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EXPERIMENTAL CHEMOTHERAPY OF BURNS AND SHOCK I. METHODS. II. EFFECTS OF LOCAL THERAPY UPON MORTALITY FROM SHOCK [1]

By SANFORD M. ROSENTHAL, *Principal Pharmacologist, United States Public Health Service*

The experimental approach to the evaluation of therapeutic agents employed in burns and shock has been a difficult task and the results obtained have been to a large measure controversial. The use of large laboratory animals has been complicated by such factors as anesthesia, difficulty in producing a uniform degree of shock, brief duration of experiments, and the inability to employ under uniform conditions a sufficient number of animals to yield values of statistical significance. No review of the literature will be attempted. Recent surveys in this field have been made by Wiggers (*1*) and by Harkins (*2*).

The need for a simplified procedure that can be carried out upon small laboratory animals is apparent. This seemed to be best filled by exposing mice to a standardized degree of heat and accepting the time-mortality curve as a criterion of assay. As the work has progressed several factors responsible for variations have been revealed, and an increasing uniformity in the production of shock has been realized.

I. Methods

Female albino mice of an inbred strain raised at the Institute were employed. They were allowed free access to food and water at all times. The hair was removed from the posterior two-thirds of the animal by means of electric clippers. The line of shearing was extended approximately to the level of the xyphoid process.

Anesthesia was produced by dropping the mouse in the bottom of a large jar containing coarse mesh wire gauze placed over a layer of cotton which had been saturated with ether. Anesthesia was obtained in 10 to 20 seconds. The burn was produced by seizing the animal behind the neck with a pair of forceps and immersing the shaved

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

area into a beaker of water maintained at a constant temperature. The forelegs were held in such a position that the forepaws were often also immersed. The animal was dried by gently blotting with paper towels, and placed upon sawdust in a bucket. Only a few seconds of exposure are required so that within 1 to 2 minutes from the onset of anesthesia the animal is able to run about the bucket. Mice can be subjected to this exposure at the rate of two per minute. In a given experiment all exposed mice are placed together to insure homogeneity, and then selected by rotation for each group to be studied.

After experimenting with temperatures from 50° to 99° C., a selection of 70° C. was made as best suited to our needs. At this temperature, dependent upon length of exposure and other factors to be discussed, three types of effects can be obtained: (*a*) Animals exposed for 10 seconds die within a few hours. (*b*) Following exposure for 3 to 5 seconds the animals recover from the acute effects and run about the cage for several hours, showing irritability and increased respiratory rate. They gradually develop increasing dyspnea and prostration and from 30 to 100 percent die within 3 days. Some hours prior to death the skin becomes cold and clammy. (*c*) Mice surviving this period usually die within 3 days to 3 weeks with symptoms and gross autopsy findings indicative of toxemia and secondary infection. It is possible to adjust conditions so that the majority of deaths occur within this period.

Locally, swelling of the legs and tail develops within an hour following immersion. Within 1 to 2 days gangrene of these parts begins. In most animals this is of the dry type with shrinking of the affected areas; in others, the legs and tail remain swollen and moist, with infection appearing in those animals that have a sufficiently long period of survival.

HEMOCONCENTRATION

The presence of hemoconcentration was established in a series of mice as a corollary to the observed symptoms of shock. Hemoglobin determinations were made the day prior to exposure in 10 mice weighing 28 to 33 gm., upon 0.01 ml. of blood obtained from the tail. Five and one-half to seven and one-half hours after an exposure of 4 seconds at 70° C., the determinations were repeated upon blood obtained from the heart, as local edema fluid made values from tail blood inaccurate. A mean of 127 percent (table 1) of the control value was obtained, which is significant in view of the fact that under these conditions only part of the animals die within the first few days.

TABLE 1.—*Hemoglobin values before and 6½ to 7½ hours after production of a standardized burn in 10 mice*

[Exposure 4 seconds at 70° C.]

Hemoglobin before exposure (Gm. percent)	Hemoglobin after exposure (Gm. percent)
13.5	16.5
10.5	14.0
13.5	17.5
12.5	18.3
12.6	15.0
16.5	18.7
15.8	21.4
14.0	18.5
14.5	16.8
13.8	17.8
[1] 13.72	[1] 17.45

[1] Mean.

INFLUENCE OF AGE AND ENVIRONMENTAL TEMPERATURE

While a sufficient number of control animals was employed in each experiment to make deviations significant, it was desired to obtain time-mortality curves, as uniform as possible, upon the controls. The age (size) of the mice and the temperature of the room were found to affect results appreciably.

