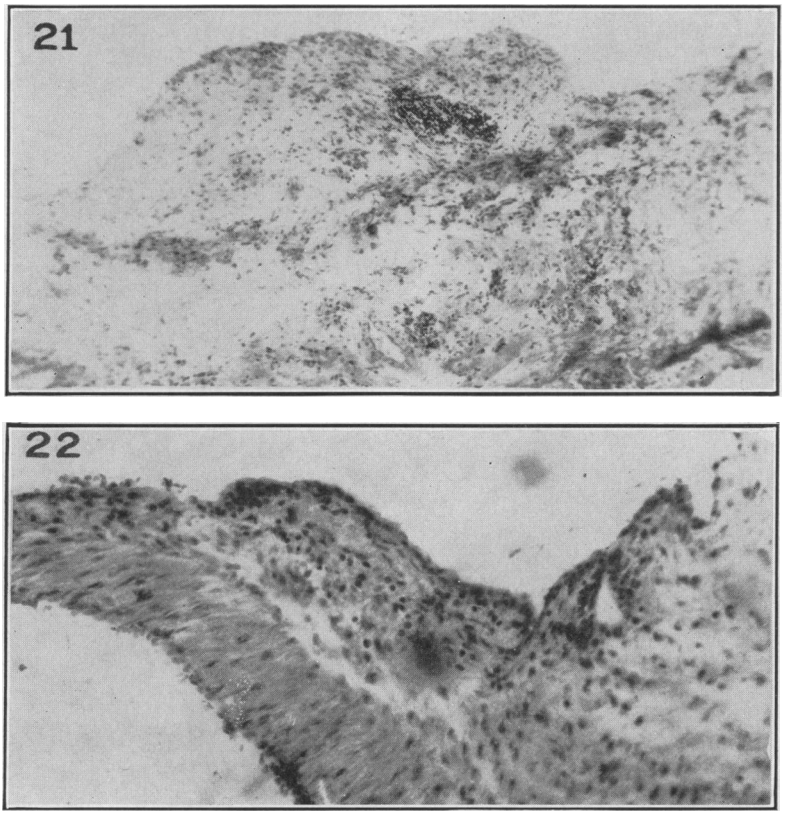
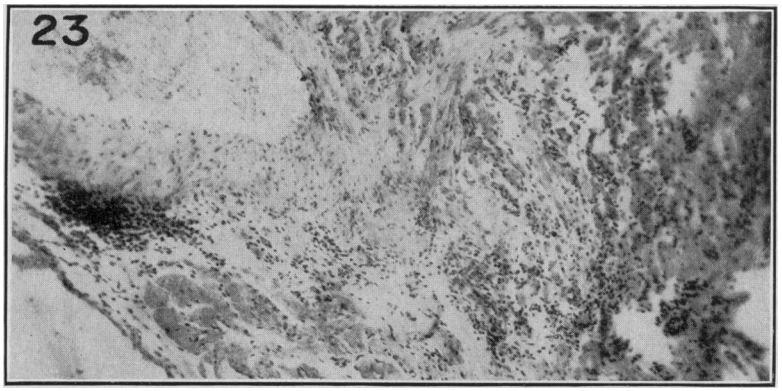


FIGURE 21.—(A. M. M. 64859, guinea pig No. 9. Received desiccated thyroid for 85 days; infected 60 days.) Ventricular endocardium. A circumscribed proliferation of the endocardium containing an accumulation of lymphocytes. Lymphocytes are also scattered in the subjacent myocardium. (×100.)

FIGURE 22.—(A. M. M. 64867, guinea pig No. 5. Received desiccated thyroid for 87 days; infected 35 days.) Base of the mitral valve. There is localized endothelial proliferation with leucocytic infiltration. (×230.)



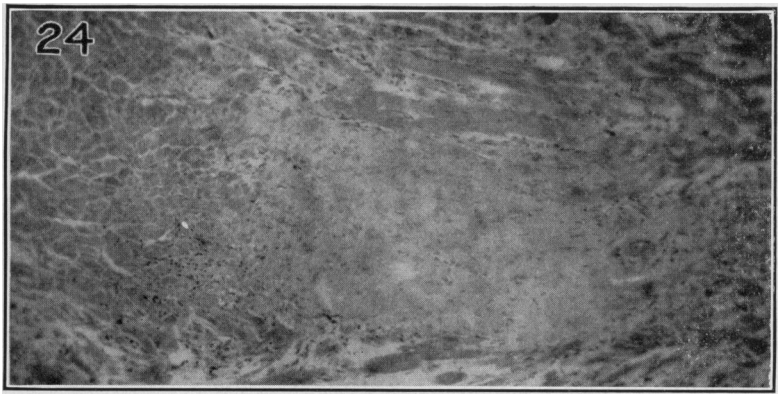


FIGURE 23.—(A. M. M. 64858, guinea pig No. 10. Received desiccated thyroid for 86 days; infected 61 days.) Base of the mitral valve. Lymphocytes are accumulated in the valve and diffusely scattered in the adjacent myocardium. (×100.)

FIGURE 24.—(A. M. M. 64856, guinea pig No. 13. Received desiccated thyroid for 87 days; infected 62 days.) Myocardium. Over a circumscribed area, there is degeneration of all muscle fibers. A few diffusely scattered lymphocytes are at the borders of this lesion. (×100.)

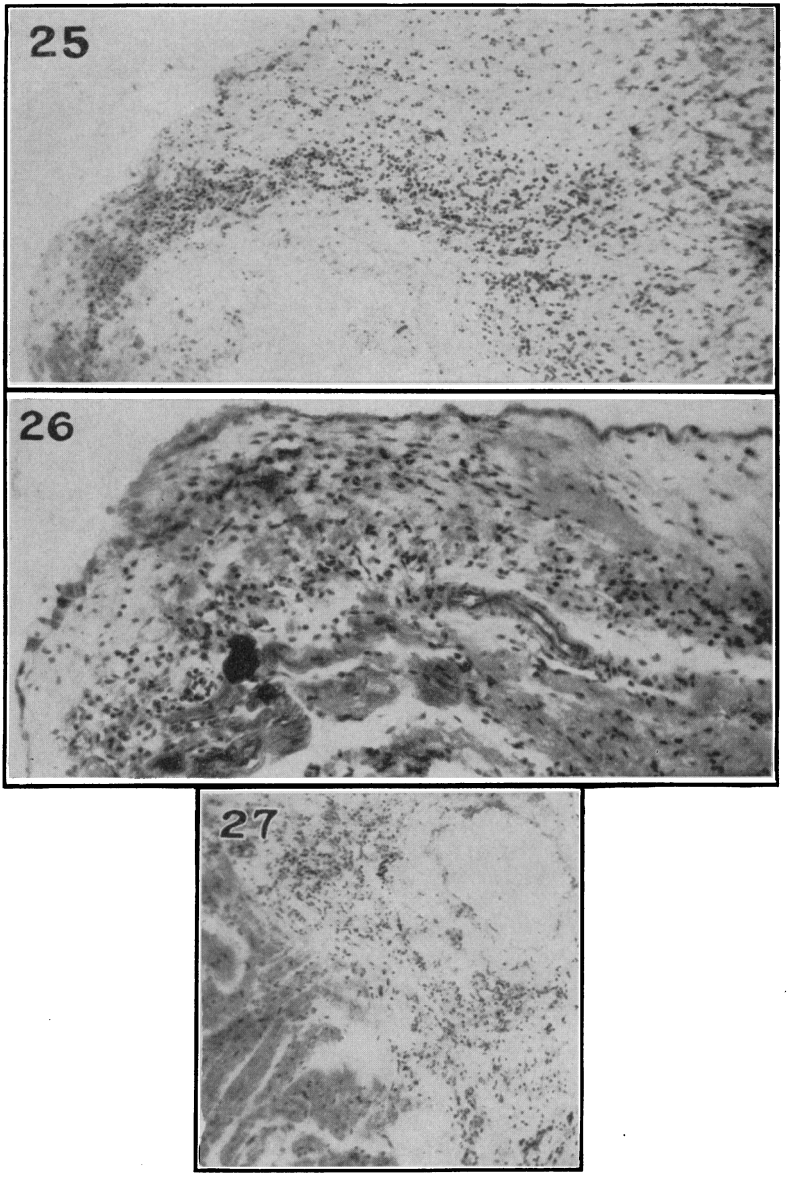


FIGURE 25.—(A. M. M. 64854, guinea pig No. 9. Received desiccated thyroid for 85 days; infected 60 days.) Pericardium. The pericardium is thickened, due in part to fibroblast proliferation. Lymphocytes are distributed through the deeper tissue. (×100.)

FIGURE 26.—(A. M. M. 64855, guinea pig No. 8. Received desiccated thyroid for 85 days; infected 60 days.) Auricular epicardium. There is proliferation of fibroblasts in the thickened epicardium where lymphocytes are diffusely scattered. (×165.)

FIGURE 27.—(A. M. M. 64847, guinea pig No. 10. Received desiccated thyroid for 87 days; infected 62 days.) Coronary vein. There is perivascular edema with diffusely scattered lymphocytes and plasma cells. (×100.)

Epicardium and pericardium.—These structures in only three instances were the sites of similar lesions consisting of small, rather sharply defined areas in which lymphocytes were diffusely distributed near the surface of the thickened membrane. Many round, faintly staining nuclei with small but conspicuous nucleoli were also present (fig. 19).

Blood vessels.—Arteriolar hypertrophy, characterized by thickened media, was frequently observed, and has been described in association with myocardial lesions. Less extensive alterations were found in otherwise uninvolved portions of the myocardium. In the adventitia small, poorly defined, localized lesions were sometimes present. Usually the change was limited to the occurrence of a few pyknotic nuclei in an irregular area of fibrinoid degeneration. Occasionally, however, one or two giant cells with large, pale, vesicular nuclei were seen about the border. Such a lesion is illustrated in figure 18. Slight, localized, endothelial proliferation was also observed as shown in the same figure. Diffuse, perivascular accumulations of lymphocytes were frequently seen (fig. 17).

No pathological changes were found in the larger, more proximal coronary branches. In four instances, however, localized lesions of the inner one-third of the aortic media were seen, usually in the ascending portion. In these areas the fibrillar structure was disrupted and irregular, densely staining nuclei, resembling those of uninvolved regions of the media, were closely packed together. Nuclear fragments were thickly interspersed, but no bacteria or other cellular elements were identified (fig. 20). No other lesions of the aorta were found in any group.

Group D.—The hearts of two of the four rabbits in which infection had been maintained for 125 to 130 days before thyrotoxicosis was induced were essentially negative (table 1). In the remaining two, however, there were lesions comparable in character and extent to those observed in the four affected hearts of rabbits receiving thyroxin only for less than 80 days (group C).

The results indicate that chronic focal hemolytic streptococcus infection of the type employed has slight effect upon the hearts of untreated rabbits unless a blood-stream infection supervenes, in which case purulent, focal myocarditis develops. The latter complication occurs less frequently in thyroxin-treated rabbits subject to the same type of infection; on the other hand, extensive nonpurulent carditis does appear in such animals. Thyroxin given to uninfected rabbits in corresponding doses induces only minor alterations in the heart. When infection has become well established before thyroxin treatment is begun similar minor lesions are induced.

Body temperature.—The rectal temperature of representatives of each group, except group E, was taken each afternoon for about 2 weeks following the induction of infection. The data on animals which were later found to have developed bacteriemia were disregarded. The thyroxin-treated, infected rabbits were more frequently febrile than those in which the infection was uncomplicated and the average temperature in the latter group was slightly lower. Those receiving thyroxin but not infected occasionally showed a slight elevation of temperature.

Erythrocyte sedimentation rate.—The erythrocyte sedimentation rate was determined in representatives of each group at intervals of approximately 2 weeks. Here also figures from rabbits with bacteriemia were discarded, leaving for consideration the results of 219 observations on 54 animals. Sedimentation rates more rapid than 3 mm. per hour were not observed in the controls. Over three-fourths of the infected animals had increased rates, usually to 20–25 mm. per hour, about half the time during the period of observation. Although thyroxin treatment did not discernibly affect the results in infected animals, those receiving thyroxin only occasionally developed slightly accelerated rates.

Agglutinins.—The agglutinin titer in the sera of representatives of each group for streptococcus (K158b) was determined fortnightly, again excluding animals with bacteriemia. Agglutinin titers ranged from 1:4 to 1:8,192, and positive reactions were observed somewhat earlier during the course of infection in the thyroxin-treated, infected animals than in those with uncomplicated infection. Thyroxin treatment, however, apparently did not influence the ultimate concentration of antibody attained.

