# PUBLIC HEALTH REPORTS 

## SICKNESS AMONG MALE INDUSTRIAL EMPLOYEES DURING THE FIRST THREE MONTHS OF $1935{ }^{1}$

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The rate of occurrence of new cases of sickness and nonindustrial accidents causing disability for 8 consecutive days or longer among 157,662 male industrial employees was 8 percent higher in the first 3 months of 1935 than in the corresponding period of 1934 . For these 2 years the experience of employees of the same group of companies, 33 in number, is under comparison. Although the rate was somewhat higher in 1935 than in the preceding year, it was 15 percent below the 5 -year average for the first quarter. This period of the year is usually the worst from the standpoint of disease prevalence; hence the rate of 100.7 cases per 1,000 men recorded for the first 3 months of 1935 may be regarded as definitely favorable in comparison with rates of 113.4 and 112.4 for the full years 1928 and 1929, respectively, or even with 97.5 for the year 1932 as a whole.

The higher rate in the first quarter of 1935 as compared with the corresponding period of 1934 was principally due to an increase in the frequency of influenza. The incidence rate of this disease was 27.0 cases per 1,000 men per year, as compared with 17.7 in the first quarter of 1934, an increase of 52 percent. Pneumonia also was more frequent, the rate being 31 percent higher than in the same months of the preceding year. A relatively small increase, 16 percent, occurred in the frequency of 8-day and longer cases of tonsillitis and other diseases of the pharynx and tonsils. Bronchitis, pulmonary tuberculosis, and "other" respiratory diseases occurred at approximately the same rate as in the first quarter of 1934.

[^0]Table 1.-Frequency of disability lasting 8 calendar days or longer in the first quarter of 1935, compared with the first quarter of preceding years. (Male morbidity experience of industrial companies which reported their cases to the United States Public Health Service) ${ }^{1}$

| Diseases and disease groups which caused disability. (Numbers in parentheses are disease title numbers from the International List of the Causes of Death, fourth revision, Paris, 1929) | Annual number of disabilities per 1,000 men in the Arst quarter of - |  |  |
| :---: | :---: | :---: | :---: |
|  | 1935 | 1934 | $\begin{gathered} \text { Five } \\ \text { years, } \\ \text { 1930-34 } \end{gathered}$ |
|  | 100.7 | 93.0 | 118.1 |
| Nonindustrial injuries. | 10.2 | 11.8 | 11.2 |
| Sickness ${ }^{2}$ - | 90.5 | 81.2 | 106.9 |
| Respiratory diseases. | 47.0 | 36.2 | 56.4 |
| Bronchitis, acute and chronic (108) | 4.5 | 4.7 | 5.6 |
| Diseases of the pharynx and tonsils (115a) | 5.1 | 4.4 | 6.4 |
| Influenza, grippe (11). | 27.0 | 17.7 | 34.1 |
| Pneumonia, all forms (107-109) -----...- | 3.8 | 2.9 | 3.5 |
| Tuberculosis of the respiratory system (23) | 1.0 | . 9 | 1.1 |
| Other reepiratory diseases (104, 105, 110-114) | 5.6 | 5.6 | 5.7 |
| Nomrespiratory diseases. | 43.5 | 45.0 | 50.6 |
| Diseases of the stomach, eancer excepted (117-118) | 3.5 | 3.4 | 4.0 |
| Diarrhea and enteritis (120) | . 9 | . 9 | . 9 |
| Appendicitis (121) | 3.7 | 4.1 | 3.8 |
| Hernia (1228) | 1.3 | 1.4 | 1.8 |
| Other digestive diseases (115b, 116, 1226-129) | 3.1 | 2.9 | 3.2 |
| Rheumatic group, total. | 9.8 | 9.8 | 12.5 |
| Rheumatism, acute and chronic (56,57) | 4.4 | 4.8 | 6.4 |
| Diseases of the organs of locomotion (156b) | 2.9 | 2.9 | 8.6 |
| Neuralgia, neuritis, seiatica (87a)....-...-- | 2.5 | 2.1 | 2.5 |
| Neurasthenia and the like (part of 87b). | . 8 | . 4 | 1.1 |
| Other diseases of the nervous system (78-85, part of 87b) | 1.1 | 1.7 | 1.4 |
| Diseases of the heart and arteries, and nephritis (90-99, 102, 130-132) | 3.9 | 3.9 | 4.3 |
| Other genito-urinary diseases (123-138). | 2.4 | 2.6 | 2.8 |
| Diseases of the skin (151-153)......... | 2.3 | 2.3 | 2.7 |
| Epidemic and endemic diseases except influenza (1-10, 12-18, 33, 37, 38 , part of 39 and 44). | 2.7 | 3.9 | 3.3 |
| Ill-defined and unknown causes (200) --- | 1.9 | 2.0 | 2.0 |
| All other diseases (19-22, 24-32, 36, part.0f 39 and 44, 40-43, 45-55, 58-77, $88,89,100,101,103,154-150 \mathrm{a}, 157,162$. | 6.1 | 5.7 | 7.2 |
|  | 157, 662 | 145, 728 | 140,605 |
|  | 33 | 33 | 36 |

1 In 1934 and 1936 the same companies are included. The rates for the first quarter of the years 1930 to 1934 include 19 of these companies, which employed an average of 119,592 men during these months, or 80 perceint of the 149,605 men representing the sample popilation for the 5 years.
a Exclusive of disability from the venereal diseases, and a few numerically unimportant causes of disability.

## In the industrial population of the United States and Canada the

 influenza mortality rate was 66 percent higher in the January-March period of 1935 than in the same 3 months of 1934. The Metropolitan Life Insurance Co. reports: "The influenza death rate, despite its rise, is below the 5 -year average for the winter season, and the increase has not been accompanied this year by higher pneumonia mortality." As will be noted in table 1, the influenza morbidity rate was also below the 5 -year average. Although no increase occurred in pneumonia mortality among the industrial policyholders of the Metropolitan Life Insurance Co., a definite rise in the frequency[^1]of cases of this disease is shown from the records of industrial sickbenefit associations and company relief departments made available to the Public Health Service.

Nonrespiratory diseases as a whole occurred at a slightly lower rate than that of a year ago, and considerably below the 5 -year average for the winter months. No marked decrease occurred in the incidence of any of the disease categories comprising the broad group of nonrespiratory diseases, with the exception of the epidemic and endemic diseases exclusive of influenza, which decreased about 30 percent from the level of a year ago; but in general the rates were slightly lower than those of 1934, and in certain instances a sizable decrease in frequency is revealed when comparison is made with the 5 -year average, as for example, in disability due to diseases of the stomach, hernia, the rheumatic group, and diseases of the skin.

Nonrespiratory diseases showing unfavorable comparison with the rates for 1934 are neurasthenia, and neuralgia, neuritis, and sciatica.

As explained in earlier communications, the sickness rates presented are based on reports of cases causing disability_for more than 1 week and for which sick benefits have been paid from funds provided either by the employing company, by its employees, or jointly by both. Venereal diseases and a few numerically unimportant causes of disability are not reported. The employees of the cooperating companies live in almost all parts of the United States, but most of them are located in the North Central, North Atlantic, and New England States.

## A STUDY OF 450 FATAL CASES OF HEART DISEASE OCCURRING IN WASHINGTON (D. C.) HOSPITALS DUṘING 1932, WITH SPECIAL REFERENCE TO ETIOLOGY, RACE, AND SEX ${ }^{1}$

By O. F. Hedley, Passed Assistant Surgeon, United States Public Health Service
During the past 20 years a number of analyses of heart disease have been made from different viewpoints and by studying various types of clinical and post-mortem cases. Practically all of these reviews have stressed the importance of etiology. Many of them have noted the difference in the kind of heart disease found in males and females. With the exception of Allen (1), in Cincinnati, and certain writers in the South, not enough attention has been paid to the striking contrasts in the etiological types of heart disease and the ages of death among the white and colored races.

Source and distribution of material.--Through the kind cooperation of every hospital in the District of Columbia, it was possible to review the clinical and necropsy records of the fatal cases of heart disease
occurring during 1932. Altogether, 516 case records were studied. Of these, 66 were discarded for lack of sufficient evidence, consisting, for the most part, of coroners' cases and other instances in which death occurred en route or within the first 24 hours. The fact that these cases are omitted should be taken into consideration in interpreting the results; for had they been included it is quite likely that the percentages of deaths from arteriosclerotic and hypertensive forms of heart disease and from the manifestations of syphilitic aortitis would have been increased and the proportion of deaths from other causes thereby reduced.


Figure 1.-Comparative age distribution of deaths, by race and sex, among 450 fatal cases of heart disease occurring in Washington (D. C.) hospitals during 1932.

Among the 450 fatal cases surveyed, 258 were in white and 192 in colored persons. In the white group there were 192 males and 66 females; in the colored group, 104 males and 88 females. The larger size of the white male group cannot be interpreted solely as being due to more fatal heart disease among white males. There are a number of hospitals in the District of Columbia whose clientele consists largely of white males, such as the Walter Reed General Hospital, the Naval Hospital, the Veterans' Administration Hospital, the hospital for the Soldiers' Home, and, to a less extent, St. Elizabeths Hospital, the Federal neuropsychiatric institution.

Ages at death.-The average age at death among the entire series was 52.3 years. Among the white group it was 55.5 years- 57.1 years for the males and 52.5 for the females. The reason for the older
ages at death among the males than among the females is due to the inclusion of the fatal cases from the Soldiers' Home, and probably does not represent a true picture of the comparison between the ages at death for white males and females, since it is generally observed that males die at earlier periods than females. The average age at death among the colored hospitalized cardiac cases was 47.4 years- 46.9 years for males and 49.0 for females. The colored group died at an average of nearly 8 years younger than the white. The reason for this will be made apparent when consideration is given the various etiological factors.

Not only does the average age at death but also the distribution of fatal cases vary with race and to a lesser degree with sex, as shown in figure 1 and table 1. The maximum number of fatal cases among both sexes of the white race occurs in the 60-69 year age period, whilo the peak number of fatal cases in the colored race, both sexes, occurs during the 40-49 year age period.