Mice of 30 to 35 gm. were immersed for 4 seconds in water at 70° C. and groups of 15 were placed (*a*) in the cold room at 5° C., (*b*) in the incubator room at 37° C., (*c*) in a room maintained at 26.6° to 30° C., (*d*) in a room at 18.3° to 20.5° C.

Animals in groups (a) and (b) died within 10 hours. The mortality curve of group (d) was appreciably higher than that of group (c) (fig. 1). The sensitivity of these animals to changes in environmental temperature suggests that a disturbance in the ability to regulate body temperature may be an important factor in the mechanism of shock produced by extensive scalds. The unfavorable influence of heat and cold are of interest in view of the recent experiments of Blalock (*3*) and Wiggers (*4*) on the survival time of anesthetized dogs with traumatic shock, as influenced by the local and general applications of heat and cold to the body. Their results suggested a possible beneficial effect from cold.

In addition to the large mice employed in the above experiments, a group of 15 mice weighing 14 to 18 gm. were included and kept in the room with group (c) at 26.6° to 30° C. The young animals proved to be much more susceptible to thermal shock, with a mortality of 80 percent within 24 hours, as compared to 20 percent for the older mice.

It is evident that mice of a fairly uniform size kept at an even room temperature are prerequisites for the standardization of a time-mortality curve. There are undoubtedly other influences, such as the degree of hydration of the animal, relation to feeding, etc., that deserve study. In the latter part of the work reported below it was attempted to employ mice weighing 20 to 25 gm. for all experiments and to maintain the room temperature between 26.6° and 30° C.

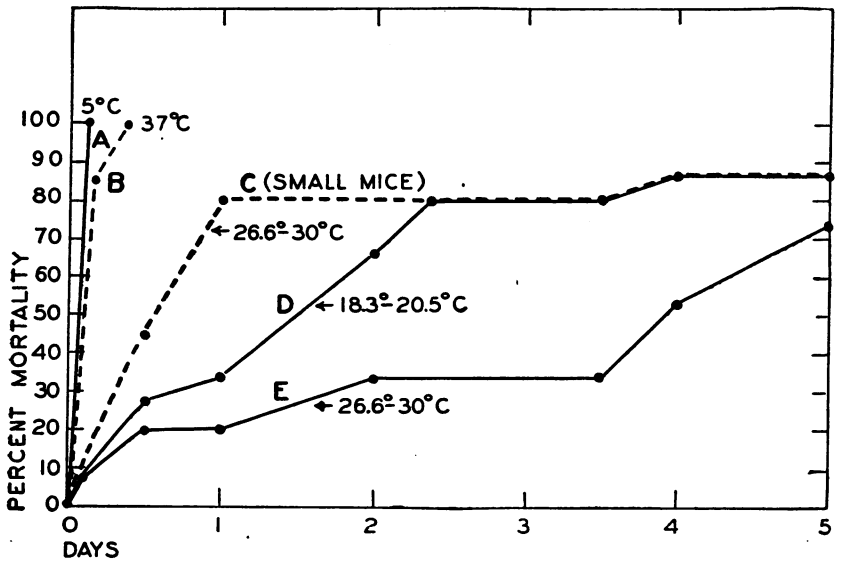

FIGURE 1.—The influence of size of mice and of room temperature on early mortality following a standard burn. In curve C, mice of 14 to 18 gm. were used. All others were old mice of 30 to 35 gm. Fifteen mice were in each group and were immersed in water at 70° C. for 4 seconds.

II. Effects of Local Therapy upon Mortality from Shock

For the assay of therapeutic measures it was desired to have 40 to 60 percent of the control animals succumb within 3 days. The majority of experiments fall within this range. However, when these experiments were begun, the influence of age and of room temperature was not fully appreciated. Mice of fairly uniform size were always used for each experiment, but variations have been present in different experiments, and the mortality curves among control groups have likewise varied. However, the significant criterion is the deviation from the control which occurs as a result of treatment, and these deviations have in repeated experiments yielded results of a constancy that testify to the reliability of the procedure and that can be subjected to statistical analysis. In some cases it may be advantageous to have high or low control curves in order to bring out better the influence of therapy.