EXPERIMENT 2. EFFECTS OF INTRAVENOUS INJECTION OF HEMOLYTIC STREPTOCOCCI IN THYROXIN-TREATED RABBITS

The fact that in response to chronic, focal, hemolytic streptococcus infection, thyroxin-treated rabbits developed antibodies somewhat earlier than untreated animals suggested that their response to intravenous immunization might also be altered. To investigate this possibility, the group A hemolytic streptococcus strain London MA was selected. Although intravenous injections of this strain regularly induce agglutinin development, rabbits vary considerably in their production of corresponding anti-M precipitins.

The following groups of rabbits were included in this experiment:

Group A.—Nine untreated rabbits served as controls.

Group B.—Six received intravenous injections of vaccine, followed by living culture, for 7 weeks.

Group C.—Five received injections of vaccine or living streptococci in doses equivalent to those given members of group B but meanwhile also received thyroxin intravenously.

Blood sera were tested weekly for anti-M precipitins and agglutinins for strain London MA. The former antibody appeared in no instance, although agglutinin titers rose rather irregularly in groups B and C as high as 1: 3,200. After 7 days agglutinins were present only in one thyroxin-treated animal, and after 14 days serum from two members of this group showed a titer of 1: 3,200, although a titer of this height was not demonstrated in the untreated group until the twenty-first day. At this time, however, there was no definite difference between the two groups with respect to agglutinin concentration and no further differences were observed.

At autopsy no alterations were apparent to examination in the gross except a diminution of fat in the thyroxin-treated group. Microscopically, in three of the nine controls lesions similar to those in the corresponding group of experiment 1 were observed. In half the otherwise untreated rabbits receiving bacteria intravenously (group B) the hearts were negative, while in five, lesions comparable to those seen in the infected group of experiment 1 were apparent. There was extensive myocardial fibrosis in the hearts of all the thyroxin-treated rabbits receiving culture intravenously. Over large areas of the myocardium the parenchyma was replaced by connective tissue of the adult type. The endocardium was thickened over irregular areas, but no active lesions were observed. Arterioles were thick-walled and showed distinct hypertrophy of the media.

EXPERIMENT 3. THE RESPONSE OF THYROXIN-TREATED RABBITS TO BACTERIAL HYPERSENSITIZATION

Inasmuch as bacterial hypersensitivity develops during the course of focal infection of the type employed in experiment 1, it was of interest to study the response to bacterial hypersensitization in thyroid-toxic rabbits. A strain of indifferent streptococcus (K155) which had been found effective in inducing hypersensitivity was employed.

The following groups of rabbits were treated in the manner described in the section on "Methods":

Group B.—Six were rendered hypersensitive.

Group D.—Five were rendered hypersensitive while receiving thyroxin intravenously.

Because this experiment was run concurrently with experiment 1, the untreated group A and the group receiving thyroxin only (group C) in that experiment served as controls with respect to the pathological findings here.

The degree of cutaneous sensitivity to the strain employed was tested at the outset of the experiment and after 14, 21, and 60 days. Mod-

erate cutaneous hypersensitivity was demonstrable in groups B and D after 2 weeks but was more pronounced after 3 weeks. During the succeeding month of observation, sensitizing doses of bacteria were given only at infrequent intervals. This degree of hypersensitivity persisted until the end of the experiment. At the beginning of the experiment when cutaneous reactivity was first tested and at each succeeding observation as hypersensitivity developed, the lesions of thyroxin-treated animals were found to be smaller and less edematous than those of the untreated rabbits. They also regressed more rapidly and with healing became more indurated. Agglutinin titers in the two groups were comparable.

The animals were killed after 60 days of observation. Their internal organs were negative to examination in the gross except for the lack of fat in members of group D. Microscopically the hearts in group B were similar to those in the infected group (B) in experiment 1. The microscopic cardiac lesions in group D both at 59 and 70 days were similar to those observed in group D of experiment 1 in rabbits which had been infected from 11 to 30 days.

EXPERIMENT 4. THE RESPONSE TO CHRONIC HEMOLYTIC STREPTOCOCCUS INFECTION IN GUINEA PIGS RECEIVING DESICCATED THYROID ORALLY

Chronic infection was induced by strain J20 of group C hemolytic streptococcus in guinea pigs receiving desiccated thyroid orally. The following groups were observed:

Group A.—Twenty untreated guinea pigs served as controls.

Group B.—Thirty guinea pigs were subjected to the influence of chronic hemolytic streptococcus infection only (including 4 with chronic spontaneous hemolytic streptococcus adenitis).

Group C.—Ten guinea pigs received desiccated thyroid by mouth.

Group D.—Thirteen guinea pigs were subjected to the combined effects of chronic hemolytic streptococcus infection and desiccated thyroid orally, after the latter had been administered for 25 days.

Group E.—Thirteen guinea pigs suffering from spontaneous hemolytic streptococcus adenitis were given desiccated thyroid by mouth.

Group A, controls.—In view of the frequency of spontaneous hemolytic streptococcus infection in the guinea pigs available, care was taken that individuals comprising this group were healthy. The criteria for the absence of infection were as follows: Continuous gain in weight (the animals were not mature), absence of clinical signs on semiweekly examination, and negative macroscopic postmortem observations, supplemented by a microscopic study of sections of the lungs. Half the animals in this group presented no discernible cardiac lesions, while in the other half the changes were minimal. Most commonly seen were small, compact, localized accumulations of mononuclear cells (sometimes associated with a few eosinophiles) in the mural

endocardium and occasionally extending beneath it, but without evidence of damage to adjacent myocardial fibers. Such foci were multiple and were observed in the walls of all the chambers, although only a few were seen in one heart; a predilection for the papillary muscles of the left ventricle was evident. Similar collections were occasionally present in the myocardium where they were limited to a few mononuclear cells, occasionally in perivascular location. Pericardial lesions were invariably perivascular and consisted of a few sparsely scattered lymphocytes and mononuclear cells; in one instance eosinophiles were also present. The only other cardiac lesions presented in the control group were occasional small, recent myocardial hemorrhages.

Group B, infection only.—Infection was induced in 26 guinea pigs. Six which died were found to have acute, focal, purulent myocarditis. Of the remaining 20 animals, none died during an 85-day observation period. Four with chronic spontaneous hemolytic streptococcus lymphadenitis when they came under observation were observed for a period of 60 days. None of the latter died and none developed purulent carditis.

In the hearts of these 30 infected animals, with the exception of the 6 developing purulent carditis, lesions were observed similar to those in the control animals. Although they were slightly more extensive here, a discernible difference in the character of the lesions was evident only in the instances of spontaneous infection. In those cases eosinophiles were invariably present in the small, cellular accumulations occasionally noted in the mural endocardium.

Group C, desiccated thyroid only.—No evidence of infection was found in this group. One animal died after 22 days and the remainder were killed at intervals between the fifty-first and eighty-sixth days. In about half of these animals the heart showed changes comparable in character and extent to those observed in the uninfected control group (A).

Group D, desiccated thyroid and induced chronic infection.—No member of this group died; the viscera were macroscopically normal and no purulent myocarditis was observed. As in the rabbits, metastatic infection was less frequent in thyroid-toxic than in untreated animals. Members of this group were killed at intervals between the fifty-second and eighty-seventh days.

The myocardium was less extensively involved than in rabbits similarly treated, and the lesions were in an early stage of development. A typical example was found in the heart of guinea pig No. 13 (fig. 24). Over a rather sharply delimited area the muscle fibers showed advanced degenerative changes. The borders of individual fibers as well as their internal structures were indefinite and only a

few pale nuclei remained. At the periphery of such areas there were diffuse accumulations of lymphocytes and plasma cells.

Endocardial lesions were not uncommon; the most frequent alteration consisted of collections of lymphocytes and plasma cells diffusely scattered through this layer and the subjacent myocardium or distributed in dense seams underneath the endothelium or at the endocardial-myocardial junction. Lesions of the latter type were most common in the auricles. Circumscribed endocardial proliferations were found in both auricle and ventricle. A characteristic example of such involvement in the latter location was found in guinea pig No. 9 (fig. 21). Over a sharply delimited region the endocardium was much thickened. The rather homogenous tissue toward the endocardial surface showed no fibrillar structure and contained many, large, elongated, pale nuclei with a line of granular stippling along their longitudinal axis. At the base of this nodular thickening there was active fibroblast proliferation; and a rather closely packed accumulation of lymphocytes occupied part of the intermediate region.

Valvular lesions were found exclusively at the base of the mitral and consisted of cellular infiltration or endocardial proliferation. A lesion of the former type was found in guinea pig No. 10 (fig. 23). In the adjacent myocardium, in the connective tissue and interstices between the muscle fibers, and extending up the base of the valve, lymphocytes and plasma cells were diffusely scattered or gathered in rather dense clumps. There was some associated fibroblast proliferation. Proliferation of the endothelium at the base of the mitral valve, as seen in guinea pig No. 5 (fig. 22), was also common.

Epicardial and pericardial lesions were more usual than in rabbits similarly treated. Lymphocytes and plasma cells were diffusely scattered or, in the pericardium, more densely distributed in a seam under the endothelium. A moderate degree of fibroblast proliferation was usually associated with these lesions (figs. 25 and 26).

Granulomatous perivascular formations were not seen. In this locality also diffuse or compact accumulations of lymphocytes and plasma cells constituted the only type of lesion (fig. 27).

Group E, treatment with desiccated thyroid after the establishment of infection.—Four members of this group died between the seventeenth and twenty-ninth days of treatment while the remaining nine animals were killed at intervals between the forty-fourth and fifty-eighth days. The internal organs were all macroscopically normal and there was no purulent myocarditis. The microscopic cardiac lesions, however, were of slight degree and corresponded in frequency and character to those seen in uncomplicated spontaneous infection (group B).

EXPERIMENT 5. CHRONIC HEMOLYTIC STREPTOCOCCUS INFECTION IN RABBITS TREATED WITH DINITROPHENOL

In order to study the influence upon the response to infection of an accelerated metabolic rate dissociated from other thyroxin effects, rabbits were given maximum doses of dinitrophenol. Four groups were investigated:

Group A.—Six rabbits receiving no treatment served as controls.

Group B.—Ten rabbits were infected using the methods of experiment 1.

Group C.—Five rabbits received dinitrophenol intravenously.

Group D.—Five rabbits, infected as those in group B, received dinitrophenol as those in group C, beginning 2 days before the infection was instituted.

Three animals of thirteen from which groups C and D were later formed died during the first 2 days of treatment and 3 in group B succumbed after they had been infected. Dinitrophenol treatment caused the rabbits in groups C and D to remain stationary in weight or to lose slightly, a more pronounced effect than that obtained from thyroxin in the dosage employed in experiment 1. Individuals were autopsied at intervals between the forty-sixth and seventy-sixth days of treatment. Aside from the inguinal and axillary abscesses in members of groups B and D, no lesions were apparent upon examination in the gross although there was little fat in the dinitrophenol-treated individuals. Upon microscopic examination, purulent, focal myocarditis was found in the 3 infected animals which succumbed. The incidence, character, and extent of minor cardiac lesions in groups A and B corresponded to those observed in comparable individuals of experiment 1, while the findings in group D differed in no particular from those in group B. Thus, treatment with dinitrophenol failed to induce cardiac lesions of itself or in combination with infection.

DISCUSSION

Attention has been drawn to the resemblances between the cardiac lesions which have been observed in experimental hyperthyroidism and those of rheumatic fever (*28*, *34*). This is of particular interest because of the tendency for the latter disease and exophthalmic goiter to develop in the same individuals (*35*). The endocardial, perivascular, and pericardial lesions which have been described here, however, only remotely resemble those of rheumatic fever in that fibrinoid degeneration, connective tissue proliferation, and endocardial destruction and proliferation were present with an occasional multinucleated giant cell while polymorphonuclear leucocytes or pseudoeosinophiles were rare. Few granulomatous lesions were seen and none developed sufficiently to warrant comparison.

Myocardial fibrosis, very similar in appearance to that which has been described in exophthalmic goiter, was the most common and extensive change observed. This suggests that infections during the course of activity of this disease may result in the development of permanent cardiac damage and emphasizes the desirability of early operative treatment for hyperthyroidism.