Table 1.-Number and percentage of deaths in each age group and average age at death among 450 fatal cases of heart disease in Washington (D. C.) hospitals during 1932

| Age group (years) | White |  |  |  |  |  | Colored |  |  |  |  |  | Total |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Males |  | Females |  | Total |  | Males |  | Females |  | Total |  |  |  |
|  | No. | Percent | No. | Percent | No. | Percent | No. | Percent | No. | Percent | No. | Percent | No. | Percent |
| 0-9. | 7 | 3.6 | 7 | 10.6 | 14 | 5.4 | 4 | 3.8 | 3 | 3.4 | 7 | 3.6 | 21 | 4.6 |
| 10-19 | 5 | 2.6 | 2 | 3. 0 | 7 | 2.7 | 4 | 3.8 | 3 3 3 | 3.4 | 7 | 3.6 | 14 | 3. 17 |
| 20-29 | ${ }^{6}$ | 3.1 | 4 | 6. 1 | 10 | 3. 8 | + | 2.9 17.3 | 18889 | 23.4 | 36 | 18.7 | 16 53 | 11.7 |
| 30-39 | 13 | 6.7 13.0 | 5 | 6.1 7.6 | 17 30 | $\underline{11.5}$ | ${ }_{27}^{18}$ | 26.1 | 21 | 23.9 | 48 | 25.0 | 78 | 17.7 |
| 50-59 | 32 | 16.7 | 8 | 12.1 | 40 | 15.5 | 23 | 22.1 | 14 | 15.9 | 37 | 19.3 | 77 | 17.7 |
| 60-69 | 50 | 28.1 | 18 | 27.3 | 68 | 26.4 | 16 | 15.3 | 14 | 15.9 | 30 | 15.6 | 98 | 21.7 |
| 70-79. | 34 | 17.7 | 14 | 21.2 | 48 | 18.8 | 8 | 7.7 | 10 | 11.4 | 18 | 9.4 | 66 | 13.7 |
| 80 and over. | 20 | 10.4 | 4 | 6.1 | 24 | 9.3 | 1 | . 9 | 2 | 2.3 | 3 | 1.6 | 27 | 6.2 |
| Tctal. | 192 | 100 | 66 | 100 | 258 | 100 | 104 | 100 | 88 | 100 | 192 | 100 | 450 | 100 |
| Average age at death. |  | 57.1 |  | 52.5 |  | 55.5 |  | 46.9 |  | 49.0 |  | 47.3 |  | 52.3 |

Among white males 28.1 percent of the deaths from heart disease occurred at 70 years or older, as compared with 27.3 percent among the females. On the other hand, only 8.6 percent of the colored males and 13.7 percent of colored females dying of heart disease lived to be 70 years of age. Among the white males 16.0 percent of these deaths occurred before 40 years of age, as compared with 25.8 percent among the white females. It will be shown later that this difference is due to the higher incidence of rheumatic heart disease among white females. This difference in incidence is not reflected to any great extent in the official mortality statistics, since deaths from rheumatic heart disease occurring during or shortly after attacks
of rheumatic fever are not usually tabulated under heart disease. In the colored group, 27.8 percent of the total deaths from heart disease in males occurred before 40 years of age, as compared with 30.6 percent in females. A considerable difference is noted between the white and colored groups in the distribution of deaths prior to 40 years of age. As shown in table 1 , there is a much higher mortality among the colored groups during the $30-39$-year age period than among the whites. This is due to the influence of syphilis of the aorta and heart and to the greater and earlier havoc wrought by the degenerative diseases, particularly hypertensive heart disease, among the colored population.
Etiological factors involved.-All of the more common and some of the rarer etiological types of heart disease are included in this series of 450 fatal cases of heart disease. As shown in figure 2 and table 2,


Figure 2.-Percentage of total heart disease in each race and sex due to various etiological factors among 450 fatal cases of heart disease occurring in Washington (D. C.) hospitals during 1932. (Information obtained by review of elinical records.)
arteriosclerosis and hypertension, rheumatic infection, syphilis, bacterial endocarditis and pericarditis, congenital malformations, and hyperthyroidism were encountered as causes of heart disease in varying degrees of frequency among the races and sexes. Among the less frequent causes, pulmonary arteriosclerosis (cor pulmonale) and tuberculous pericarditis were noted. In other instances it appeared clear that the patient had succumbed to some form of heart disease to which no etiological agent could be attributed or in which it appeared that more than one etiological factor was involved. Not all of the known etiological types of heart disease were found. Trauma, scarlet fever, diphtheria, echinococcus disease, primary neoplasms of the heart, and even rarer conditions are known to exist, but were not met with in this group.

Table 2．－Etiological factors concerned in 450 fatal cases of heart disease，by color and sex of decedents，in Washington（D．C．）hospitals during 1932．（From information obtained by study of clinical records．）

| Etiological factors | White |  |  |  |  |  | Colored |  |  |  |  |  | Total |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Males |  | Females |  | Total |  | Males |  | Females |  | Total |  |  | 免 |
|  |  | 䔍 | $\begin{array}{\|l\|} \hline \stackrel{y}{0} \\ \text { 兑 } \\ \stackrel{y}{z} \end{array}$ | $\begin{aligned} & \text { 若 } \\ & \text { م } \end{aligned}$ | $\begin{aligned} & \hline \stackrel{5}{5} \\ & \text { 首 } \\ & \text { 2 } \end{aligned}$ | $\begin{aligned} & \text { प } \\ & \text { ¢ } \\ & \text { H } \\ & \text { H } \end{aligned}$ | $\begin{aligned} & \hline \stackrel{y}{\circ} \\ & \text { 品 } \\ & \underset{Z}{2} \end{aligned}$ |  |  | $\begin{aligned} & \text { 若 } \\ & \text { م } \end{aligned}$ |  | $\begin{aligned} & \text { 免 } \\ & \text { N00 } \\ & \text { م } \end{aligned}$ |  |  |
| Arteriosclerosis and hypertension．－－ | 130 | 67.7 | 38 | 57.6 | 168 | 65.1 | 48 | 47.1 | 59 | $67 . C$ | 108 | 56.3 | 276 | 61.4 |
| Rheumatic infection． | 30 | 15． 6 | 14 | 21.2 | 44 |  | ${ }^{8}$ | 8.6 | 7 | 8.0 | 16 | 8.3 | 60 | 13.3 |
| Syphilis－－．．－．．．．－． | 11 | 5． 8 | 2 | 3． 15 | 13 | 5.6 | 35 | 33.6 | 6 | 6． 7 | 41 | 21.3 | 54 | 12.0 |
| Bacterial infections．－．－．．．－ | 11 | 5． 8 | 3 | 4.5 | 14 | 5． 4 | 3 | 2.9 | 4 | 4.5 | 7 | 3．6 | 21 | 4．7 |
| Congenital malformations． | 3 | 1.5 | 2 | 4． 5 | ¢ | 2.3 .8 | 2 | 1.9 | 4 | 4.1 | 4 | 2.5 | 6 | 2.0 1.3 |
| Cor pulmonale | 1 | ． 5 | 0 |  | 1 | ． 4 | 1 | ． 9 |  |  | 1 | ． 5 | 2 | ． 4 |
|  | 0 |  |  |  | 0 |  | 4 | ． 8.8 | C |  | 1 | ． 5 | 21 | .$^{2}$ |
| Etiology undetermined．．．－－－－－．－－－． | 6 | 3.1 | 4 | 6.1 | 10 | 3.8 | 4 | 3.8 | 7 | 8.0 | 11 | 5.7 | 21 | 4.7 |
| Total | 192 | 100 | 66 | 100 | 258 | 100 | $1 \mathrm{C4}$ | 100 | 88 | 100 | 192 | 100 | 450 | 100 |

## ARTERIOSCLEROTIC AND HYPERTENSIVE HEART DISEASES

Incidence．－Arteriosclerotic－hypertensive forms of heart disease caused 276 deaths， 61.4 percent of the total number．Considerable variation is shown（table 2 and fig．2）in the incidence of degenera－ tive heart diseases among the different races and sexes．In the white group，these types of the disease caused 65.1 percent of the total cardiac deaths－ 67.7 percent in the males，and 57.6 percent in the females．In the colored group they caused 56.3 percent of the total cardiac deaths－ 47.1 percent in the males and 67.0 percent in the females．

It may be that the high incidence of cardiovascular syphilis among Negro males accounts for the lower incidence of deaths due to arterio－ sclerosis and hypertension．Deaths from cardiovascular syphilis occur approximately a decade earlier than those from degenerative diseases and probably result in many deaths in persons who would have otherwise later succumbed to arteriosclerosis and hypertension．

Ages at death．－The average ages at death in the arteriosclerotic－ hypertensive group differs considerably with the races，but little with the sexes in each race．The average ages at death among white males and females were 66.7 and 66.6 years，respectively，an average of 66.6 years for the entire white group．The average ages at death among colored males and females were 55.0 years and 54.2 years， respectively，an average of 54.6 years for the two sexes．The average age at death for the entire series of 276 cases in this diagnostic group was 61.9 years．The marked difference in the ages at death between the races is indicative of the greater havoc inflicted upon the colored race by arteriosclerotic－hypertensive forms of heart disease．From the point of view of morbidity from heart disease as seen among
hospital and out-clinic cases, this has been commented upon by Stone and Vanzant (2) and by Schwab and Schulze (3).

The maximum number of deaths from degenerative forms of heart disease, as shown in figure 3, occurs among whites in the 60-69 year age period, in which 35.1 percent occurred while the peak of this cause among the colored occurred in the 40-49 year age period, in which 25 percent were found. There are, however, nearly as many deaths among the colored group in the other decades between 40-69 years.