The temperature of the water into which the mice were immersed was 70° C. in all cases. With a few exceptions the length of exposure was 4 seconds. The occasional mouse that showed immediate collapse as a result of exposure was discarded before selection into groups was made. An average of approximately 1 hour elapsed between exposure to heat and institution of therapy. Local therapy was applied by dipping the exposed area of the animal into a small conical glass containing the substance to be tested.

EFFECTS OF COD LIVER OIL AND MINERAL OIL

Three experiments each were performed to test the therapeutic effects of cod liver oil, U. S. P., and mineral oil, light, U. S. P. The mice were dipped into the oil within an hour after the scald and again 24 hours later.[2] In all experiments a striking increase in mortality during the first 3 days occurred as a result of this treatment; no appreciable difference was observed between cod liver oil and mineral oil. The mortality curves in the former group were slightly higher, but in both cases the increase in mortality, as compared with the corresponding controls, was doubled during the first 3 days as a result of treatment (table 2, figs. 2 and 3).

TABLE 2.—*A summary of experiments showing the influence of various substances applied locally on the acute mortality of mice subjected to a standardized burn. The difference between the mortality of the control and of the treated groups and its probable error ($P.E._{Diff.}$) is shown on each day following the burn*[1]

	Number of mice	1 day—percent mortality	Difference ± P. E. Diff.	2 days—percent mortality	Difference ± P. E. Diff	3 days—percent mortality	Difference ± P. E. Diff.
Cod liver oil	41	95.1	47.5±5.7	100	47.6±5.2	100	38.0±5.1
Controls	42	47.6		52.4		62	
Mineral oil	44	70.5	35.7±6.6	81.8	42.6±6.2	88.6	43.0±5.9
Controls	46	34.8		39.2		45.6	
10 percent tannic acid solution	44	75	37.3±6.7	84.1	44.1±6.1	84.1	41.9±6.3
Controls	43	37.7		40		42.2	
Tannic acid-sulfadiazine ointment	37	54	24.8±7.3	64.8	28.2±7.3	70.2	16.6±7.3
Controls	41	29.2		36.6		53.6	
5 percent sodium sulfadiazine	29	96.5	43.2±6.6	96.5	43.2±6.6	96.5	43.2±6.6
Controls	30	53.3		53.3		53.3	
Ringer's solution	48	50	18.7±6.6	60.4	8.3±6.6	62.5	12.5±6.3
Controls	48	68.7		68.7		75.0	
Epinephrine	34	35.3	38.2±7.5	44.1	29.4±7.7	55.8	26.7±7.2
Ringer's solution	34	55.8	17.7±7.7	61.7	11.8±7.6	64.7	17.8±7.1
Controls	34	73.5		73.5		82.5	

[1] Dr. Selwyn D. Collins, Principal Statistician of the Division of Public Health Methods, kindly conducted the statistical analysis of this material.

The mechanism is not known by which application of oil to large areas of body surface following burns brings about an increase in early mortality.

[2] It was not possible to prevent the oil from spreading over the unburned area.

EFFECTS OF TANNIC ACID

Three experiments were carried out with 10 percent aqueous solutions of tannic acid, U. S. P. Two batches of the drug were used. The mice were immersed in the solution at 1, 4, 7, and 24 hours follow-

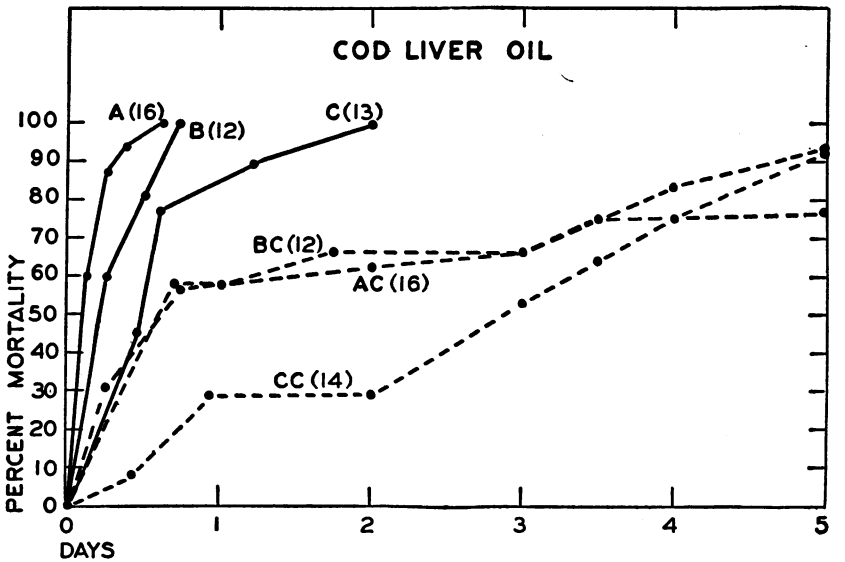

FIGURE 2.—The effect of external application of cod liver oil on early mortality following burns. Curves A, B, and C are treated animals; AC, BC, and CC are the corresponding controls. Figures in parentheses indicate the number of mice used.