The pathogenesis of cardiac lesions developing in animals suffering infection during treatment with thyroid products is not clear. Those who have observed similar pathological changes resulting from apparently uncomplicated, experimental hyperthyroidism have concluded variously that the damage resulted from a direct toxic effect of the thyroid hormone on the myocardium or that it was indirectly incident to overwork of the organ. Our investigations do not exclude the possibility that cardiac lesions may be induced by intense uncomplicated hyperthyroidism operating through such mechanisms, for those who have reported the development of pathological changes in experiments of this kind, apparently not complicated by infection, gave relatively larger doses of active thyroid products than were employed here. It is improbable, however, that the lesions induced in the present experiments were due simply to intensification of the thyroid effect by infection because no unusual pathological changes were observed in the hearts of either rabbits or guinea pigs in which infection was well established before thyroidism was induced. The size of the subcutaneous abscesses and the trend of body weight, however, in these animals indicated that they were exposed to the effects of infection equivalent in intensity to those experienced by animals which were infected after thyroidism had been induced and which consequently developed cardiac lesions. The fact that infection was found not to intensify the minor lesions induced in rabbits and guinea pigs by repeated doses of adrenalin (*36*) is further indication that cardiac overwork of itself does not render the heart susceptible to damage incident to the type of infection employed here. This also suggests that the conditioning effect of thyroxin and dried thyroid was not mediated by stimulation of the sympathetic system.

Alterations of biochemical relationships which have been found in the heart during thyroidism and which might be responsible for altered reactivity in infection include a reduced content of creatin (*37*), phosphates (*34*), adenyl-pyrophosphoric acid (*38*), and glycogen (*39, 40, 41*), and an increased concentration of lactic acid (*42*) and nonprotein nitrogen (*43*). It is probable, however, that a general altered reactivity of the entire body plays a part as reflected by the slightly enhanced antibody formation during infection and intravenous immunization with hemolytic streptococcus in thyroxin-treated rabbits. We have also demonstrated a comparable, relative increase in antibody production rate in thyroxin-treated rabbits

injected with horse serum (*44*), while Blom (*45*) observed that thyroidism increased the susceptibility of guinea pigs to anaphylactic shock.

The concentration of agglutinins developing during the course of bacterial hypersensitization was not found to be affected by treatment with thyroid products. It has been demonstrated, however, that humoral antibodies are quite irregularly associated with the state of bacterial hypersensitivity (*46*), while in their concentration there they never represent the potential maximum response of the individual. In bacterial hypersensitivity, as in hypersensitivity to horse serum (*44*) in rabbits, the influence of thyroidism is manifest by the relatively small size of the cutaneous lesions and their comparatively rapid evolution.

The fact that treatment with dinitrophenol did not produce an effect similar to that of thyroxin or desiccated thyroid indicates that increased metabolic rate alone is not responsible for the apparent conditioning effect of the latter substances. The factors which predispose to the development of nonpurulent carditis as a complication of infection, however, are probably associated with a state of accelerated metabolism, for lesions of this type also appear in the hearts of infected scorbutic (*47*, *48*, *49*) and insulin-treated guinea pigs (*50*). The effect of insulin in accelerating carbohydrate metabolism is recognized, while the preponderance of evidence indicates that the metabolic rate is elevated in scurvy (*51*). When the degree of thyroid hyperactivity is further increased in the latter condition by exposure of the affected animals to ultraviolet radiation (*52*), we have found that the concurrent presence of infection induces more severe cardiac lesions than those which appear in guinea pigs similarly treated but not irradiated (*53*).

On the other hand, the numerous reports of an essentially antagonistic relationship between thyroxin and ascorbic acid in their physiological effects (*38*, *54*, *55*) suggest that the alterations in response to infection observed may have been due to a vitamin C deficiency incident to the depleting effects of treatment with thyroid products. This possibility is strengthened by the observation that the onset of scurvy in guinea pigs is hastened by the concurrent feeding of thyroid gland (*56*). The fact that each treated animal in these experiments consumed at least 50 gm. of fresh cabbage daily, however, probably eliminates this factor, for the minimum amount which has been found to protect guinea pigs against scurvy is only 5 gm. (*57*).

Both indifferent and hemolytic streptococcus infections were effective in inducing nonpurulent carditis in thyroid-treated animals. The absence of pathological change in the hearts of individuals exposed to thyroidism after infection was established, however, suggests that for the production of carditis of this type, it is necessary for the infected

animal while in a state of thyroidism to pass through a stage of immunization or hypersensitization. Since the induction of both immunity and hypersensitivity were found to be effective in inducing nonpurulent carditis under these conditions, the precise nature of the influence of infection remains obscure.

SUMMARY

1. Chronic, focal, hemolytic streptococcus infection, intravenous immunization with this micro-organism, or the induction of cutaneous hypersensitivity to indifferent streptococci, while of slight effect upon the hearts of untreated rabbits, are associated with the development of extensive, nonpurulent carditis in members of this species treated with thyroxin. A similar relationship between chronic, focal, hemolytic streptococcus infection and the feeding of desiccated thyroid is demonstrable in guinea pigs. Equivalent doses of thyroid products in the absence of infection, or given after infection is well established in these species, induce only minor cardiac lesions. Chronic, focal, hemolytic streptococcus infection in rabbits with elevated metabolic rates induced by dinitrophenol treatment is also ineffective in this respect.

2. The myocardial changes resemble those described in exophthalmic goiter and, by some observers, in apparently uncomplicated experimental thyroidism. The endocardial, perivascular and pericardial lesions only remotely resemble those of rheumatic fever, but include fibrinoid degeneration, proliferation, and destruction without the presence of purulent inflammation.

3. In thyroxin-treated rabbits infected subcutaneously or injected intravenously with hemolytic streptococci, agglutinins appear somewhat earlier than in those untreated. The cutaneous lesions induced during the course of hypersensitization to indifferent streptococci are smaller and evolve more rapidly in thyroxin-treated rabbits than in controls, but cutaneous hypersensitivity develops in both groups and the concurrent concentration of agglutinins is apparently not influenced by treatment.

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SIMILARITY OF AUSTRALIAN "Q" FEVER AND A DISEASE CAUSED BY AN INFECTIOUS AGENT ISOLATED FROM TICKS IN MONTANA [1]

By R. E. DYER, *Senior Surgeon, United States Public Health Service*

In 1935 a filter-passing agent was recovered by Davis and Cox (*1*) from ticks (*Dermacentor andersoni*) collected near Nine Mile Creek, about 32 miles west of Missoula, Mont. This agent was shown to pass Berkefeld filters N and W which were impermeable to ordinary bacteria and to the viruses of typhus and Rocky Mountain spotted fever. Cox (*2*) has so far been unable to cultivate the filter-passing agent on media free from living cells, but has found that it multiplies freely in tissue cultures. He has described rickettsialike organisms which are present in abundance in tissues of infected guinea pigs and in tissue cultures. Parker (*3*) has shown that this infectious agent survives in and can be transmitted by nymphal and adult *D. andersoni* that have ingested the virus in the larval stage and that it also survives through the eggs of infected female ticks to the larval stage. Davis and Cox (*1*) have described the infection in guinea pigs in some detail, and they have also shown that white rats, mice, and rabbits are susceptible, although they were unable to carry the infection beyond the third transfer in rabbits. No agglutinins for *Proteus* X strains were found in these rabbits. They were unable to infect monkeys (*M. rhesus*) in three attempts, two monkeys being used at each trial.

Following an incubation period, usually of from 4 to 6 days, the infection produces a definite febrile reaction in guinea pigs which lasts from 2 to 8 days. No scrotal reaction has been observed. Following subcutaneous inoculations, the guinea pigs develop a marked inflammatory thickening of the skin at the site of inoculation. Death of the guinea pigs is not infrequent. The chief post mortem finding is an enlarged spleen.

Dyer (*4*) has reported the accidental infection of a laboratory worker with the Montana infection and has suggested a relationship between this infection and "Q" fever of Australia, his suggestion being based on the fact that he found that guinea pigs which had recovered from infection with "Q" fever virus were subsequently immune to the infectious agent isolated by Davis and Cox from Montana ticks.

"Q" fever was described by Derrick (*5*) in 1937 as an acute illness with a febrile period of from 7 to 24 days. The first cases were noted by Derrick in 1935. Headache is a prominent symptom, other complaints being malaise, anorexia, and pain in back and limbs. A rash is not a feature of the disease, being present in only one of the first

[1] From the Division of Infectious Diseases, National Institute of Health.

nine cases described. Blood counts have been essentially normal, blood cultures negative, and no production of agglutinins for *Proteus* X19 or K, undulant fever, typhoid and paratyphoid fever, or leptospirosis has been found.

The Australian cases have so far been found chiefly among workers in abattoirs and among dairy farmers.

Burnet and Freeman (*6*) described a rickettsia in the spleens of mice infected with "Q" fever. This rickettsia has been named *Rickettsia burneti* by Derrick (*7*). Burnet (*8*) points out that this rickettsia differs from other recognized rickettsiae in that it fails to produce agglutinins for either *Proteus* X19 or XK in man or animals, and as yet no arthropod vector has been found. There is also some question in regard to its filterability, since Burnet found that it was filterable to some extent through gradacol type membranes of 0.7μ average pore diameter. This rickettsia, like other recognized rickettsiae, grows readily in tissue cultures but not on ordinary media.

The Australian workers have found monkeys (*Macacus rhesus*), guinea pigs, white mice, and several native rodents susceptible (*12*). They reported in their early publications that they had not succeeded in infecting rabbits. Apparently they have more recently found this rodent susceptible. Various wild animals have also been found susceptible, particularly the bandicoot (*Isoodon macrourus*), a marsupial, and some evidence has been presented that this animal may act as a reservoir in nature (*9*). They report failure in their attempts to infect two species of mites and one species of fleas. They have not published reports of trials with ticks, but Derrick in a personal communication states that he has apparently been able to infect one species of tick.

In their identification of "Q" fever in man and animals the Australian workers (*10*, *11*) rely largely on the agglutination of rickettsia suspensions prepared from mouse spleens and upon cross immunity tests. The titers of the agglutinating sera are low, one series of 4 human laboratory infections yielding titers of 1:10, with one additional case showing a titer of 1:100. They have failed to find agglutinins for *Rickettsia burneti* in Rocky Mountain spotted fever sera, and in a typhus serum which agglutinated *Proteus* X19 at 1:1280. Cross immunity (guinea pig tests) has been found lacking between "Q" fever and leptospirosis, rat-bite fever, and caseous lymphadenitis of sheep. In addition, Rocky Mountain spotted fever vaccine does not protect against "Q" fever.

Derrick reports that in guinea pigs "Q" fever produces a definite febrile reaction of from 4 to 6 days duration following an incubation period of 2 to 18 days. Some guinea pigs have fever lasting only one day, while inapparent infections have been noted. Scrotal reactions have not been observed and the mortality is nil. The chief post-

mortem finding is an enlarged spleen. No particular local reaction was found at the site of subcutaneous inoculations.

The infection isolated by Davis and Cox from Montana ticks was contracted by a member of the staff of the National Institute of Health in May 1938. The course of this illness was similar to that described for "Q" fever, except that headache was absent. During illness the infection was recovered from the patient's blood and was established in guinea pigs. On the recovery of the patient it was shown that his blood contained neutralizing antibodies when tested against the virus previously recovered from the blood. This definitely indicated that the infection in the guinea pigs and that in the patient was the same. This strain of the Montana infection has been referred to in a previous publication (*4*) as the X strain and the same designation will be used in this paper. Further study of this strain in comparison with the original strain isolated in Montana and maintained in guinea pigs has shown that these two strains are identical.

At the time this study was in progress at the National Institute of Health, a strain of "Q" fever supplied by Dr. Burnet was also being carried in guinea pigs. In the course of these studies 5 guinea pigs which had recovered from "Q" fever were inoculated with the X strain and found to be immune. Unfortunately, about this time the "Q" fever strain was lost through secondary infection in the guinea pigs. Dr. Burnet again sent his "Q" fever strain to this laboratory in the form of two infected mouse spleens, and the strain was again established in mice and guinea pigs and has been maintained for approximately 4 months. This "Q" fever strain has, in this laboratory, never given quite as definite reactions in guinea pigs as those described by the Australian workers. The incubation period has been somewhat longer, the fever of shorter duration, and the spleen, although enlarged, has not been found enlarged to the extent indicated by the Australian reports. Rickettsiae have been observed in smears from the cut surface of the spleens of mice, but they have never been numerous. We have been of the impression that the infection in guinea pigs, although definite, is not as marked as that described by the Australian investigators. It is thought that this strain may have lost virulence during its storage en route from Melbourne, the total time of storage for one mouse spleen being 63 days, and for the other, 47 days.