Figure 3.-Comparative age distribution of deaths, by race and sex, among 276 fatal cases of artario-sclerotio-hypartensive heart diseases occurring in Washington (D. C.) hospitals during 1932

In the white group, 40.4 percent of the deaths occurred at 70 years or older, as compared with only 16.7 percent among the colored. In both races 31.1 percent occurred at 70 years or older. Only 0.6 percent of the white deaths from arteriosclerotic-hypertensive forms of heart disease occurred before 40 years of age, while 12.9 percent of the colored deaths occurred in this relatively early period. Among colored females, 17.0 percent of the total number of deaths due to degenerative types of heart disease occurred prior to 40 years of age. This high percentage of deaths among colored females from degenerative forms of heart disease is due to the frequency and rapid course of essential hypertension in this group. This will be further explained in discussing the influence of arterial hypertension.

Factors involved.-This group is unlike the others in that it is a rather heterogeneous one, embracing two factors-arteriosclerosis, a structural change, and hypertension, a physiological alteration resulting in a greater load on the myocardium. In a broad sense, the pathological changes are characterized by atherosclerosis, beginning with intimal lipoidosis of the various arteries, and arteriolarsclerosis, an extensive thickening of the walls of the arterioles as the result of prolonged hypertension. Arteriosclerosis and arterial hypertension are so intermingled that in a study of this sort it is impossible to ascribe to each its true relative significance. This, incidently, is quite often the case in clinical medicine. One clinician will consider a given case as hypertensive heart disease with arteriosclerotic changes, while another equally capable one will consider it as arteriosclerotic heart disease with associated arterial hypertension. As the ultimate cause of neither of these conditions is understood, the difficulties in attempting to appraise the relative significance of each are at present insurmountable. An endeavor will be made, however, to indicate the serious aspects of arteriosclerotic changes and of arterial hypertension.

Arteriosclerosis of the coronary arterial tree.-There is an old and wellknown clinical adage to the effect that a man is as old as his arteries. This should be changed to "certain arteries." The damage done by arteriosclerosis cannot be measured on a quantitative basis. An individual may have severe generalized arteriosclerotic involvement, including such vessels as the aorta and the arteries to the extremities, and yet the condition may not be inimical to long life. On the other hand, a relatively slight amount of atheromatous changes in the coronary arterial system, certain of the vessels supplying the brain, the renal arteries, or the mesenteric vessels, may result in serious disturbances and often portend an early death.

Arteriosclerosis not infrequently has its incipiency in early life; and while it accompanies advanced years, it should not be looked upon solely as the inevitable result of old age. When attacking the aorta or coronary arteries it is characterized by atheromatous changes in the intima. It may be very selective in its localization, or quite diffuse. It is of metabolic rather than of inflammatory origin. Leary (4) has shown that lipoid cells are not infrequently deposited in the intima of the coronary arteries quite early in life. In the young human being this may result in progressive fibrosis, with narrowing of the lumen, which is due to fibrous changes in the intima. In elderly individuals large collections of lipoid cells accumulate in the intima, with minimal amount of fibrous tissue support. Both of these conditions predispose to nutritional difficulties, as a result of which thromboses may result-due in the young, to endothelial
necrosis, and among elderly individuals to the rupture of atheromatous masses into the lumen, with secondary thrombosis.

Either as a result of sudden occlusion following thrombosis or to a gradual narrowing of the intima in its more slowly progressive phases, coronary arteriosclerosis seriously interferes with the nutrition of the myocardium, the conduction system, or both. Arteriosclerosis of the coronary arteries, their branches and ramifications, impoverish the blood supply to these structures. As a result of this inanition, atrophy with replacement fibrosis occurs. This may result in myocardial insufficiency, or the more serious arrhythmias, such as auricular fibrillation or flutter, varying degrees of auriculo-ventricular dissociation, or bundle-branch block. There is no very close correlation between the amount of myocardial scarring and the extent of functional impairment. There appear to be "silent" areas in the heart as in other organs, regions capable of undergoing considerable pathological alterations without seriously disturbing the function of the organ. On the other hand, only slight changes in certain parts of the heart, notably around the conduction system, may result in serious damage.

In a study comprised of examinations of clinical records from a number of institutions it is extremely difficult to estimate the number of cases showing coronary arteriosclerosis or the extent of coronary involvement. Probably most of the older cases had a certain amount of coronary arteriosclerosis, and in the younger ones among those deaths not directly attributable to essential hypertension. The fact that the total number of instances of coronary arteriosclerosis is not stated numerically is not to be considered as in any way minimizing its significance as compared with other factors.

Coronary thrombosis.-One of the most significant aspects of coronary arteriosclerosis is that it results in a tendency for the vessel to undergo thrombosis. The mechanism of the thrombosis has been mentioned. While the degree of atheromatous changes may be slight, thrombosis of an entirely normal coronary artery is nearly inconceivable. This does not refer, however, to the infrequent occlusions of coronary vessels as the result of embolic phenomena due to particles of vegetations being set free in the general circulation in bacterial endocarditides or after the resumption of a regular sinus rhythm following auricular fibrillation; but these conditions are beside the point of this discussion.

There was clinical evidence of thrombosis of the coronary arteries in 34 instances, 30 of which were among white males and 4 among white females. The average age at death was 66.3 years. The youngest age at death was 44 years. In only 5 instances was the condition associated with other manifest evidence of cardiovascular degeneration, a factor to be considered later in greater detail. This
is indicative of the relative selectivity of arteriosclerotic processes. Coronary thrombosis, as in angina pectoris, is relatively infrequent among Negroes, the reason for which no satisfactory explanation has been furnished.

Anginal syndrome.-The mechanism of cardiac pain has intrigued clinicians and research workers during the past 150 years. While the original description of the condition and the term "angina pectoris" has been popularly ascribed to Heberden, who wrote about this disorder in 1768, Bishop (5) states that William Harvey described the condition about 100 years previously. Jenner, in 1768, attributed the condition to hardening of the coronary arteries and was able to prove his theory later in the case of John Hunter. From the beginning the cause of this condition has been the subject of much controversy. Albutt and Wenchebach attributed the syndrome to arteriosclerotic ehanges in the aorta, Mackenzie to myocardial ischemia, while the modern (also the original) view is that angina pectoris is the symptom complex due to insufficiency of the coronary circulation, most often the result of arteriosclerosis of the coronary arteries. There is probably an added neurogenic factor, a state of hyperirritability or the establishment of certain reflex arcs in some individuals which predisposes to this condition.

There is considerable evidence to justify the be $i$ f that anginal syndrome is usually the result of coronary insufficiency. In about 75 percent of cases dying of angina pectoris there is post-mortem evidence of coronary arteriosclerosis. The age distribution and predominance of the male sex correspond with other forms of coronary arteriosclerosis. The site and radiation of the pain simulates that produced by thrombosis of the coronary arteries. Electrocardiographic tracings obtained during attacks are not dissimilar to those following coronary occlusion. Nevertheless, conditions unassociated with disease of the coronary arteries produce cardiac pain. Furthermore, bona fide cases of angina pectoris occur in which there is insufficient gross evidence to incriminate the coronary arteries.

Due to the fact that pain around the hes $t$ and throughout the chest is not infrequent during the terminal stages of almost any type of heart disease, and may be due to such conditions as terminal pneumonias or congestive heart failure, it was not considered feasible to attempt to evaluate the significance of cardiac pain in this series. Furthermore, deaths from angina pectoris are not so frequent among hospital patients, most of whom are admitted for congestive heart failure or coronary thrombosis.

Other forms of arteriosclerotic heart disease.-Although a large proportion of the diagnoses made of valvular conditions among elderly individuals is commonly due to the misinterpretation of cardiac murmurs associated with hypertrophy and dilatation, or to minor
thickening of the cusps and leaflets of the valves produced by relatively insignificant sclerotic changes of insufficient importance to be considered as real clinical entities, nevertheless arteriosclerotic valvular diseases do occur. Such lesions take the form of calcareous changes in the leaflets and cusps, and in the valve rings, especially of the aortic valve. Aortic stenosis is the predominant clinical and post-mortem manifestation, although regurgitations may accompany. These lesions have been described by Mönckeberg (6); Margolis, Ziellesen, and Barnes (7); and by Christian (8).

There were 3 cases of aortic valvular insufficiency and 1 of aortic stenosis apparently coming within this category. There were numerous other cases in which mitral murmurs were described, but these were not taken as indicative of real valvular disease. In three other instances there were fairly clearly defined histories of rheumatic heart disease with clinical evidence of mitral stenosis. These were found among elderly individuals, and the rheumatic element appeared to be residual. These patients lived to a ripe old age despite their rheumatic heart disease. It is doubtful that such deaths should be considered as due to rheumatic heart disease any more than an individual with healed tuberculous lesions of the lung should be considered as succumbing to pulmonary tuberculosis.

Other forms of arteriosclerotic heart disease, such as dissecting aortic aneurysms, were not encountered.

Relation to other forms of vascular degeneration.-Arteriosclerotic heart disease may occur as an isolated phenomenon in a vascular tree practically free of other atherosclerotic changes, or it may be but the clinical manifestation of wide-spread, frequently generalized arteriosclerosis. Even in the instances in which heart disease is the chief clinical point of interest, wide-spread arteriosclerotic changes may coexist without playing an important part in the clinical picture. From a broad biological viewpoint, the heart in such cases has either undergone somewhat more advanced involution, or is clinically more vociferous about the pathological changes it has suffered.

Among younger individuals with arteriosclerosis of the coronary arteries it is not uncommon to find that the remainder of the circulatory system is practically unimpaired, since arteriosclerosis is sometimes highly selective in its localization. As the cause of arteriosclerosis is still an enigma, there is no explanation for this. However, from the personal, clinical, and public health aspects there is much difference in the case of the middle-aged business man who falls dead from coronary thrombosis and that of the person in the seventies who develops a cerebral apoplexy, the immediate effects of which are survived, but who later develops congestive heart failure and dies of bronchopneumonia.