CROSS IMMUNITY TESTS

A series of cross immunity tests have been made between the "Q" fever strain, the X strain of the Montana infection, two strains of typhus fever, one (W) an endemic strain, the other (B) an epidemic strain, and two strains of Rocky Mountain spotted fever, one (BR) a virulent strain isolated in Montana, the other (K) a milder strain iso-

lated from a patient who contracted his infection in Maryland. Complete cross immunity exists between the two strains of typhus and between the two strains of spotted fever, while it is lacking between the typhus strains and the spotted fever strains.

Figures 1 to 5 [2] show cross immunity tests of the "Q" and X strains.

It will be seen that there is no cross immunity between the X strain and the typhus and spotted fever strains and none between the "Q"

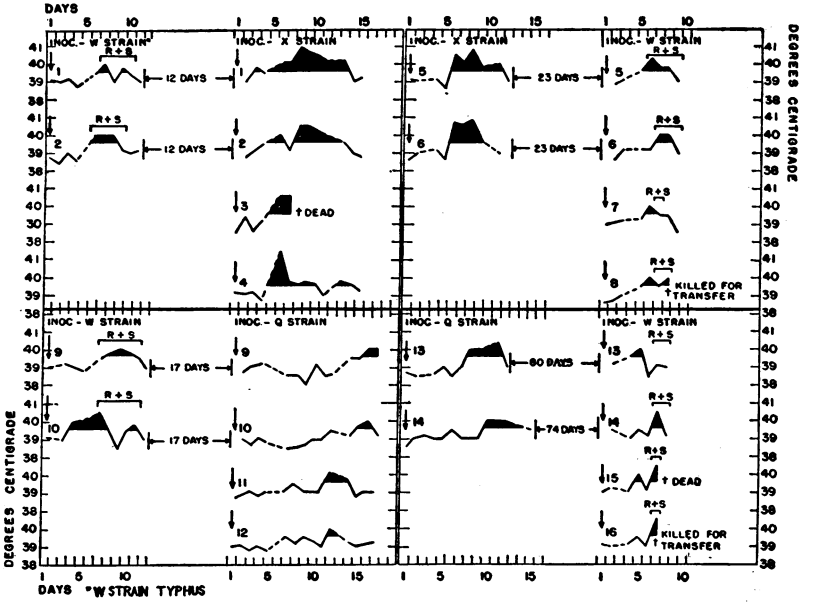

FIGURE 1.

fever and spotted fever strains. There is a suggestion of some degree of immunity produced by the typhus strains against the "Q" fever strain but the reverse of this is not true. There is complete cross immunity between the X strain and the "Q" fever strain.

AGGLUTINATION TESTS

Dr. Burnet kindly supplied us with a suspension of rickettsiæ prepared by him. This suspension was tested by Dr. Topping, of the National Institute of Health staff, against the serum from case X drawn after recovery from infection with the Montana virus, and against control specimens of sera from a recovered case of "Q" fever, supplied by Dr. Derrick, an immune rabbit serum from Australia, and sera from two men at the National Institute of Health. One of

[2] In these figures, the temperature records of guinea pigs are shown. Arrows pointing down indicate the day of inoculation. Guinea pig identification number is given above each temperature curve. In each test guinea pigs were inoculated intraperitoneally with the strain shown on the chart. After recovery following this inoculation these guinea pigs and additional guinea pigs were inoculated with the strain indicated. "R+S" = Scrotal redness and swelling typical for the strain used.

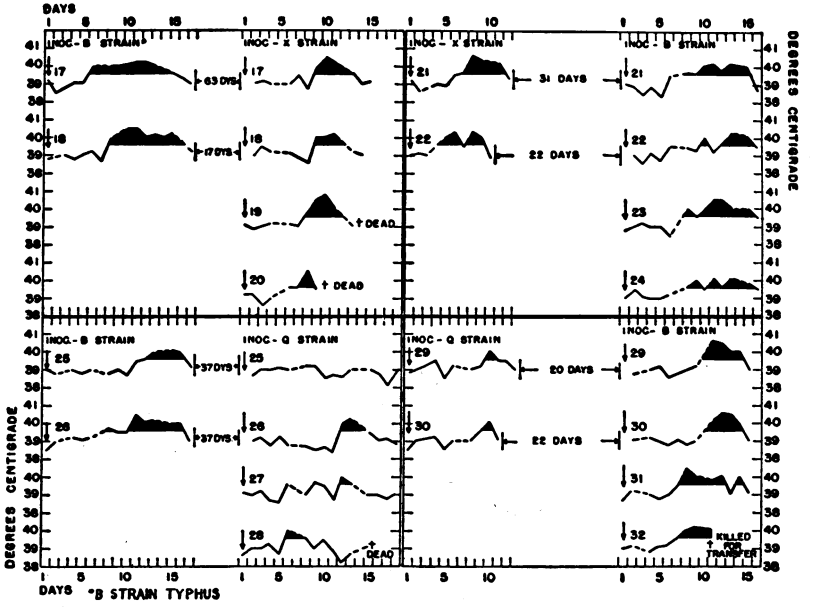

FIGURE 2.

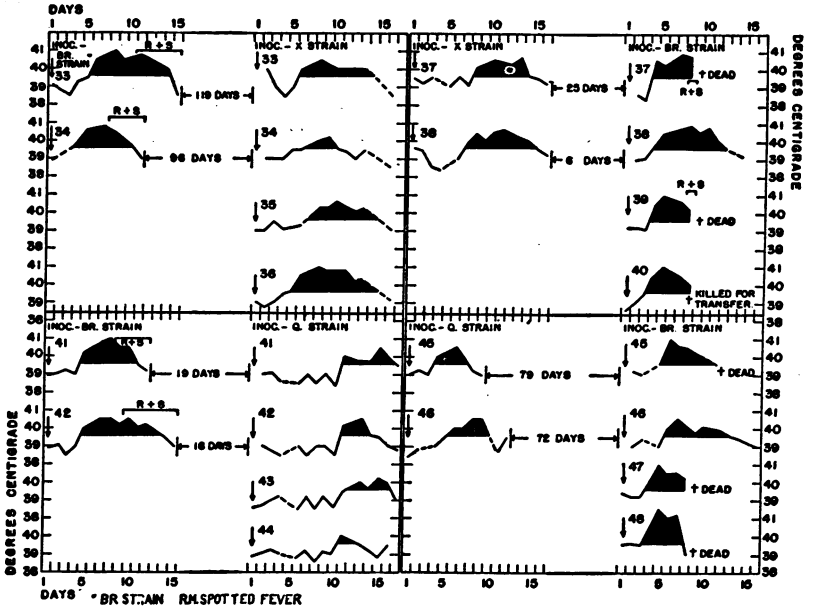

FIGURE 3.

the men had had typhus fever and had been repeatedly vaccinated against spotted fever. The second had had no previous rickettsial infection. The rabbit serum contained no agglutinins for X19 or X2. An additional human serum from a suspected but unproven case of

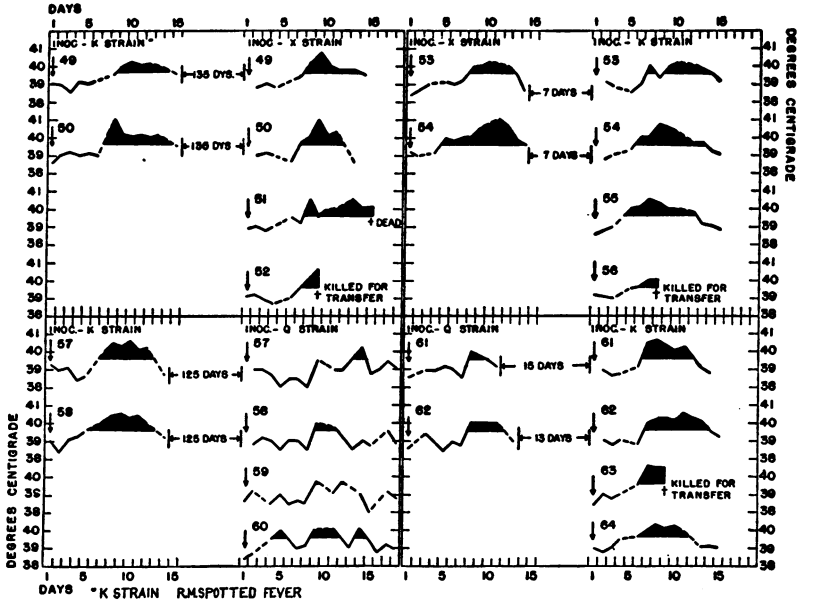

FIGURE 4.

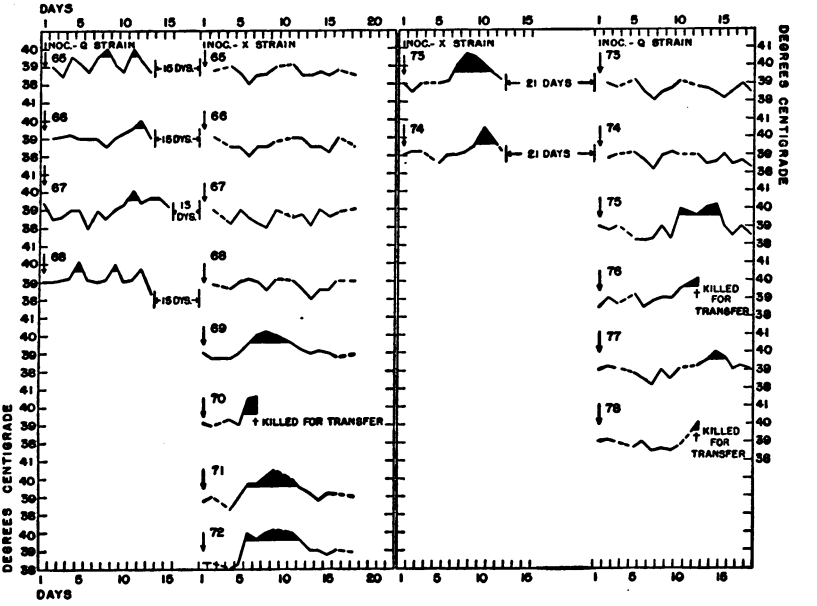

FIGURE 5.

infection with the same Montana infection suffered by X was also tested.

The results of this agglutination test are shown in table 1.

It should be noted that the Australian authors consider agglutinations in dilutions as low as 1:5 and 1:10 as significant.

TABLE 1.—*Agglutination of* Rickettsia burneti [1] *by certain sera*

Serum	Serum dilution					
	1:5	1:10	1:20	1:40	1:80	1:160
X	[2]3	2	2	1	0	0
Kn	0	0	0	0	0	0
Hi	0	0	0	0	0	0
Co	2	0	0	0	0	0
Q	3	2	0	0	0	0
Rabbit	4	4	4	3	3	2

[1] Rickettsia suspension prepared by Dr. Burnet (Australia).
[2] 4=complete; 3=incomplete; 2=partial; 1=trace.
Serum identification:
X.—Serum from Case X. Previous history: Typhus fever, vaccinated against Rocky Mountain spotted fever, infected with the infectious agent from Montana ticks.
Kn.—Previous history: Typhus, vaccinated against Rocky Mountain spotted fever.
Hi.—No previous rickettsial infection nor vaccination.
Co.—Vaccinated against Rocky Mountain spotted fever. Possible previous infection with the infectious agent from Montana ticks.
Q.—Australian case of "Q" fever.
Rabbit.—Infected with "Q" fever in Australia.