Of the 276 deaths due to arteriosclerotic-hypertensive heart diseases, 134 were regarded as primarily cardiac and 142 as instances of heart failure as part of manifest generalized cardiovascular renal degeneration. In 97 of these there was clinical evidence of other manifestations of vascular deterioration. Renal insufficiency, as exhibited by clinical evidence of nephritis, elevated blood chemistry determinations, and other laboratory tests was considered present in 58 instances. Albuminuria per se was not considered as sufficient evidence to warrant regarding the patient as having renal disease. This may occur in congestive heart failure from any cause. Furthermore, certain cases of renal insufficiency were omitted, as they were thought to be merely terminal events. Hemiplegias due to cerebral hemorrhage or thrombosis occurred in 24 instances, arteriosclerotic psychoses in 23 cases, diabetes mellitus 3 times, peripheral vascular occlusions 3, arteriosclerotic gangrene in 3, and hypertrophic arthritis once. In addition to these cases attended by manifest evidences of generalized arteriosclerosis, in 46 others there was evidence of generalized arteriosclerosis on physical examination, notations of senility, or the supervention of death after prolonged periods of general debility or weakness. These latter cases were all in elderly individuals, the average age at death of whom was 73 years. Death in such cases was the result of generalized arteriosclerosis, with cardiac insufficiency acting as the clinical straw breaking the back of the arteriosclerotic changes. The average age at death among the 142 cases with associated cardiovascular renal changes was 68.8 years, as compared with 54.8 years among those dying of heart disease unattended by other clinical manifestations of vascular deterioration.

Relation of arterial hypertension.-One of the most interesting aspects of the degenerative group of cardiac diseases is its relationship to arterial hypertension. This condition, the cause of which is unknown, frequently begins in early middle life. From the anatomical perspective it is characterized by spasm followed by hypertrophy, fibrosis, and eventually sclerosis of the arterioles (9) (10). Owing to frequent and rather easily discernible involvements of the arterioles of the kidneys, and to the fact that the more rapidly progressive forms of essential hypertension frequently terminate in the form of renal insufficiency with uremia, an attempt has been made to associate essential hypertension with renal arteriosclerosis, as if that condition causes the elevated blood pressure or is the chief manifestation. Arteriolarsclerosis, however, occurs generally, having been found in the arterioles of the brain, retina, spleen, pancreas, skeletal muscles, and other structures (11). These wide-spread changes in the terminal portions of the arterial system are probably resultant upon changes in hemodynamies rather than the cause of the abnormally high arterial blood pressure. These changes are not, however, entirely
specific for arterial hypertension, as they are found in individuals not exhibiting this physiological abnormality. It seems, however, that the presence of arterial hypertension accelerates changes in the arterioles.

As a result of the increased arterial load, the heart undergoes enlargement, manifested by hypertrophy of the fibers of the myocardium. This is prejudicial to the osmosis of nutrient fluids to the heart muscle fibers, as a result of which myocardial fatigue and subsequent failure ensues.

It is frequently impossible to distinguish the arteriosclerotic factors from the hypertensive, so closely are they interwoven. Quite often one is unable to determine to what extent each is responsible for the clinical condition. As with the riddle of the hen and the egg, it is not possible to state which preceded the other. To what extent hypertension accentuates arteriosclerotic changes in the heart cannot be answered categorically, although there is sufficient evidence to believe that arterial hypertension predisposes to the development of coronary arteriosclerosis. Certainly the association of the two does not help the prognosis in a given case.

Among these 276 cases in which death was due to degenerative forms of heart disease, blood pressure determinations were obtained in 225 , of which 163 , or 72.4 percent, showed evidence of arterial hypertension. The criteria used in determining hypertension in this series are minimum systolic arterial blood pressures of 150 millimeters of mercury or diastolic arterial blood pressures of 100 millimeters or higher, regardless of the systolic blood pressure. Since the records of a large number of these cases were begun only a short time before death, it is probable that the percentage here reported showing evidence of hypertension is somewhat lower than that actually occurring. For instance, in most cases of coronary thrombosis there are varying degrees of shock, which results in a diminution of the arterial blood pressure. Patients admitted to hospitals in such a state show an unnaturally low blood pressure, and may well have had an elevation of arterial blood pressure before suffering from this cardiac catastrophe. Furthermore, in essential hypertension there may be at times a return to normal limits, especially after congestive failure sets in. In other cases, even before there is evidence of congestive heart failure, it appears that the hypertension, somewhat analogous to a not infrequent occurrence in hyperthyroidism, burns itself out, leaving a permanently damaged vascular system, but no evidence of hypertension at the time of examination. The fact that in nearly 75 percent of deaths due to arteriosclerotic-hypertensive forms of heart disease the patients maintain an elevated arterial blood pressure until approaching the fatal termination suggests that the mechanism of hypertension is not merely due to an increased resistance in the
arterioles, but that there is an intense dynamic urge, probably associated with the necessity for maintaining an adequate circulation in the vital centers.

In table 3 is shown the age distribution of the 225 fatal cases of arteriosclerotic-hypertensive forms of heart disease upon which bloodpressure determinations were made. Of the 51 deaths occurring before 50 years of age, 48 , or approximately 94 percent, of the patients had elevated arterial blood pressures during their fatal illnesses. In other groups somewhat smaller percentages showed evidence of hypertension. It is felt that, in most deaths from degenerative forms of heart disease under 50 years of age, and in a predominantly high proportion under 60 years of age, hypertension is a factor of paramount importance. There are certain deaths due to a rather selective type of coronary arteriosclerosis, usually accompanied with a thrombosis, in which it is not possible to elicit histories or find evidence of arterial hypertension, but the aggregate of these is relatively small, especially under 50 years of age.

Table 3.-Arterial blood pressure in 225 cases in which death was subsequently due to arteriosclerotic-hypertensive forms of heart disease in Washington (D. C.) hospitals during 1932. Minimum systolic blood pressures of 150 millimeters or diastolic pressures of 100 millimeters were considered as manifest evidence of arterial hypertension. Distribution based on ages at death

| Age periods | Total blood pressure determinations | Number elevated | Number normal | Percent elevated |
| :---: | :---: | :---: | :---: | :---: |
| 80-39 years*- | 15 | 14 | 1 | 93.3 |
| 40-49 years. | 36 | 34 | 2 | 94.4 |
| 60-69 years. | 49 | 34 | 15 | 60.1 |
| 60-69 years. | 61 | 36 | 25 | 59.0 |
| 70-79 years. | 41 | 29 | 12 | 70.8 |
| 80 and over | 23 | 16 | 7 | 60.6 |
| Total. | 225 | 163 | 62 | 72.4 |

*Including 1 in 20-20-year age group.
The problem of essential hypertension is the gravest in middle adult life, not even excepting that of cancer. To the individual at the age of 40 the greatest single factor in estimating the probability of living to a ripe old age is whether the arterial blood pressure has begun to show signs of elevation. Fahr (12) estimates that 140,000 persons in the United States die annually of "hyperpiesis", or essential hypertension. He estimates that approximately 50 percent of this number die of heart failure, 40 percent of apoplexy, and 10 percent of uremia. On this basis, he believes that 70,000 die annually of hypertensive heart disease.

In table 4 is shown the average systolic and diastolic arterial blood pressures and the percentages of cases with diastolic blood pressures of 100 millimeters or higher among 163 cases showing evidence of in-
creased blood pressures. These records in a number of cases were probably scmewhat lower than the average during the patients' illnesses, and probably very infrequently represent the maximum arterial blood pressures. Paradoxical as it may seem, however, it often happens that there is a rise in the arterial blood pressure during a bout of congestive heart failure, with subsequent reduction upon improvement in the action of the heart. It may have been that some of the determinations were made during these crises. The diastolic pressures are probably more accurate, as they show less tendency to fluctuate and tend to maintain a more constant level after congestive heart failure sets in. This table shows that both the systolic and diastolic pressures are higher in those cases in which death occurs before 50 years of age. Furthermore, while 86.0 percent of those dying before 50 had diastolic elevations of 100 millimeters or higher, among those surviving 70 years of age only 51.1 percent showed this diastolic rise.

Table 4.-Avbrage systolic and diastolic arterial blood pressures, with percentage of elevated diastolic pressures, among 163 patients manifesting evidences of arterial hypertension and subsequently dying of arteriosclerotic-hypertensive forms of heart disease in Washington (D. C.) hospitals during 1932. Tabulated according to ages at death

| Age period | Average systolic blood pressure | Average diastolic blood pressure | Percentage with diastolic elevation of 100 millimeters of mercury or higher |
| :---: | :---: | :---: | :---: |
| Under 50 years. | 205 | 130 | 86.0 |
| 60-59 years..... | 193 | 118 | 78.4 |
| 60-69 years | 172 | 107 | 72.2 |
| 70 years and over | 170 | 100 | 51.5 |

In table 5 is shown the percentage in each race and sex with evidence of arterial hypertension. Due to the conditions under which the blood-pressure determinations were obtained, the figures cited in this table cannot be taken as absolute. Nevertheless, they serve as an index to the greater frequency of hypertension in the colored race than in the white. It is noted that, among those patients upon whom blood-pressure determinations were obtained, 89.4 percent of the colored group showed evidence of hypertension as compared with only 60.3 percent in the white group. This tends to discredit the presumption that hypertension predisposes to changes in the coronary arteries, since both clinical and pathological evidence of coronary arteriosclerosis are relatively infrequent among Negroes, while hypertension is far more common in the colored than in the white population. It may be, however, that among Negroes a fatal termination occurs before a great deal of coronary arteriosclerosis has resulted. To support this, it is noted that, among the degenerative cases with
hypertension, death occurred at the average age of 52.8 years- 54 years and 51.6 years among males and females, respectively. Among the whites the average age at death of cases manifesting arterial hypertension was 65.1 years- 66.2 and 61.4 years among males and females, respectively. The average for all cases showing hypertension was 59.5 years.