PROTECTION TESTS

Protection tests have been made using various human sera and the X strain of virus. In these tests, 0.5-cc. amounts of the serum being

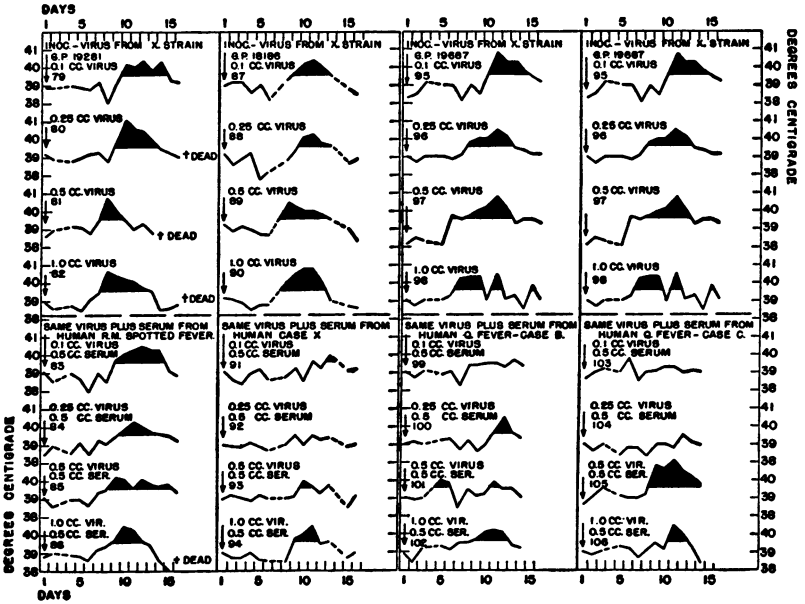

FIGURE 6.

tested were mixed in conical vials with different amounts of blood serum drawn from a guinea pig at the height of its infection. The amounts of this guinea pig blood serum virus (X strain) were 0.1, 0.25, 0.5, and 1.0 cc. The mixtures were allowed to stand at room temperature for 30 minutes and then injected intraperitoneally into

guinea pigs. Control guinea pigs were inoculated with like amounts of the same blood serum virus.

The sera used were from the following cases:

One case of Rocky Mountain spotted fever. This serum gave complete protection against spotted fever.

One sample from Case X from which virus X was recovered.

Two sera supplied by Dr. Derrick from human cases of "Q" fever.

The results of these protection tests are shown in figure 6. It will be seen from this figure that definite protection against X virus was afforded by the X serum and the "Q" fever sera while the control spotted fever serum showed no protection.

SUMMARY

The points of similarity and dissimilarity between the Montana infection and the "Q" fever of Australia may be summarized as follows:

Epidemiology.—"Q" fever has been recognized principally in persons associated with animals, which suggests infection from direct contact with infected animal tissues or with animal parasites.

The epidemiology of the Montana infection is unknown, but the presence of the virus in ticks suggests that human infections may be found in rural areas.

Clinical.—The one recognized human infection with the Montana virus was very similar to the published descriptions of the Australian "Q" fever cases.

Susceptibility of animals.—As far as work has been carried out, the only point of difference in susceptibility of animals to the two infections is the failure of the American workers to find the monkey susceptible, in contrast to the success in infecting this animal in Australia.

The susceptibility of rabbits has not been studied thoroughly enough to warrant definite statement.

Serology in man and animals.—Neither disease has been found to produce agglutinins for *Proteus* X strains. It should not be forgotten that the opportunity to study this point in human beings in this country has been limited to one case.

Reactions in guinea pigs.—The clinical pictures in guinea pigs, as described in the literature, are similar, with the exception that the Montana infection has been reported to produce a definite local skin reaction following subcutaneous inoculation, while the Australian workers state that no particular local reaction follows subcutaneous inoculation. A comparison of the two strains in this laboratory shows that the "Q" fever strain produces general reactions in guinea pigs which, although similar to those produced by the Montana virus, are milder. This fact may be explained by the attenuation of the "Q" virus during transit to this country. Rickettsiae have not been ob-

served in guinea pigs with "Q" fever, while they are present in abundance in guinea pigs infected with the Montana virus.

Cross immunity tests.—These tests are identical, with the exception that epidemic typhus, and, to a lesser extent, endemic typhus apparently produce more immunity to "Q" fever than to the Montana virus.

Agglutination of rickettsia.—In one well-controlled test the serum from one recovered case of the Montana infection gave results identical with one serum from a recovered case of "Q" fever when tested with a suspension of *Rickettsia burneti* prepared in Australia.

Protection tests.—As far as these tests have been tried no immunological difference has been noted between the virus of "Q" fever and that isolated from Montana ticks.

CONCLUSION

There are many points yet remaining to be cleared up by further comparative study of the infection isolated from ticks in Montana and "Q" fever. The evidence so far submitted indicates that the two infections are closely related.

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DEATHS DURING WEEK ENDED JUNE 17, 1939

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

	Week ended June 17, 1939	Corresponding week, 1938
Date from 88 large cities of the United States:		
Total deaths	7,601	[1] 7,684
Average for 3 prior years	[2] 7,628	
Total deaths, first 24 weeks of year	214,532	206,773
Deaths under 1 year of age	475	[1] 496
Average for 3 prior years	[2] 486	
Deaths under 1 year of age, first 24 weeks of year	12,598	12,837
Data from industrial insurance companies:		
Policies in force	67,194,608	69,250,632
Number of death claims	10,156	12,077
Death claims per 1,000 policies in force, annual rate	7.9	9.1
Death claims per 1,000 policies, first 24 weeks of year, annual rate	11.3	9.8

[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

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.

Cases of certain diseases reported by telegraph by State health officers for the week ended June 24, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median
NEW ENG.												
Maine	6	1	0	0	----	----	2	1	863	143	53	53
New Hampshire	0	0	0	0	----	----	----	----	223	22	9	9
Vermont	0	0	0	0	----	----	----	----	2,600	194	97	39
Massachusetts	1	1	1	6	----	----	----	----	836	711	521	521
Rhode Island	15	2	0	1	----	----	----	----	664	87	10	43
Connecticut	3	1	3	6	9	3	----	----	1,033	348	69	107
MID. ATL.												
New York	8	19	30	39	[1]3	[1]4	[1]2	[1]2	459	1,146	2,573	1,985
New Jersey [2]	10	8	15	8	7	6	2	2	46	39	332	647
Pennsylvania	8	15	14	37	----	----	----	----	96	189	778	1,362
E. NO. CEN.												
Ohio	3	4	10	17	6	8	----	4	22	29	419	472
Indiana [2]	6	4	6	6	1	1	----	5	13	9	80	80
Illinois	10	16	32	42	10	15	9	9	14	22	422	438
Michigan [2]	8	8	8	8	1	1	1	1	271	256	1,416	288
Wisconsin	0	0	4	4	23	13	11	15	703	400	1,614	1,432
W. NO. CEN.												
Minnesota	4	2	2	2	6	3	----	1	176	91	196	103
Iowa [2]	4	2	0	3	10	5	----	----	170	84	192	41
Missouri	9	7	12	13	----	----	9	23	10	8	27	27
North Dakota	15	2	0	0	124	17	2	1	73	10	41	31
South Dakota	0	0	0	1	8	1	----	----	338	45	----	2
Nebraska	4	1	5	5	----	----	----	----	198	52	75	30
Kansas	8	3	5	5	11	4	----	1	151	54	123	123
SO. ATL.												
Delaware	0	0	0	0	----	----	----	----	177	9	3	15
Maryland [2 3]	3	1	3	4	15	5	1	1	244	79	81	119
Dist. of Col.	8	1	3	6	8	1	----	----	776	96	22	22
Virginia [2]	22	12	6	6	32	17	----	----	463	247	167	167
West Virginia	11	4	6	6	13	5	8	8	30	11	124	100
North Carolina [2 4]	13	9	14	10	----	----	2	1	281	192	696	343
South Carolina [4]	14	5	4	1	295	108	46	52	22	8	48	48
Georgia [2 4]	13	8	6	6	22	13	----	----	70	42	55	----
Florida [4]	12	4	4	5	12	4	----	----	136	45	13	7

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended June 24, 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			
	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median
E. SO. CEN.												
Kentucky	3	2	10	7	10	6	4	3	10	6	63	131
Tennessee [2]	2	1	4	3	18	10	9	13	85	48	44	44
Alabama [4]	5	3	2	8	81	46	3	6	83	47	85	36
Mississippi [2]	0	0	3	5	------	------	------	------	------	------	------	------
W. SO. CEN.												
Arkansas	5	2	1	1	22	9	4	4	27	11	60	9
Louisiana	24	10	8	12	12	5	9	9	56	23	17	9
Oklahoma	0	0	3	3	4	2	15	20	121	60	46	20
Texas [4]	7	9	25	25	42	51	130	66	144	174	67	158
MOUNTAIN												
Montana	9	1	0	0	84	9	------	------	674	72	55	21
Idaho [2]	0	0	0	0	------	------	7	1	357	35	4	5
Wyoming [2]	22	1	0	0	------	------	------	------	873	40	5	5
Colorado [2 3]	48	10	5	3	14	3	------	------	332	69	94	94
New Mexico	62	5	2	2	------	------	1	------	86	7	12	16
Arizona	12	1	3	2	380	31	17	15	147	12	12	12
Utah [2]	0	0	4	0	------	------	------	------	804	81	252	41
PACIFIC												
Washington	0	0	1	1	------	------	------	------	2,618	849	16	178
Oregon	0	0	4	2	55	11	8	8	423	85	33	33
California	18	22	24	31	16	20	11	24	851	1,038	511	511
Total	8	207	292	364	21	437	313	371	296	7,325	11,632	11,632
25 weeks	16	9,980	11,940	12,789	281	149,068	43,332	101,981	532	329,389	730,197	634,539

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median
NEW ENG.												
Maine	0	0	0	0	0	0	0	0	24	4	19	13
New Hampshire	0	0	0	0	0	0	0	0	51	5	8	7
Vermont	0	0	0	0	0	0	0	0	27	2	8	8
Massachusetts	1.2	1	1	1	1.2	1	0	1	114	97	218	155
Rhode Island	0	0	0	0	0	0	0	0	46	6	11	14
Connecticut	0	0	0	0	0	0	1	0	74	25	45	45
MID. ATL.												
New York	0.8	2	4	7	0.4	1	1	3	87	217	350	350
New Jersey [2]	0	0	1	1	0	0	0	0	83	70	50	84
Pennsylvania	4	7	1	7	0.5	1	0	0	88	174	128	359
E. NO. CEN.												
Ohio	0	0	1	4	0	0	2	2	38	50	74	169
Indiana [2]	1.5	1	1	1	1.5	1	0	0	61	41	28	35
Illinois	2	3	4	4	1.3	2	1	1	114	174	173	290
Michigan [2]	1.1	1	0	2	2.1	2	0	0	220	208	309	283
Wisconsin	0	0	5	2	0	0	0	1	128	73	84	242
W. NO. CEN.												
Minnesota	1.9	1	0	0	1.9	1	0	0	37	19	48	58
Iowa [2]	2	1	0	0	0	0	0	0	47	23	22	55
Missouri	0	0	0	1	0	0	0	0	32	25	61	22
North Dakota	0	0	0	0	0	0	1	0	44	6	13	29
South Dakota	8	1	0	0	0	0	2	0	30	4	6	6
Nebraska	0	0	0	0	4	1	0	0	50	13	10	10
Kansas	0	0	1	0	0	0	0	0	92	33	25	25

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended June 24, 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			
	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median
SO. ATL.												
Delaware	0	0	0	0	0	0	0	0	20	1	5	3
Maryland [2 3]	6	2	2	2	0	0	0	0	28	9	45	36
Dist. of Col	0	0	1	1	0	0	0	0	40	5	13	7
Virginia [2]	6	3	3	4	4	2	2	2	21	11	18	12
West Virginia	0	0	1	1	0	0	1	1	22	8	22	24
North Carolina [2 4]	0	0	3	3	4	3	1	1	23	16	13	13
South Carolina [4]	2.7	1	1	0	82	30	0	0	3	1	3	1
Georgia [2 4]	1.7	1	0	0	5	3	3	0	3	2	10	8
Florida [4]	0	0	0	1	3	1	1	1	30	10	8	2
E. SO. CEN.												
Kentucky	0	0	3	3	0	0	1	1	16	9	22	15
Tennessee [2]	0	0	4	2	4	2	1	1	25	14	8	8
Alabama [4]	4	2	4	2	4	2	7	5	25	14	3	3
Mississippi [2]	2.5	1	0	0	0	0	4	0	10	4	0	4
W. SO. CEN.												
Arkansas	0	0	0	0	7	3	0	0	15	6	6	6
Louisiana	0	0	0	1	0	0	4	2	12	5	5	5
Oklahoma	2	1	1	1	2	1	1	1	14	7	14	10
Texas [4]	0	0	2	2	2.5	3	0	0	12	15	64	31
MOUNTAIN												
Montana	0	0	0	0	0	0	0	0	56	6	8	13
Idaho [2]	0	0	0	0	0	0	0	0	20	2	2	2
Wyoming [2]	0	0	0	0	0	0	0	0	44	2	3	3
Colorado [2 5]	0	0	0	0	14	3	0	0	96	20	30	18
New Mexico	0	0	0	0	0	0	0	0	49	4	9	9
Arizona	0	0	0	0	74	6	1	0	12	1	2	6
Utah [3]	0	0	0	0	0	0	0	0	50	5	18	18
PACIFIC												
Washington	0	0	0	0	0	0	0	0	59	19	18	34
Oregon	0	0	0	0	0	0	0	0	30	6	24	23
California	1.6	2	0	4	11	14	2	9	88	107	110	134
Total	1.2	31	44	73	3.3	83	37	82	63	1,578	2,168	2,937
25 weeks	1.8	1,139	1,857	3,546	1.1	713	514	657	174	109,521	128,743	155,134

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases
NEW ENG.											
Maine	0	0	0	0	0	0	1	1	441	73	35
New Hampshire	0	0	0	0	0	0	1	0	183	18	0
Vermont	0	0	0	0	0	0	0	0	335	25	18
Massachusetts	0	0	0	0	1	1	0	2	169	144	93
Rhode Island	0	0	0	0	8	1	1	0	313	41	18
Connecticut	0	0	0	0	3	1	1	1	160	54	96
MID. ATL.											
New York	0	0	0	0	4	10	6	11	145	362	483
New Jersey [3]	0	0	0	0	2	2	6	4	325	273	231
Pennsylvania	0	0	0	0	4	7	6	12	272	536	177

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended June 24, 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		
	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases	1934–38, median	June 24, 1939, rate	June 24, 1939, cases	June 25, 1938, cases
E. NO. CEN.											
Ohio	5	6	0	0	3	4	4	10	84	109	100
Indiana [2]	15	10	29	1	9	6	10	3	111	75	15
Illinois	3	5	8	8	1	2	9	9	203	310	224
Michigan [3]	2	2	2	0	1	1	4	4	171	162	325
Wisconsin	0	0	4	4	2	1	0	3	341	194	206
W. NO. CEN.											
Minnesota	16	8	7	7	0	0	1	1	31	16	44
Iowa [3]	6	3	19	17	4	2	3	1	38	19	14
Missouri	3	2	53	2	8	6	6	9	36	28	37
North Dakota	0	0	1	1	0	0	0	1	66	9	14
South Dakota	0	0	7	7	0	0	1	0	8	1	3
Nebraska	19	5	0	8	0	0	0	0	95	25	1
Kansas	3	1	9	4	8	3	2	3	101	36	167
SO. ATL.											
Delaware	0	0	0	0	0	0	0	0	177	9	12
Maryland [2 3]	0	0	0	0	0	0	3	4	197	64	56
Dist. of Col	0	0	0	0	0	0	0	0	437	54	8
Virginia [2]	0	0	0	0	36	19	5	7	240	128	99
West Virginia	3	1	1	0	32	12	5	5	32	12	77
North Carolina [2 4]	0	0	3	1	16	11	33	13	390	267	349
South Carolina [4]	0	0	0	0	30	11	32	26	197	72	79
Georgia [2 4]	0	0	0	0	58	35	50	50	78	47	55
Florida [4]	0	0	0	0	6	2	0	1	72	24	14
E. SO. CEN.											
Kentucky	0	0	0	0	19	11	18	18	76	44	43
Tennessee [2]	14	8	1	0	18	10	24	17	120	68	44
Alabama [4]	2	1	0	0	14	8	13	17	99	56	64
Mississippi [3]	0	0	2	0	8	3	18	11	0	-------	-------
W. SO. CEN.											
Arkansas	10	4	4	0	22	9	15	15	50	20	25
Louisiana	0	0	0	0	53	22	22	21	89	37	38
Oklahoma	16	8	3	2	16	8	10	10	8	4	51
Texas [4]	0	0	8	3	20	24	42	26	73	88	246
MOUNTAIN											
Montana	9	1	1	3	19	2	0	2	131	14	26
Idaho [2]	0	0	6	4	0	0	4	1	10	1	7
Wyoming [2]	22	1	1	2	0	0	0	0	44	2	6
Colorado [2 5]	14	3	0	1	29	6	7	1	241	50	23
New Mexico	12	1	6	0	12	1	2	4	408	33	23
Arizona	0	0	8	0	12	1	3	3	380	31	45
Utah [3]	0	0	0	0	10	1	1	1	457	46	69
PACIFIC											
Washington	0	0	17	6	56	18	1	1	43	14	65
Oregon	50	10	19	4	0	0	1	2	104	21	48
California	10	12	7	7	3	4	5	10	120	146	244
Total	4	92	225	144	11	265	376	371	156	3,862	4,117
25 weeks	13	8,164	11,750	5,398	6	3,498	3,939	3,940	158	98,028	107,601

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended June 24, 1939, 21 cases as follows: New Jersey, 1; Indiana, 1; Iowa, 2; Maryland, 4; Virginia, 2; North Carolina, 2; Georgia, 1; Tennessee, 2; Idaho, 1; Wyoming, 2; Colorado, 3.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended June 24, 1939, 54 cases as follows: North Carolina, 1; South Carolina, 1; Georgia, 24; Florida, 2; Alabama, 16; Texas, 10.
[5] Colorado tick fever, week ended June 24, 1939, Colorado, 5 cases.

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
May 1939										
Alabama	5	14	832	361	734	20	2	19	3	22
California	7	127	237	17	13,052	8	32	766	65	46
Colorado	3	52	27	----	1,452	----	0	192	18	7
Florida	2	10	134	33	549	50	11	30	0	26
Georgia	0	32	618	165	460	55	11	39	3	23
Illinois	5	108	197	15	191	1	5	1,723	52	17
Kansas	2	17	17	2	355	1	2	224	39	4
Louisiana	5	41	53	82	401	8	3	45	2	48
Massachusetts	8	22	----	----	4,913	----	0	735	0	3
Montana	1	11	148	----	751	----	1	63	6	4
Nevada	0	0	9	----	272	----	0	1	0	0
Ohio	6	68	159	----	220	----	2	1,538	76	40
Oklahoma	2	23	316	155	1,010	27	1	82	137	27
Rhode Island	2	4	----	----	577	----	0	48	0	5
South Dakota	0	3	50	----	1,459	----	1	68	89	0
Utah	0	2	46	----	483	----	0	99	1	5
Washington	1	10	17	----	4,898	----	0	166	13	9

May 1939

	Cases
Actinomycosis:	
Utah	1
Anthrax:	
California	1
Beriberi:	
California	1
Chickenpox:	
Alabama	95
California	3,367
Colorado	342
Florida	99
Georgia	183
Illinois	1,937
Kansas	425
Louisiana	63
Massachusetts	741
Montana	153
Ohio	1,603
Oklahoma	69
Rhode Island	125
South Dakota	42
Utah	288
Washington	770
Colorado tick fever:	
Colorado	36
Conjunctivitis, infectious:	
Georgia	3
Utah	2
Dengue:	
Florida	1
Diarrhea (enteritis included):	
Ohio (under 2 years)	20
Washington (under 2 years)	1
Washington (over 2 years)	3
Dysentery:	
California (amoebic)	13
California (bacillary)	41
Colorado (amoebic)	1
Florida (amoebic)	2
Florida (bacillary)	1
Georgia (amoebic)	6
Georgia (bacillary)	45
Illinois (amoebic)	4
Illinois (amoebic carriers)	20
Illinois (bacillary)	14
Louisiana (amoebic)	5

May 1939—Continued

	Cases
Dysentery—Continued.	
Louisiana (bacillary)	1
Massachusetts (bacillary)	5
Ohio (bacillary)	3
Oklahoma (bacillary)	13
Utah (amoebic)	2
Encephalitis, epidemic or lethargic:	
Alabama	1
California	1
Colorado	2
Illinois	2
Kansas	5
Louisiana	1
Massachusetts	3
Ohio	1
Oklahoma	2
Washington	1
Food poisoning:	
California	65
Kansas	5
German measles:	
Alabama	11
California	175
Illinois	52
Massachusetts	90
Ohio	18
Rhode Island	11
Utah	18
Washington	23
Granuloma, coccidioidal:	
California	4
Hookworm disease:	
Florida	341
Georgia	1,056
Louisiana	164
Impetigo contagiosa:	
Illinois	2
Kansas	9
Montana	5
Ohio	29
Rhode Island	1
Jaundice, infectious:	
California	9
Lead poisoning:	
Ohio	7
Leprosy:	
Louisiana	1

May 1939—Continued

	Cases
Mumps:	
Alabama	192
California	3,614
Colorado	27
Florida	67
Georgia	169
Illinois	753
Kansas	868
Louisiana	6
Massachusetts	679
Montana	58
Nevada	18
Ohio	2,384
Oklahoma	32
Rhode Island	334
South Dakota	39
Utah	895
Washington	222
Ophthalmia neonatorum:	
California	1
Illinois	1
Louisiana	1
Massachusetts	64
Puerperal septicemia:	
Ohio	2
Rabies in animals:	
Alabama	21
California	88
Florida	1
Illinois	29
Louisiana	8
Oklahoma	23
Washington	46
Rabies in man:	
Kansas	2
Rocky Mountain spotted fever:	
Colorado	9
Illinois	2
Montana	10
Nevada	4
Utah	9
Washington	4
Scabies:	
Kansas	5
Montana	2
Septic sore throat:	
California	8
Colorado	8
Florida	7

Summary of monthly reports from States—Continued

May 1939—Continued	Cases
Septic sore throat—Con.	
Georgia	70
Illinois	9
Kansas	18
Louisiana	1
Massachusetts	25
Montana	5
Ohio	13
Oklahoma	95
Rhode Island	17
South Dakota	4
Washington	6
Tetanus:	
Alabama	1
California	8
Florida	1
Georgia	2
Illinois	9
Louisiana	3
Massachusetts	2
Ohio	1
South Dakota	1
Trachoma:	
California	10
Illinois	31
Ohio	3
Oklahoma	5
Trichinosis:	
California	3
Illinois	1
Massachusetts	1
Tularaemia:	
Alabama	8
Colorado	1
Georgia	12
Illinois	4
Louisiana	1
Oklahoma	1
Utah	7
Washington	1
Typhus fever:	
Alabama	31
California	1
Florida	11
Georgia	66
Louisiana	5
Undulant fever:	
Alabama	4
California	19
Colorado	4
Florida	1
Georgia	16
Illinois	22
Kansas	3
Louisiana	5
Massachusetts	4
Montana	1
Ohio	8
Oklahoma	115
Utah	5
Vincent's infection:	
Florida	10
Illinois	20
Kansas	9
Montana	1
Oklahoma	11
Washington	1
Whooping cough:	
Alabama	220
California	1,093
Colorado	271
Florida	209
Georgia	219
Illinois	1,001
Kansas	131
Louisiana	90
Massachusetts	635
Montana	76
Nevada	4
Ohio	805
Oklahoma	55
Rhode Island	356
South Dakota	20
Utah	306
Washington	92

PLAGUE INFECTION IN CALIFORNIA AND WASHINGTON

IN A RABBIT AND IN FLEAS FROM GROUND SQUIRRELS IN LINCOLN COUNTY, WASH.

Under date of June 19, 1939, Senior Surgeon C. R. Eskey reported plague infection proved in a pool of 45 fleas from 16 ground squirrels, *C. townsendi*, shot 6 miles north of Odessa, Lincoln County, Wash., on May 25, and in tissue from 1 cottontail rabbit and a pool of 44 fleas from 21 *C. townsendi* taken May 27, at a location 8 miles northwest of Odessa. This is stated to be the first demonstration of plague infection in a rabbit in nature.

IN FLEAS FROM GROUND SQUIRRELS IN VENTURA COUNTY, CALIF.

Under date of June 23, 1939, Dr. W. M. Dickie, State Director of Public Health of California, reported plague infection proved in a pool of 151 fleas from 10 ground squirrels, *C. beecheyi*, submitted to the laboratory on June 8 from an estate 5 miles northwest of Ventura, in Mills Canyon, Ventura County, Calif.