Table 5.-Number and percentage of fatal cases of arteriosclerotic-hypertensive types of heart disease in Washington (D. C.) hospitals during 1932 showing evidence of arterial hypertension. Based on color and sex of decedents

|  | White |  |  | Colored |  |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Males | Females | Total | Males | Females | Total |  |
| Total cases. | 130 | 38 | 168 | 49 | 59 | 108 | 276 |
| Number with blood pressure determinations.- | 102 | 29 | 131 | 43 | 51 | 94 | 225 |
| Number with elevated blood pressures...---- | 601 | 18 | -79 | 37 | 47 | 84 | 163 |
| Percent showing elevated blood pressures. | 60.0 | 63.1 | 60.3 | 86.0 | 92.9 | 89.4 | 72.4 |

Blood Wassermann and Kahn reactions.-In this series of 276 deaths from arteriosclerotic-hypertensive forms of heart disease, 146 Wassermann or Kahn reactions were recorded. Of these (table 6) 22, or 15.1 percent, were positive. This is not in excess of the percentage expected among each race and sex group in the general population. However, among Negroes it was found that the average age at death among those with positive serological reactions was 46.2 years, as compared with 54.7 years among those with negative serology. This suggests that, while syphilis has little, if any, effect in initiating arteriosclerotic-hypertensive forms of heart disease, the concurrence of these conditions results in a higher fatality.

Table 6.-Blood Wassermann or Kahn reactions, according to color and sex, obtained in routine serological examinations among 146. of 276 cases of arterioscle-rotic-hypertensive forms of heart disease subsequently dying in Washington (D. C.) hospitals during 1938

|  | White |  |  | Colored |  |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male | Female | Total | Male | Female | Total |  |
| Total cases......-.........-- | 130 | 38 | 168 | 49 | 69 | 108 | 276 |
| Cases upon which serological reactions were obtained. | 68 | 16 | 84 | 28 | 34 | 62 | 146 |
|  | 4 | 1 | 5 | 10 | 7 | 17 | 22 |
|  | 6.0 | 6.3 | 6.0 | 35.8 | 20.6 | 27.4 | 15.1 |

Necropsy evidence.-There were 81 necropsies performed on this group, the results of which are presented in table 7. As compared with other etiological groups there is a lack of characteristic findings here. This is to be expected, as among these cases the myocardial
and vascular changes are frequently but part of a general involution and not a specific entity as is syphilitic mesaortitis or rheumatic carditis.

Tabli 7.-Necropsy findings in 81 cases (by color and sex) of arteriasclerotiohypertensive forms of heart disease occurring in Washington (D. C.) hospitals during 1932

|  | White |  |  | Colored |  |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male | Female | Total | Male | Female | Total |  |
| Total cases. | 130 | 38 | 168 | 49 | 59 | 108 | 276 |
| Total necropsies. | 41 | 9 | 50 | 11 | 20 | 31 | 81 |
| Coronary arterioscleresis.- | 25 | 6 | 31 | 4 | 2 | 6 | 37 |
| Coronary thrombosis. | ${ }_{33}^{12}$ | $\frac{1}{8}$ | ${ }_{38}$ | 0 5 | 0 | ${ }^{0}$ | 13 |
| Cardiac enlargement (hypertrophy and dilatation) | 10 | 6 | 16 | 8 | 14 | 2 | 8 |
| Hydropericardium... | 4 | 2 | 6 | 8 | 2 | 7 | 13 |
| Hemopericardium. | 3 | 0 | 3 | 1 | 0 | 1 |  |
| Adhesive pericardium. | 4 | $\theta$ | 4 | 0 | 4 | 4 |  |
| Yalvalar sclarosis...- | 13 | 2 | 15 | 4 | 8 | 12 | 27 |
| Residual rheumatic lesions. | 0 | 0 | 0 | 1 | 0 | 1 |  |
| Arteriosclerotic changes in aorta.. | 31 | 9 | 40 | 8 | 4 | 12 | 58 |
| Syphilitic acrtitis--..-...-. | 0 | 0 | 0 | 0 | 5 | 5 | 5 |
| Evidence of congestive heart failure. Renal | ${ }_{21}^{18}$ | 8 | ${ }_{26}^{26}$ | 9 7 | 16 15 | 22 | 51 |

In this part of the study, owing to lack of uniformity among pathologists in the many hospitals throughout the city it was necessary to select general terms. For example, one pathologist will describe a given case as coronary thrombosis, while another will term a similar lesion coronary occlusion. Chronic myocarditis, myocardial fibrosis, and fibrous myocarditis are used interchangeably. The findings in table 7 are not offered for the purpose of showing the exact proportion in which the various lesions occur, but to show the general trend in necropsy diagnoses in the hospitals of a large city. It is quite probable that had these post-mortem examinations been performed by one individual, the results would have been somewhat different. On the other hand, the findings here described were obtained by a "jury of pathologists" and the element of personal opinion is thereby lessened.

Arteriosclerosis of the coronary arteries was diagnosed as such in 37 instances, of which 31 were white subjects. Coronary thrombosis was found in 13 autopsies, all of which were on white subjects. The relative infrequency of angina pectoris in the colored race is a widely attested clinical observation. Libman (18) believes that this is due to the fact that the colored race is relatively hyposensitive to pain. It appears, nevertheless, that coronary arteriosclerosis is a less frequent disease among American Negroes than among the white population. Thus the lack of symptomatic evidence of coronary disease depends more on lack of organic changes than on any nervous mechanism, unless it can be shown that the development of coronary arte-
riosclerosis is dependent upon nervous influences. This, of course, has not been proved.

If the cases in which coronary arteriosclerosis, coronary thrombosis, and myocardial fibrosis are grouped together, 55 of the 81 necropsies showed evidence of changes which may be interpreted as due to arteriosclerosis of the coronary arteries or their branches.

Valvular sclerosis occurred in 27 instances, most of which were in the form of minor degrees of thickening of the valve cusps. Stenosis of the aortic valve occurred in one instance. There was 1 case of mitral insufficiency, due to a rupture of a papillary muscle following a coronary occlusion with myocardial infarction. A residual rheumatic lesion was noted in 1 instance. Luetic aortitis was noted in 5 cases. Arteriosclerosis of the aorta was recorded in 52 instances. Cardiac enlargement was reported in 38 necropsies. This relative infrequency is frankly open to question, as is also the frequency with which no mention was made of evidence of congestive heart failure. Had all of the hearts been weighed, it is likely that a greater number would have been recorded as enlarged.

## RHEUMATIC HEART DISEASE

Rheumatic heart disease is, perhaps, the most important form of heart disease from economic and social points of view. It results in cardiac crippling and death among those so afflicted during the periods of education, greatest earning capacity, and when most needed in the home. This type of heart disease is due to rheumatic infection, the ultimate cause of which is still unknown. Such evidence as exists at present indicts, but has not yet convicted, hemolytic streptococci as the responsible micro-organisms. The writer has reviewed the bacterial investigations in another article (14).

Rheumatic infection is a chronic systemic disease characterized by tendencies to recrudescences and recurrences. Its pathological manifestations occur in the forms of proliferative and exudative reactions, involving in particular endothelial and subendothelial tissues. It rarely directly involves parenchymal structures. It has a predilection for the tissues of the heart, serous membranes, periarthritic structures, skeletal muscle, and the brain. Its manifestations are frequently from both clinical and pathological standpoints quite evanescent, tending to move from structure to structure. It is frequently associated with tonsillitis and pharyngitis, attacks of which often precede the onset of the disease or initiate recurrences and relapses. There is a strong tendency toward chronicity, the disease existing in a smouldering state, with periods of activity resulting from factors not yet fully understood.

The heart is almost always involved to a greater or less degree during active infection. Permanent damage to this organ frequently, but
not invariably, occurs. Cardiac involvement occurs in the forms of endocarditis, valvulitis, myocarditis, and pericarditis, singly or in combination, and in all degrees of severity. Rheumatic involvement of the coronary arteries, aorta, and pulmonary arteries is not uncommon. Death may be due either to active rheumatic carditis or to mechanical difficulties, the result of previous infection with subsequent fibrous changes. Furthermore, owing to previous infection, the resistance of the valves may be so impaired that organisms gain a foothold, causing death from infective or bacterial endocarditis.

Incidence.-Rheumatic heart disease was the etiological type involved in 60 deaths, or 13.3 percent of the 450 deaths due to heart disease included in this study. It caused 44, or 17.1 percent, of deaths from heart disease among white patients, as compared with 16, or 8.3 percent, of the cardiac deaths among the hospitalized colored cases. This is in accord with the general observation that the disease is more common in the white race. It was the cause of 30 , or 15.6 percent, of the cardiac deaths among white males, and 14, or 21.2 percent, among white females. While the absolute number of deaths was higher among white males, the incidence is higher among white females. It is generally noted that rheumatic fever and rheumatic heart disease are more common among white females. Among the colored cases there was practically no difference in either the actual number of deaths or the incidence of deaths due to rheumatic heart disease in the sexes, this condition accounting for 9 , or 8.6 percent, of the total deaths from heart disease among the males and for 7, or 8.0 percent, among the females.

Ages at death.-The average age at death was 29 years. For the white group it was 30.5 years- 30.9 years for males and 28 for females. For the colored, both sexes, the average age at death was 24.7 years26.6 for males and 22.9 for females. Coombs (15), in England, in a series of 98 deaths due to rheumatic heart disease examined post-mortem, found that the average age at death was 28.3 years, a very close approximation of this series.
Rheumatic carditis as a cause of death.-Findlay (16) has indicated that there are two factors causing death in this disease, infection and myocardial fatigue. Many die of rheumatic carditis, rarely during the first attack of rheumatic fever, but usually as the result of a long continued progressive cardiac infection characterized by periods of exacerbation with intervening periods of remission. This has been described by Coburn (17) as the "rheumatic state," and is essentially a generalized chronic infection tending to smoulder, but which flares up on provocation, with or without joint involvement. These patients suffer from a certain amount of cardiac insufficiency, becoming more marked with increased activity of the infection. Frank congestive heart failure is a late manifestation, occurring either as
the result of severe rheumatic carditis or as the summation of many low grade inflammatory processes. There were 27 deaths in which infection was regarded as the factor of paramount significance. The average age at death among these cases was 13.8 years.