TULARAEMIA FROM MUSKRAT BITE REPORTED IN NEW YORK STATE

A definite case of tularaemia, clinically typical of the ulceroglandular type, in which the patient's blood serum gave an agglutination reaction with *B. tularense* in a dilution of 1:2,560, was reported in a resident of Oswego County, N. Y., according to Health News for June 19, 1939, issued by the New York State Department of Health. The patient, a trapper, gave a history of having been bitten by a muskrat on April 10, 1939, and developed first symptoms on April 13.

This case is of interest in that it is the first time that a case of tularaemia resulting from the bite of a muskrat has been recognized in New York State. In investigating this case it was learned that a second trapper had had sores on his arms and hands during the past trapping season as well as at intervals during the past 10 years of his trapping experience. A sample of blood was obtained from the second trapper, and the serum agglutinated *B. tularense* in a dilution of 1:40.

In a report by Francis [1] on 6,000 cases of tularaemia reported in the United States through 1935, only 2 cases were attributed to contact with the muskrat.

WEEKLY REPORTS FROM CITIES

City reports for week ended June 17, 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		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	140	48	21	4,120	420	1,318	13	388	44	1,016	----
Current week	67	49	14	2,571	228	676	8	327	38	1,082	----
Maine:											
Portland	0	----	0	1	0	0	0	0	0	16	20
New Hampshire:											
Concord	0	----	0	0	0	0	0	0	0	0	14
Manchester	0	----	1	0	1	0	0	0	0	0	15
Nashua	0	----	0	1	0	1	0	0	0	0	5
Vermont:											
Barre	0	----	0	0	1	0	0	0	0	9	3
Burlington	0	----	0	14	0	0	0	0	0	0	9
Rutland	0	----	0	0	0	0	0	0	0	0	6
Massachusetts:											
Boston	1	----	0	184	15	38	0	7	2	26	180
Fall River	0	----	0	1	0	0	0	2	0	0	37
Springfield	0	----	0	7	0	1	0	0	1	2	27
Worcester	0	----	0	30	1	10	0	1	0	7	35
Rhode Island:											
Pawtucket	0	----	0	11	0	0	0	0	2	1	16
Providence	1	14	0	68	2	2	0	1	0	37	70
Connecticut:											
Bridgeport	0	----	0	3	1	1	0	1	0	0	40
Hartford	0	----	1	7	1	4	0	0	0	8	42
New Haven	0	----	0	143	0	2	0	1	0	7	36
New York:											
Buffalo	0	----	0	81	5	19	0	6	0	13	103
New York	13	6	2	162	49	101	0	66	5	92	1,390
Rochester	0	2	0	83	1	10	0	0	0	6	58
Syracuse	0	----	0	126	1	10	1	2	1	21	47
New Jersey:											
Camden	2	----	0	0	1	2	0	0	0	4	30
Newark	1	----	0	2	4	20	0	1	0	51	76
Trenton	0	----	0	0	2	3	0	0	0	1	39
Pennsylvania:											
Philadelphia	2	----	0	34	15	21	0	21	2	117	408
Pittsburgh	2	1	0	0	2	22	0	4	0	41	134
Reading	0	----	0	3	1	0	0	2	0	2	27
Scranton	0	----	----	0	----	7	0	----	0	1	----
Ohio:											
Cincinnati	2	----	0	0	4	3	0	6	0	3	131
Cleveland	3	2	0	4	9	33	0	10	0	53	178
Columbus	2	1	1	4	0	2	0	2	0	7	84
Toledo	0	----	0	42	2	7	0	2	0	34	52

[1] Francis, Edward: Sources of infection and seasonal incidence of tularaemia in man. Pub. Health Rep., 52: 103 (January 22, 1937).

City reports for week ended June 17, 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								
Indiana:											
Anderson	0	------	0	0	0	1	0	0	0	2	7
Fort Wayne	0	------	0	0	0	4	0	1	0	0	30
Indianapolis	2	------	0	0	5	9	0	6	0	23	99
Muncie	0	------	0	0	0	0	0	0	0	0	8
South Bend	0	------	0	0	2	0	0	1	0	9	13
Terre Haute	0	------	0	0	1	0	0	1	2	0	11
Illinois:											
Alton	0	------	0	0	0	0	0	0	0	0	8
Chicago	9	8	1	12	13	118	0	52	1	74	622
Elgin	0	------	0	0	0	0	0	0	0	1	6
Moline	0	------	0	0	0	1	0	0	0	1	5
Springfield	0	------	0	0	0	0	0	0	0	6	27
Michigan:											
Detroit	2	------	0	49	4	66	0	16	0	67	210
Flint	1	------	0	25	1	5	0	0	0	0	15
Grand Rapids	0	------	0	2	0	17	0	0	0	0	24
Wisconsin:											
Kenosha	0	------	0	0	0	0	0	0	0	4	11
Madison	0	------	0	55	1	2	0	0	0	11	16
Milwaukee	0	1	1	2	3	16	0	3	0	23	92
Racine	0	------	0	1	0	2	0	0	0	2	12
Superior	0	------	0	12	0	0	0	0	0	0	6
Minnesota:											
Duluth	0	------	0	2	0	0	0	0	0	0	25
Minneapolis	0	------	0	22	2	8	0	0	0	14	87
St. Paul	0	------	0	10	0	1	0	1	0	12	59
Iowa:											
Cedar Rapids	0	------	--------	3	-------	0	0	-------	0	3	--------
Davenport	0	------	--------	0	-------	2	1	-------	0	1	--------
Des Moines	0	------	0	4	0	6	4	0	0	0	28
Sioux City	0	------	--------	1	-------	0	0	------	0	6	--------
Waterloo	1	------	--------	2	-------	1	0	-------	0	0	--------
Missouri:											
Kansas City	0	------	1	1	2	5	0	2	0	3	86
St. Joseph	0	------	0	0	0	1	0	0	0	2	23
St. Louis	1	------	1	2	2	15	0	3	1	21	164
North Dakota:											
Fargo	0	------	0	2	0	0	0	0	0	0	7
Grand Forks	0	------	--------	0	-------	0	0	-------	0	0	--------
Minot	0	------	0	1	0	0	0	0	0	0	6
South Dakota:											
Aberdeen	0	------	--------	5	-------	0	4	-------	0	0	--------
Nebraska:											
Lincoln	0	------	--------	9	-------	0	0	-------	0	28	--------
Omaha	0	------	0	6	2	0	0	2	0	0	47
Kansas:											
Lawrence	0	------	0	0	0	0	0	0	0	0	4
Topeka	0	------	0	2	0	8	4	0	0	2	18
Wichita	1	------	0	16	2	1	0	0	0	1	21
Delaware:											
Wilmington	0	------	0	4	0	4	0	1	0	4	2
Maryland:											
Baltimore	2	2	1	45	3	3	0	10	0	38	189
Cumberland	0	------	0	0	1	0	0	1	0	0	9
Frederick	0	------	0	0	1	0	0	0	0	0	2
Dist. of Columbia:											
Washington	0	------	0	144	2	4	0	6	3	30	165
Virginia:											
Lynchburg	0	------	0	25	0	2	0	0	0	47	4
Norfolk	0	------	0	0	3	1	0	1	0	2	20
Richmond	1	------	0	105	3	3	0	1	0	0	50
Roanoke	0	------	0	3	0	0	0	0	0	5	9
West Virginia:											
Charleston	0	------	0	0	3	0	0	0	1	0	30
Huntington	0	------	--------	0	-------	0	0	-------	0	0	--------
Wheeling	0	------	--------	1	-------	1	0	-------	1	5	--------
North Carolina:											
Gastonia	0	------	--------	1	-------	0	0	-------	0	0	--------
Raleigh	1	------	0	0	2	0	0	0	0	8	15
Wilmington	0	------	0	1	1	0	0	0	0	2	10
Winston-Salem	0	------	0	0	0	0	0	3	1	2	19

City reports for week ended June 17, 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								
South Carolina:											
Charleston	0	1	0	0	1	0	0	1	0	0	20
Florence	0	------	0	0	0	0	0	0	0	0	14
Greenville	0	------	0	0	2	0	0	0	0	2	13
Georgia:											
Atlanta	1	7	0	3	2	3	0	5	2	2	70
Brunswick	0	------	0	3	1	0	0	0	0	2	5
Savannah	0	1	0	0	0	0	0	3	1	3	40
Florida:											
Miami	0	------	0	0	2	1	0	5	0	6	44
Tampa	0	1	1	28	0	1	0	0	0	0	22
Kentucky:											
Ashland	0	------	0	0	1	0	0	0	0	0	6
Covington	0	------	0	0	0	0	0	3	0	0	10
Lexington	0	------	0	0	0	0	0	0	0	0	19
Louisville	0	------	0	4	4	6	0	4	0	5	71
Tennessee:											
Knoxville	0	------	1	4	1	3	0	1	0	0	29
Memphis	0	------	1	1	0	0	0	4	2	36	67
Nashville	0	------	0	2	0	1	0	0	0	1	47
Alabama:											
Birmingham	0	1	1	0	1	2	0	3	0	4	60
Mobile	0	------	0	2	0	0	0	0	1	0	12
Montgomery	0	------	--------	0	-------	0	0	-------	0	0	--------
Arkansas:											
Fort Smith	0	------	--------	1	-------	0	0	-------	0	0	--------
Little Rock	0	------	0	0	2	0	0	2	0	0	4
Louisiana:											
Lake Charles	0	------	0	1	0	0	0	0	0	0	5
New Orleans	4	------	0	0	13	4	0	10	5	0	123
Shreveport	0	------	0	1	3	1	0	4	0	0	60
Oklahoma:											
Oklahoma City	0	------	0	8	1	1	0	1	0	0	52
Tulsa	6	------	--------	0	-------	2	--------	-------	0	0	--------
Texas:											
Dallas	2	------	0	9	4	3	0	3	0	1	56
Fort Worth	0	------	0	3	3	2	0	1	1	1	31
Galveston	0	------	0	1	2	0	0	2	0	0	13
Houston	3	------	0	7	4	0	0	3	1	1	68
San Antonio	0	------	0	1	0	1	0	13	1	0	72
Montana:											
Billings	0	------	0	1	1	0	0	0	0	0	9
Great Falls	0	------	0	50	0	1	0	0	0	0	7
Helena	0	------	0	1	0	0	0	0	0	0	1
Missoula	0	------	0	6	0	0	0	0	0	0	11
Idaho:											
Boise	0	------	0	0	1	0	0	0	0	1	5
Colorado:											
Colorado Springs	0	------	0	2	0	0	0	2	0	0	16
Denver	1	------	1	13	3	4	0	3	0	15	75
Pueblo	0	------	0	17	1	1	0	1	0	24	4
New Mexico:											
Albuquerque	0	------	0	0	0	0	0	1	0	8	11
Utah:											
Salt Lake City	0	------	0	11	1	7	0	1	0	21	31
Washington:											
Seattle	0	------	0	357	4	3	0	1	0	3	88
Spokane	0	------	0	46	2	6	0	0	0	1	23
Tacoma	0	------	0	4	0	1	0	1	1	0	23
Oregon:											
Portland	0	2	0	6	3	7	0	3	0	0	84
Salem	0	4	--------	1	-------	0	0	-------	0	0	--------
California:											
Los Angeles	4	5	0	258	6	26	0	20	3	22	297
Sacramento	2	------	0	42	4	3	3	1	0	2	27
San Francisco	1	1	1	6	2	11	0	3	0	16	133

City reports for week ended June 17, 1939—Continued

State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Worcester	0	1	0
New York:			
New York	2	0	1
Pennsylvania:			
Philadelphia	1	0	0
Scranton	0	0	1
Ohio:			
Cleveland	1	0	0
Indiana:			
South Bend	0	0	1
Nebraska:			
Omaha	1	0	0
North Carolina:			
Winston-Salem	0	1	0
South Carolina:			
Charleston	0	0	10
Greenville	0	1	0
Georgia:			
Savannah	0	0	1
Tennessee:			
Memphis	1	0	0
California:			
Los Angeles	0	0	1

Encephalitis, epidemic or lethargic.—Cases: Sacramento, 1.

Pellagra.—Cases: Philadelphia, 1; Columbus, 1; Baltimore, 1; Lynchburg, 2; Charleston, S. C., 3; Atlanta, 2; Savannah, 2; Louisville, 1; San Francisco, 1.

Typhus fever.—Cases: New York, 2; Charleston, S. C., 1; Atlanta, 2; Miami, 2; Mobile, 2.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended June 3, 1939.—During the week ended June 3, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				1						1
Chickenpox		9	12	131	166	29	11	13	69	440
Diphtheria		3	2	38	2	6	3			54
Dysentery				6	1					7
Influenza		69			6				26	101
Measles		15		961	936	18		27	11	1,968
Mumps	4	7		56	73	27		6	2	175
Pneumonia	1	13			16				6	36
Poliomyelitis					1					1
Scarlet fever		5	16	61	122	4	4	14	11	237
Tuberculosis	5	22	35	87	61	51		3		264
Typhoid and paratyphoid fever				15	5		1		1	22
Whooping cough	2	81		72	96	12	26	3	30	322

Vital statistics—Fourth quarter 1938 and year 1938.—The Bureau of Statistics of the Dominion of Canada has published the following preliminary statistics for the fourth quarter of 1938. The rates are computed on an annual basis. There were 19.0 live births per 1,000 population during the fourth quarter of 1938 as compared with 18.3 per 1,000 population during the fourth quarter of 1937. The death rate was 9.3 per 1,000 population for the fourth quarter of 1938 and 9.8 per 1,000 population for the corresponding quarter of 1937. The infant mortality rate for the fourth quarter of 1938 was 63 per 1,000 live births and 72 per 1,000 live births for the fourth quarter of 1937. The maternal death rate was 3.8 per 1,000 live births for the fourth quarter of 1938 and 4.4 per 1,000 live births for the same quarter of 1937.

The accompanying tables give the numbers of births, deaths, and marriages, by Provinces, for the fourth quarter of 1938 and the year 1938, and deaths by causes in Canada for the fourth quarter of 1938 and the corresponding quarter of 1937, and for the years 1938 and 1937.

Number of births, deaths, and marriages, fourth quarter 1938

Province	Live births	Deaths (exclusive of still-births)	Deaths under 1 year of age	Maternal deaths	Marriages
Canada [1]	53,542	26,228	3,374	205	25,389
Prince Edward Island	452	256	27	----------	217
Nova Scotia	2,549	1,216	172	7	1,136
New Brunswick	2,624	1,211	203	11	914
Quebec	18,357	7,795	1,429	85	5,885
Ontario	15,484	9,223	779	58	8,051
Manitoba	3,205	1,530	186	5	2,062
Saskatchewan	4,158	1,542	238	16	2,701
Alberta	3,809	1,471	202	16	2,636
British Columbia	2,904	1,984	138	7	1,787

[1] Exclusive of Yukon and the Northwest Territories.

Deaths by cause, fourth quarter, 1938

Cause of death	Canada [1] (fourth quarter)		Province								
	1937	1938	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia
Automobile accidents	458	499	4	24	17	168	198	19	12	25	32
Cancer	3,051	3,032	33	146	111	798	1,114	200	172	167	291
Cerebral hemorrhage, cerebral embolism and thrombosis	516	511	8	35	43	117	196	25	25	29	33
Diarrhea and enteritis	885	601	6	22	22	296	116	41	51	34	13
Diphtheria	158	143	------	8	16	95	2	7	8	6	1
Diseases of the arteries	2,417	2,526	34	115	96	459	1,189	167	139	127	200
Diseases of the heart	4,361	4,478	29	190	173	1,068	1,928	264	231	243	352
Homicides	37	19	------	------	------	6	7	1	2	------	3
Influenza	637	506	2	17	16	193	146	35	35	29	33
Measles	155	37	------	1	------	26	3	------	3	2	2
Nephritis	1,570	1,598	28	66	44	693	486	38	74	64	105
Pneumonia	1,991	1,897	18	109	149	474	647	116	115	138	131
Poliomyelitis	36	18	------	------	1	6	5	2	------	3	1
Puerperal causes	226	205	------	7	11	85	58	5	16	16	7
Scarlet fever	78	52	------	------	1	26	13	2	4	3	3
Smallpox	------	2	------	------	------	------	------	------	2	------	------
Suicides	223	213	1	9	4	22	78	21	26	20	32
Tuberculosis	1,386	1,382	17	70	85	603	299	74	62	56	116
Typhoid fever and paratyphoid fever	149	50	------	------	3	21	12	7	3	1	3
Violence	1,042	1,000	7	51	37	241	365	55	56	67	121
Other specified causes	------	7,209	65	336	342	2,294	2,330	431	495	429	487
Unspecified or ill-defined causes	------	147	4	8	29	61	15	6	8	5	11
Whooping cough	201	103	------	2	11	43	16	14	3	7	7

[1] Exclusive of Yukon and the Northwest Territories.

Number of births, deaths, and marriages, year 1938

Province	Live births	Deaths (exclusive of still-births)	Deaths under 1 year of age	Maternal deaths	Marriages
Canada [1]	228,060	106,262	14,431	957	88,337
Prince Edward Island	1,957	1,015	112	5	591
Nova Scotia	11,614	5,750	719	42	4,060
New Brunswick	11,399	4,864	851	52	3,363
Quebec	77,985	32,586	6,480	406	25,036
Ontario	65,375	36,862	3,244	251	30,080
Manitoba	13,478	5,893	750	39	6,262
Saskatchewan	18,065	6,003	915	46	5,853
Alberta	15,819	5,861	811	68	6,960
British Columbia	12,368	7,428	549	48	6,132

[1] Exclusive of Yukon and the Northwest Territories.

Deaths by cause, year 1938, comparative

Cause of death	Canada [1]		Province								
	1937	1938	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia
Automobile accidents	1,633	1,535	6	66	58	413	677	80	48	77	110
Cancer	11,963	11,980	136	652	459	3,189	4,466	774	640	680	984
Cerebral hemorrhage, cerebral embolism and thrombosis	2,005	1,999	28	181	160	431	789	81	110	102	117
Diarrhea and enteritis	4,216	2,581	17	98	107	1,344	508	161	170	116	60
Diphtheria	369	432		23	31	302	11	16	27	18	4
Diseases of the arteries	9,609	9,932	96	557	391	1,835	4,741	601	502	455	754
Diseases of the heart	16,840	17,298	142	821	676	4,127	7,348	961	980	895	1,348
Homicides	138	125		2	4	26	44	7	13	14	15
Influenza	5,260	2,350	13	132	63	951	618	137	151	174	111
Measles	837	250	1	15	7	133	31	3	19	30	11
Nephritis	6,530	6,460	81	316	187	2,881	1,899	217	267	243	369
Pneumonia	7,731	7,397	101	472	506	2,055	2,493	408	426	486	450
Poliomyelitis	200	83		1	4	16	25	11	7	15	4
Puerperal causes		957	5	42	52	406	251	39	46	68	48
Scarlet fever	269	200		4	2	99	48	6	12	25	4
Smallpox	2	3		1					2		
Suicides	978	944	3	42	20	134	359	91	77	99	119
Tuberculosis	6,669	6,087	80	390	339	2,615	1,236	349	269	279	530
Typhoid fever	330	206	1	7	18	102	39	11	8	12	8
Violence	4,609	4,542	26	234	172	1,184	1,668	266	257	266	469
Other specified causes		29,807	262	1,633	1,441	9,879	9,462	1,626	1,922	1,747	1,835
Unspecified or ill-defined causes		601	17	55	146	194	61	17	32	30	49
Whooping cough	763	493		6	21	270	88	31	18	30	29

[1] Exclusive of Yukon and the Northwest Territories.

CUBA

Habana—Communicable diseases—4 weeks ended June 3, 1939.—During the 4 weeks ended June 3, 1939, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	4		Scarlet fever	4	
Lethargic encephalitis	1	1	Tuberculosis	6	1
Malaria	6		Typhoid fever	24	7

Provinces—Notifiable diseases—4 weeks ended May 27, 1939.—During the 4 weeks ended May 27, 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	8		6	1	3	15
Chickenpox		3				1	4
Diphtheria		2	1	4	5	1	13
Leprosy		2	1	1		5	9
Lethargic encephalitis		1					1
Malaria	18	9	1	15	7	21	71
Measles				1		4	5
Scarlet fever		8				1	9
Tuberculosis	29	42	26	49	25	44	215
Typhoid fever	21	48	6	28	7	26	136
Whooping cough				2			2

IRISH FREE STATE

Vital statistics—Quarter ended March 31, 1939.—The following vital statistics for the Irish Free State for the quarter ended March 31, 1939, are taken from the Quarterly Return of Marriages, Births, and Deaths, issued by the Registrar General and are provisional:

	Number	Rate per 1,000 population		Number	Rate per 1,000 population
Marriages	3,485	4.7	Deaths from—Continued		
Births	14,207	19.4	Influenza	685	0.9
Total deaths	13,318	18.2	Measles	20	----------
Deaths under 1 year of age	1,158	[1] 82	Puerperal sepsis	8	1.6
Deaths from:			Scarlet fever	16	----------
Cancer	910	1.2	Tuberculosis (all forms)	882	1.2
Diarrhea and enteritis (under 2 years)	146	----------	Typhoid fever	13	----------
Diphtheria	86	----------	Whooping cough	66	----------

[1] Per 1,000 live births.

ITALY

Communicable diseases—4 weeks ended March 26, 1939.—During the 4 weeks ended March 26, 1939, cases of certain communicable diseases were reported in Italy as follows:

Disease	Feb. 27–Mar. 5	Mar. 6–12	Mar. 13–19	Mar. 20–26
Anthrax	7	4	5	7
Cerebrospinal meningitis	46	41	42	39
Chickenpox	413	387	443	383
Diphtheria	552	520	544	490
Dysentery (amoebic)	14	17	25	12
Dysentery (bacillary)	2	----------	----------	1
Hookworm disease	26	16	18	27
Lethargic encephalitis	4	3	1	2
Measles	1,423	1,499	1,445	1,468
Mumps	280	233	285	237
Paratyphoid fever	37	41	29	28
Pellagra	4	1	----------	7
Poliomyelitis	20	27	29	20
Puerperal fever	35	40	36	23
Scarlet fever	229	221	213	263
Typhoid fever	372	321	300	232
Undulant fever	96	93	96	94
Whooping cough	343	338	375	314

JAMAICA

Communicable diseases—4 weeks ended June 10, 1939.—During the 4 weeks ended June 10, 1939, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	11	37	Leprosy	----------	2
Diphtheria	2	5	Puerperal fever	----------	2
Dysentery	3	6	Tuberculosis	38	74
Erysipelas	----------	1	Typhoid fever	7	43

PANAMA CANAL ZONE

Notifiable diseases—January–March 1939.—During the months of January, February, and March 1939, certain notifiable diseases, including imported cases, were reported in the Panama Canal Zone and terminal cities as follows:

Disease	January		February		March	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Chickenpox	44		52		78	
Diphtheria	18		6		7	
Dysentery (amoebic)	19	3	9		8	
Dysentery (bacillary)	5	4	13	1	7	2
Leprosy	1	1		1		
Malaria	100	4	67	2	32	2
Measles	1		2		2	
Meningococcus meningitis	1		1			
Mumps	1				1	
Pneumonia		39		20		22
Poliomyelitis			1		1	1
Tuberculosis		30		22		31
Typhoid fever	6		3		2	
Undulant fever					1	
Whooping cough			[1] 2		[1] 1	

[1] In the Canal Zone only.

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 June 30, 1939, pages 1182–1194. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

India—Madras.—During the week ended June 17, 1939, 1 case of cholera was reported in Madras, India.

Plague

Bolivia—Santa Cruz Department—Sara Province.—During the month of March 1939, 1 case of pneumonic plague was reported in Sara Province, Santa Cruz Department, Bolivia.

Peru.—During the month of April 1939, plague was reported in Peru as follows: Lambayeque Department, 1 case, 1 death; Libertad Department, 2 cases; Lima Department, 1 case; Piura Department, 7 cases, 1 death.

United States.—A report of plague infection in Lincoln County, Washington, and in Ventura County, California, appears on page 1244 of this issue of PUBLIC HEALTH REPORTS.

Smallpox

Bolivia.—During the month of March 1939, smallpox was reported in Bolivia as follows: Cochabamba Department, 8 cases; La Paz Department, 8 cases, including 7 cases in La Paz; Oruro Department, 1 case; Potosi Department, 1 case; Santa Cruz Department, 6 cases.

Typhus Fever

Bolivia.—During the month of March 1939, typhus fever was reported in Bolivia as follows: Cochabamba Department, 1 case; Potosi Department, 7 cases; Tarija Department, 1 case.

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