Myocardial fatigue.-The other factor causing death in rheumatic heart disease is myocardial fatigue. This occurs chiefly as the result of the superimposition of arteriosclerotic changes on hearts whose action has been impaired by sclerotic and calcific changes in the valves, producing severe deformities, of myocardial fibrosis following rheumatic myocarditis, as the direct result of rheumatic involvement of the coronary arteries, and in many cases by various forms of pericardial adhesions. There is good reason to believe that rheumatic heart disease accelerates arteriosclerotic changes in the heart. The actual failure may be immediately precipitated by some form of undue fatigue or exertion, or by some intercurrent infection. Furthermore, in a number of cases there are varying degrees of rheumatic activity (18). This is particularly true among the subjects examined at autopsy who died of the disease in early adult life and even up to about 40 years of age. However, from a practical clinical point of view, infection in these well-developed valvular lesions is probably secondary in importance to the actual mechanical difficulties.

In this series the cause of death appeared to be due to mechanical difficultics associated with well-formed vaivular and pericardial lesions in 33 instances. The average age at death among these cases was 41.1 years.

Histories of rheumatic infection.-In this series of 60 fatal cases of rheumatic heart disease, histories of rheumatic fever were recorded in 31 instances, chorea once, scarlet fever once, and in 8 instances there was evidence of active rheumatic pancarditis without joint involvement. Evidence of rheumatic infection was therefore found in 41 cases. Of the remaining 19 cases, history of rheumatic infection was denied in 5, and in 14 it was undetermined. Scarlet fever is here mentioned as an etiological factor, as in rare occasions after scarlet fever a rheumatic type of heart disease develops. In most of such cases there is good reason to believe that a rheumatic infection occurred conjointly with the scarlatina. In a few instances scarlet fever per se apparently produces valvular heart disease. There is no clear-cut demarcation between these conditions under such circumstances, and it is quite probable that both are produced by closely related organisms, hemolytic streptococci.

Clinical findings.-Clinically, pericarditis was described alone in 3 cases, mitral valvular disease, insufficiency or stenosis, or both, in 29 cases, combined aortic and mitral involvement in 11, aortic insufficiency without mitral lesions in 3, and combinations of pericarditis or adherent pericardium with valve lesions in 14.

Necropsy findings.-Necropsies were obtained in 27 instances. Active rheumatic pancarditis was found in 14 cases, mitral valvular disease in 6, combined aortic and mitral lesions in 6, and aortic valvular disease without involvement of other valves in 1.

Wassermann reactions.-Positive blood Wassermann or Kahn reactions were reported in 3 of 20 cases upon which such examinations were performed.

Associated hypertension.-Arterial hypertension was found in 6 of the 33 cases upon which blood pressure determinations were made. Owing to the disease being so prevalent among juveniles, blood pressure determinations are frequently omitted.

Associated conditions.-Associated conditions included renal insufficiency in 8 cases, pulmonary tuberculosis in 1 case, a psychosis in 1, cerebral embolism in 4 cases, and bacteremia in 3. These instances of streptococcal bacteremia were terminal events in cases of active rheumatic carditis, and not subacute bacterial endocarditis.

## SYPHILIS OF THE AORTA AND HEART (SYPHILITIC HEART DISEASE)

Syphilitic heart disease, or, more strictly speaking, cardiothoracic syphilis, consists of syphilitic aortitis with its extensions and complications. During the early invasion of the body by the spirochaetes, an inflammatory reaction begins around the vasa vasorum, the nutrient blood vessels in the walls of the aorta. This perivascular infiltration occurs in the middle coat of the aorta, and may consist of a simple inflammation, clinically undetectable, and be discovered only by microscopic examination after necropsy. Although the middle coat is chiefly affected, a certain amount of scarring in the adventitia is not uncommon. Also periaortitis, involvement of the adventitia without mesaortitis, sometimes occurs as an isolated phenomenon (19).

The aortitis may be so extensive that the aortic wall loses its elasticity and becomes weakened. This results in dilatation and may even progress to the aneurysmal stage. The aortitis may extend downward, separating the cusps of the aortic valve at their commissures, producing shortening and curling of the cusps and dilatation of the ring of the aortic valve, resulting in an insufficiency of the aortic valve, the most common lesion. The aortitis may surround the orifices of the coronary arteries located in the sinuses of valsalva, producing varying degrees of stenosis or atresia of the mouths of the coronary arteries, as a result of which the nutrition of the cardiac musculature becomes seriously impaired. This involvement of the coronary ostii is a frequent cause of sudden death, due either to the progress of the disease or to the injudicious use of arsenicals, as a result of which edema is produced around the coronary orifices, resulting in sudden occlusion. The luetic involvement does
not extend more than 1 or 2 cm into the coronary arteries and does not appear to be associated with sudden occlusion due to thrombosis of the coronary arteries, from which it should be distinguished. The latter condition occurs as the result of arteriosclerosis and is not observed more frequently among those afflicted with syphilis than among nonluetics.

It does not appear that there is a direct spirochaetal involvement of the myocardium. Areas of focal myocardial fibrosis are found, but these appear to be the result of impairment of the nutrition of the myocardium rather than to an active inflammation.

The earliest positive clinical signs of syphilitic involvement of the heart and aorta can be made only during the late stages of syphilis. While there are exceptions, the general course of the disease is downward and attempts at treatment are very discouraging. Death usually supervenes about 3 or 4 years after diagnosis and in less than 2 years after congestive failure sets in. Death usually results from myocardial failure, from sudden occlusion of a coronary orifice, or from rupture of an aneurysm or erosion by an aneurysm of some vital structure. In this disease, as in most other forms of heart disease occurring in adult life, arteriosclerotic changes play an important role, and appear to be accelerated as a result of luetic involvement.

Incidence.-Syphilis of the aorta and heart was responsible for 54, or 12.0 percent, of the total deaths here reported on. It was the etiological factor in 5.0 percent of the deaths from heart disease in the white group, resulting in 11, or 5.8 percent, of the deaths among the white males, and in only 2 , or 3.8 percent, among white females. Among the colored males, this condition accounted for 35, or 33.6 percent, of deaths, and among the colored females for 6, or 6.7 percent.

The marked difference in the incidence of syphilitic heart disease in the two races is in accord with other studies conducted in the South. Laws (20) in an analysis of 645 clinical cases of organic heart disease among patients admitted to the wards and out-patient department of Vanderbilt University Medical school in Nashville, Tenn., found that syphilis was the etiological factor in 15.4 percent of heart disease among Negroes as contrasted with only 2.2 percent among white patients.

The marked difference in the incidence of syphilis of the aorta and heart between colored males and females is noteworthy. Gager and Dunn (21) found a similar, but not so marked, difference in alarge series of colored dispensary patients at the George Washington University Hospital in Washington, D. C. These observers noted that syphilitic heart disease was the etiological type in 21.0 percent of heart disease among colored males, but in only 10.5 percent in colored
females. Since the incidence of syphilitic infection, as determined by blood Wassermann surveys conducted by the Public Health Sarvice in cooperation with the Julius Rosenwald Fund (22) is approximately the same in both sexes among Negroes, it is quite probable that the hard manual labor performed by colored males predisposes them to the development of clinical manifestations of luetic aortitis.

Ages at death.-The average age at death was 51.5 years among white cases and 42.7 years among the colored. For the entire series it was 46.8 years. The disease manifests itself earlier and runs a more progressive course in the colored race. Whether this is due to any inherent susceptibility of the cardiovascular system in this race to spirochaetal infection, to lack of early and adequate treatment, to greater and more fatiguing physical exertion, or to earlier ages of initial lesions has never been determined.

Ages of initial lesions.-Initial lesions were admitted in 14 cases. The average age of 12 of these was 24.7 years at the time of the initial lesion, and the average age at death was 42.5 years, an average of 17.8 years intervening between the initial lesion and the fatal termination.

Clinical diagnoses.-The clinical diagnoses consisted in simple aortitis in 7 instances, aneurysms of the thoracic aorta without valvular involvement in 2 , aneurysms of the thoracic aorta with aortic valvular insufficiency in 7, and aortic valvular insufficiency without aneurysms in 38 . There were, therefore, 45 instances of aortic insufficiency and 9 of aneurysms of the thoracic aorta, some in combination with each other, in a total of 54 cases ending fatally. Rupture of aneurysms was the immediate cause of 2 deaths.

Necropsy evidence.-Twenty-nine post-mortem examinations were performed. There were 4 cases of simple aortitis (fusiform dilatation due to luetic mesaortitis), one case of aneurysm without aortic valvular insufficiency, 7 cases of aneurysm with aortic valvular insufficiency, and 17 of aortic valvular insufficiency without aneurysms. Aortic valvular insufficiency, therefore, occurred in a total of 24 cases and aneurysms of the thoracic aorta in 8 cases examined post mortem. There were 6 instances of luetic involvement around the orifices of the coronary arteries.

Blood Wassermann and Kahn reactions.-Blood Wassermann or Kahn reactions were recorded in 42 instances, 40 , or 95.2 percent, of which were positive.

Arterial hypertension.-Blood-pressure determinations were recorded in 46 instances, 24, or 52.2 percent, of which were elevated. The average age at death among those with elevated arterial blood pressures was 48.0 years as compared with 44.2 years among those with normal blood pressures. One would expect the converse, expecially after noting that cases of arteriosclerotic-hypertensive forms of
heart disease with syphilis die earlier than those not so afflicted. It may be that the development of a certain degree of hypertension assists in maintaining the coronary circulation. A higher systolic pressure may serve as a greater head of flow to counteract the effects of a lowered diastolic pressure in cases of aortic insufficiency.

Associated conditions.-Among the associated conditions, uterine fibroids, paresis, chronic arthritis, peptic ulcer, cerebral apoplexy, and pulmonary tuberculosis were each encountered once. Renal insufficiency (other than in the terminal stages of congestive heart failure) was noted twice.

Syphilis of the aorta and heart, unlike many degenerative cardiac conditions, is a clearly defined clinical and pathological entity. Unlike many cases of arteriosclerotic-hypertensive forms of heart disease, it is usually unassociated with other clinical evidence of vascular deterioration.

## BACTERIAL ENDOCARDITIS AND PERICARDITIS

Bacterial heart disease is characterized by the implantation, usually on the valves, of organisms producing soft vegetations. Less frequently it is due to pyogenic infections of the pericardium. Both conditions are almost invariably fatal. Death supervenes after clinical courses of varying, frequently lengthy, durations, often lasting over a year. Death is directly due to wasting infection, embolic phenomena due to the escape of particles of vegetations into the arterial circulation, nephritis of toxic or embolic origin, and infrequently to congestive heart failure.

Bacterial endocarditides are most often superimposed on preexisting lesions, either rheumatic or congenital. They are only very rarely engrafted on syphilitic cardiovascular affections. Bacterial pericarditides are usually due to trauma, or follow lobar pneumonia, not infrequently occurring with empyema of the lungs. The instance included in this series was a rather atypical one, being due to a streptococcus viridans infection associated with a rheumatic adherent pericardium. This case has been reported in detail by Yater and the writer (23).

Incidence. -In this series of 450 deaths from organic heart disease, 21 , or 4.7 percent, were attributable to bacterial carditides. These types of lesions resulted in 11, or 5.8 percent, of deaths from heart disease among white males, and 3 , or 4.5 percent, of cardiac deaths among white females. Altogether, it resulted in 14 deaths, or 5.4 percent of the total deaths among the white group. Among colored males it was the cause of $\mathbf{3}$ deaths, or 2.9 percent of the heart disease mortality, and among colored females it resulted in 4 deaths, or 4.5 percent. In the entire colored group these lesions accounted for 7 deaths-3.6 percent of the total. The lower percentage of deaths
due to this type of heart disease among colored persons is probably a reflection of the lower incidence of rheumatic heart disease, the chief predisposing factor.

Ages at death.-The average age at death was 36.4 years. Three deaths occurred between 10-19 years, 2 between $20-29$ years, 7 between 30-39 years, 5 between 40-49 years, and 4 between $50-59$ years of age. That no deaths occurred under 10 years of age or past 60 is in keeping with other observations.

Clinical and necropsy diagnoses.-Among these 21 deaths, 11 were diagnosed clinically as having lesions of the mitral valve, 4 as having aortic valvular insufficiency, 3 as having combined lesions of aortic and mitral valves, 2 as other combinations of valvular lesions, and 1 as a case of purulent pericarditis. Twelve necropsies were obtained, in 6 of which vegetations were located on the mitral valve, in 2 on both mitral and aortic valves, in 3 on other combinations of valves, and 1 was a case of purulent pericarditis.

Preexisting lesions.-Infective or subacute bacterial endocarditis, especially when due to St . viridans, is commonly engrafted on preexisting rheumatic or congenital lesions. There was evidence of old rheumatic heart disease in 12 of the 21 cases. In none was there evidence of congenital maldevelopments or of syphilis of the heart and sorta.

Blood cultures.-Blood cultures were recorded in 10 of the 21 cases, 6 of which were positive. In all of these the organisms were nonhemolytic streptococci, identified as St. viridans in five instances.

Blood Wassermann and Kahn reactions.-Blood Wasserman or Kahn reactions were obtained in 14 cases, only 1 of which was positive. This tends to refute the belief that in this condition falsely positive serology is frequent.

Blood pressure determinations.-Blood pressure determinations were recorded in 17 cases, only 2 of which were elevated.

## CONGENITAL HEART DISEASE

Congenital cardiac malformations resulted in nine deaths, or 2.0 percent of this series. All deaths occurred under 10 years of age, the average age at death being about 3 months. These patients were generally physically below par in many respects. Among the associated conditions were otitis media, enteric infections, infected umbilical cords, and congenital syphilis. In one case there was a congenital malformation of the chest. Attention is invited to the frequency with which this condition is attended by other congenital maldevelopments. It is quite probable that, in this group, there were other serious malformations or deformities not detected, as there were no post-mortem examinations performed.

Thyrotoxic heart disease was responsible for six deaths, or 1.3 percent of the deaths from heart disease in the group under study. Grave's discase, or exophthalmic goiter, was the form of hyperthyroidism in 5 fatal cases and a toxic adenoma in 1 case. All of the fatal cases were among females, 4 of which were colored and 2 white. The average age at death was 44.7 years. Two deaths occurred in the age group 30-39 years, 3 in the 40-49 year age period, and 1 in the 60-69 year age period, the last case dying at the age of 67 years of heart disease the result of a toxic adenoma of the thyroid gland.

HEART DISEASE DUE TO MISCELLANEOUS AND UNDETERMINED CAUSES
Cor pulmonale.-There were two deaths due to cor pulmonale. This type of heart disease is by no means rare, and is due to pulmonary emphysema and to endarteritis and arteriosclerosis of the pulmonary arteries and their branches. As a result of these changes in the pulmonary circulation, the right ventricle must overcome an increased resistance. This produces hypertrophy and dilatation and subsequent failure of the right side of the heart.

Tuberculous pericarditis.-Tuberculosis is but an infrequent cause of heart disease; but, when occurring, it is manifested in the form of tuberculous pericarditis, and more rarely as a tuberculous myocarditis or endocarditis. There were other cases of tuberculous pericarditis encountered in reviewing hospital records, but only one case was discovered in which death appeared to be due to congestive heart failure rather than to extensive tuberculous involvement.

Heart disease of undetermined etiology.-In 21 cases, or 5.7 percent, of this series either no etiologica factor could be ascribed, or there was evidence of two or more factors being involved in the etiology of the condition, but it was impossible on the basis of the information at hand to determine their relative significance.

## SUMMARY AND CONCLUSIONS

The etiological factors in 450 deaths from heart disease occurring in Washington (D. C.), hospitals during 1932 have been studied. Arteriosclerotic-hypertensive diseases resulted in 61.4 percent, rheumatic heart disease in 13.3 percent, syphilitic aortitis with its extensions and complications (syphilitic heart disease) in 12.0 percent, bacterial endocarditides and pericarditis in 4.7 percent, congenital cardiac malformations in 2.0 percent, thyrotoxic heart disease in 1.3 percent, other conditions in 0.6 percent, and in 4.7 percent the etiological factor was undetermined.

There were considerable variations according to sex and color. Rheumatic heart disease is more common in the white race, particu-
larly among females, than in the colored race. The degenerative diseases, particularly hypertensive heart disease, are common to all races and both sexes. Hypertension is especially fatal in the colored race, more so among females than among males. Coronary arteriosclerosis and thrombosis are uncommon among Negroes. Syphilis of the aorta and heart is a very common cause of death among colored males, but not so common among colored females.

The importance of arterial hypertension in causing deaths in middle age can hardly be overstressed. In the degenerative forms of heart disease in persons under 50 years of age hypertension was evident in nearly all cases.

Heart disease results in death considerably earlier in the colored race than in the white. This is due in part to the greater prevalence of cardiovascular syphilis and to the greater frequency of arterial hypertension and to the more rapid progression of the degenerative diseases.

## ACKNOW LEDGMENTS

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## COURT DECISION ON PUBLIC HEALTH

Glass in bottle of cream held violation of agriculture and markets law.(New York Supreme Court, Appellate Division; Bourcheix v. Willow Brook Dairy, Inc., 277 N. Y. S. 292; decided February 15, 1935.) Section 50 of the agriculture and markets law provided in part as follows:

No person shall sell or exchange or offer or expose for sale or exchange any unclean, impure, unhealthy, adulterated, or unwholesome milk or any cream from the same, or any unclean, impure, unhealthy, adulterated, colored, or unwholesome cream * * *.

In an action brought against the defendant corporation the following was filed by the majority of the court:

Per curiam: Judgment in favor of plaintiff, in action predicated upon the theory of negligence to recover damages for personal injuries resulting from the consumption of foodstuffs unfit for that purpose, and order denying defendant's motion for a new trial affirmed, with costs. No opinion.

In a memorandum setting forth the views of the dissenting justices it was stated that there was agreement that, if the presence of the glass in the bottle of cream was a violation of section 50 above quoted, it would constitute negligence as a matter of law. The majority of the court took the view that section 50 applied to a case where there was glass in a bottle of cream, while the minority were not in accord with this view.

## DEATHS DURING WEEK ENDED AUG. 3, 1935

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


## PREVALENCE OF DISEASE

No health department, State or looal, can effectively prevent or control disease without lnowledge 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 recaived by the State health officers

## Reports for Weeks Ended Aug. 10, 1935, and Aug. 11, 1934

Cases af certain communicable diseases reported by telegraph by State health officers for woeeks ended Aug. 10, 1935, and Aug. 11, 1934

|  |  |  |  |  |
| :---: | ---: | ---: | ---: | ---: | ---: | ---: | ---: | ---: |

[^2]Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Aug. 10, 1935, and Aug. 11, 1934-Continued

|  |  |  |  |  |  |
| :---: | ---: | ---: | ---: | ---: | ---: | ---: | ---: | ---: |

[^3]Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Aug. 10, 1935, and Aug. 11, 1994-Continued


1 New York City only.
${ }^{2}$ Rocky Mountain spotted fever, week ended Aug. 10, 1935, 17 cases, as follows: North Dakota, 1; Maryland, 5; Virginia, 4; West Virginia, 4; North Carolina, 1; Montana, 1; Oregon, 1.
${ }^{8}$ Week ended earlier than Saturday.
${ }^{6}$ Typhus fever, week ended Aug. 10, 1935, 58 cases, as follows: South Carolina, 2; Georgia, 19; Florida, 1; Alabama, 24; Texas, 12.
${ }^{6}$ Exclusive of Oklahoma City and Tulsa.

## 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.


| July 1985 | July 1935-Continued |
| :---: | :---: |
| Chicken pos: Cases | Mumps: Cases |
| Arkansas.................. 38 | Arkansas.................. 35 |
| Conmettiout....-.-....-.. 208 | Connecticut............... 69 |
| District of Columbia..... 18 | Indian9....................- 33 |
| Indians.......-............ 24 | Iowa-......-..............- 110 |
|  | Missouri.................... 131 |
| Missourl --7.-.-.-.......s. 45 | Vermont ..................... 38 |
| North Carolina...-.-.....- ${ }^{35}$ | Ophthalmis neonatorum: |
| Varmont.................... 36 | Connecticut |
| Conjunctivitis, infectious: | North Carolina |
| Connecticut....-.-.-..... 12 | Paratyphoid fover: |
| Dysentery: | Connecticut_...........- 2 |
| Arkanets (baciliary) ....- | North Carolina............ 12 |
| Conneeticut (bacillary).- 1 | Rabies in animals: |
| Missourl.....-......-.... 58 | Indiana_-..................- 91 |
| Epldemic encephalitis: | Missouri........................ 2 |
| Distriot of Colambia.....- 1 | Rocky Mountain spotted |
| Indiana. | fever: |
| Missouri. | Diatrict of Columbia..... 2 |
| Cerman maasles: | Iowa....-.-..............- 3 |
| Connecticut. . . . . . . . . . . 125 | North Carolina.........-- 5 |
| Iow8-1..................- 7 | Septic soto throat: |
| North Caroling...........- 13 | Connecticut....--...-.-- ${ }^{9}$ |
| Vermont...-............... 236 | Missouri...-................-. 30 |
| Impeligo contagiosa: | North Carolina........... 10 |
| Iowa......- ............... 2 | Tetanus: |
| Lead poisoning: | Connecticut.............-- 2 |
| Connecticut. | Missouri.....................-. 2 |

July 1986-Continued
Trachoma: Cases
Arkansas ..... 13
Connecticut
Missouri1
8
Trichinosis: ..... 2
Tularaemia:
Arkansas ..... 5
District of Columbia
Missouri ..... 1
3
Nortb Carolina ..... 1
Typhus fever:
Missouri. ..... 1
Undulant fever:
Connecticut
District of Columbia..... 10
Iowa
Missouri10
North Carolina
Vermont ..... 16
3
Whooping cough:
Arkansas ..... 75
Connecticut ..... 164

District of Columbia ..... | 164 |
| :--- |
| 20 |

Indiana ..... 146
Iowa. ..... 89
Missour ..... 29
North Carolina
Vermont ..... 125

## PLAGUE-INFECTED GROUND SQUIRREL IN LASSEN COUNTY, CALIF.

## The Director of Public Health of California has reported one plague-

 infected ground squirrel received at the laboratory on July 11, 1935, from a ranch in Lassen County, Calif., 14 miles east and 10 miles south of Adin.
## WEEKLY REPORTS FROM CITIES

## City reports for week ended Aug. S, 1935

This table summarizes the reports received weekly from a selected list of 140 cities for the purposes of showing a cross section of the current urban incidence of the communicable disesses listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and fled for refecence.


City. reports for week ended Aug. s, 1935-Continued


City reports for week ended Aug. S, 1935-Continued


City reports for week ended Aug. s, 1935-Continued


[^4]
# FOREIGN AND INSULAR 

## CANADA

Provinces-Communicable diseases-2 weeks ended July 18, 1935.During the 2 weeks ended July 13, 1935, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada, as follows:

| Disease | Prince ward | Nova Scotia | $\left\|\begin{array}{c} \text { Now } \\ \text { Bruns- } \\ \text { wick } \end{array}\right\|$ | $\begin{aligned} & \text { Que- } \\ & \text { bec } \end{aligned}$ | Ontario | $\begin{gathered} \text { Mani- } \\ \text { toba } \end{gathered}$ | Sas-katchewan | $\begin{gathered} \text { Alber- } \\ \text { ta } \end{gathered}$ |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Cerebrospinal meningitis. |  |  |  | 2 | 1 |  |  |  |  | 3 |
| Chicken pox |  |  | 1 | 108 | 391 | 45 | 97 | 5 | 93 | 740 |
| Diphtheria. |  | 1 |  | 34 | 13 | 5 | 5 |  |  | 58 |
| Dysentery- |  | 2 |  | 4 | 1 | 3 |  |  | 1 | 18 |
| Influenzs. |  | 3 | 2 |  |  | 2 |  |  |  | 7 |
| Lethargic encephalitis.- |  |  | 1 |  |  |  |  |  |  | 1 |
| Measles...- |  | 10 | 28 | 388 | 2,955 | 139 | 359 | 28 | 259 | 4, 160 |
| Mumps ...--- |  | 14 |  |  | 216 | 60 | 8 |  | 5 | 303 |
| Paratyphoid fever |  | 1 |  |  | 1 |  | 3 |  | 7 | 12 |
| Poliomyelitis.-..........-- |  | 1 |  | 2 | 1 |  | 3 |  | 7 | 20 |
| Scarlet fever. | 1 | 31 | 7 | 162 | 210 | 31 | 23 | 6 | 20 | 491 |
| Smallpox.-. |  |  |  |  | 2 |  |  |  |  | 2 |
| Trachoms--- |  |  |  |  |  |  | 1 |  |  | 1 |
| Tuberculosis... | 8 | 49 | 28 | 152 | 105 | 32 | 1 | 4 |  | 398 |
| Typhoid fever-- |  |  | 3 | 37 | 8 |  | 3 | 2 | 6 | 59 |
| Whooping cough......-- |  | 29 | 9 | 73 | 317 | 41 | 97 |  | 53 | 619 |

Note.-No report was received from Alberta for the week ended July 6, 1935.

## ITALY

Communicable diseases-4 weeks ended June 23, 1935.-During the 4 weeks ended June 23, 1935, cases of certain communicable diseases were reported in Italy as follows:

| Disease | May 27-June 2 |  | June 3-9 |  | June 10-16 |  | June 17-23 |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Cases | Commanes affected | Cases | Communes affected | Cases | Communes affected | Cases | Communes affected |
| Anthrax. | 20 | 19 | 11 | 10 | 27 | 26 | 14 | 13 |
| Cerebrospinal meningitis | 18 | 15 | 12 | 11 | 15 | 13 | 12 | 12 |
| Chicken pox........... | 428 | 182 | 542 | 177 | 477 | 201 | 353 | 153 |
| Diphtheria and croup. | 445 | 227 | 362 | 195 | 362 | 179 | 312 | 168 |
| Dysentery-....... | 8 | 4 | 4 | 4 | 8 | 8 | 15 | 9 |
| Hookworm disease--- | 11 | ${ }_{5}^{6}$ | 40 | 8 | 16 | 11 | 18 | 9 |
| Lethargic encephalitis. |  | ${ }^{5}$ |  | ${ }^{3}$ |  |  | ${ }^{1} 1$ | 308 |
| Measles.. | 2,760 | 457 | 2,546 | 445 | 2, 292 | 449 | 2,099 | 306 |
| Paratyphoid fever | 47 | 34 | 53 | 41 | 47 | 36 | 73 | 54 |
| Poliomyelitis... | 8 | 7 | 6 |  | 14 | 12 | 23 | 14 |
| Puerperal fever. | 30 | 30 | 40 | 35 | 27 | 27 | 38 | 36 |
| Scarlet fever. | 356 | 129 | 388 | 130 | 314 | 117 | 327 | 103 |
| Typhoid fever. | 280 | 138 | 247 | 147 | 233 | 143 | 418 | 158 |
| Undulant fever | 82 | 53 | 109 | 73 | 87 | 64 | 102 | 76 |
| Whooping cough.... | 287 | 99 | 438 | 117 | 342 | 103 | 416 | 121 |

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER
(Norz.-A table giving current information of the world prevalence of quarantinable diseases appeared in the Public Healti Reports for July 26, 1935, pp. 967-983. A similar cumulative table will appear in the Public Healti Reports to be issued Aug. 30, 1935, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

## Cholera

India-Vizagapatam.-During the week ended August 3, 1935, 1 case of cholera was reported at Vizagapatam, India.

Siam-Smudsongram Province.-During the week ended August 3, 1935, 1 case of cholera was reported in the Province of Smudsongram, Siam.

## Plague

Hawaii Territory-Maui Island-Makawao District-Kahului.During the week ended August 3, 1935, 2 plague-infected rats were found about 9 miles from the port of Kahului, Makawao District, Maui Island, Hawaii Territory.

United States-California.-A report of 1 plague-infected ground squirrel in California will be found on page 1158 of this issue of Public Health Reports.

## Yellow fever

Colombia-Intendencia of Meta-Restrepo.-During the week ended July 20, 1935, 1 fatal case of the jungle type of yellow fever was reported at Restrepo, Intendencia of Meta, Colombia.

Gold Coast-Cape Coast.-On August 6, 1935, 1 fatal case of yellow fever was reported at Cape Coast, Gold Coast.


[^0]:    ${ }^{1}$ The report for the fourth quarter and the entire year of 1934 was published in the Public Hraltr Reports for April 26, 1935, vol. 50, no. 17, pp. 557-559.

[^1]:    ${ }^{2}$ Statistical Bulletin, Metropolitan Life Insurance Company, vol. 16, no. 4, April 1835, p. \&

[^2]:    See footnotes at end of tabla.

[^3]:    See footnotes at end of table.

[^4]:    Denguse Atlanta, 1 case.
    Apidewic encephalitis.-Cases: Cleveland, 1; Indianapolis, 3; Kansas City, Mo., I; St. Louis, 1
    Pellagra_-Casas: Washington, 2; Winston-Salem, 1; Charleston; 8. C., 1; Memphis, 2; Dallas, 2; Lee: Angeles 2; San Franeisco, 4.
    Typhut fever.-Gaser: Wimington, N. O 1; Savannah; 2; Mobtlo, 1; Montgomery; 2; Eouston, 1.