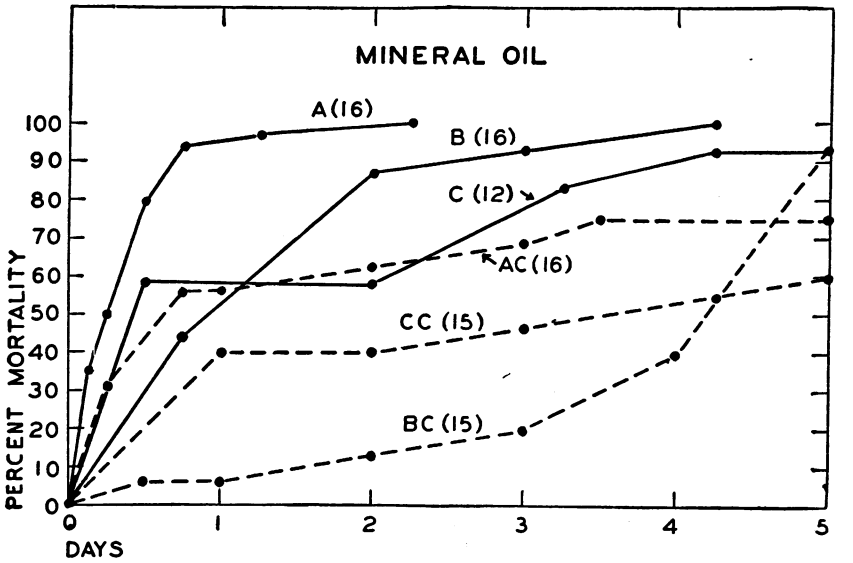

FIGURE 3.—Three experiments with mineral oil externally. A, B, and C are treated mice; AC, BC, and CC are the corresponding controls. Figures in parentheses indicate the number of mice used.

ing the burn. In these experiments early mortality was doubled as a result of this treatment (table 2 and fig. 4).

An experiment with tannic acid solutions in concentrations of 2.5, 10, and 20 percent indicated a correlation between the strength of the solution and the increase in mortality resulting from its use (fig. 5).

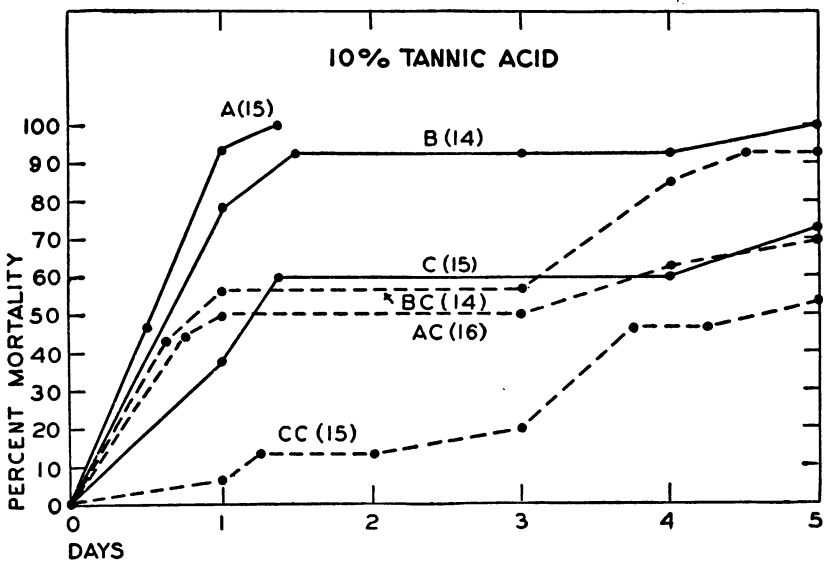

FIGURE 4.—Three experiments with 10 percent tannic acid solution locally. A, B, and C are treated mice AC, BC, and CC are the controls.

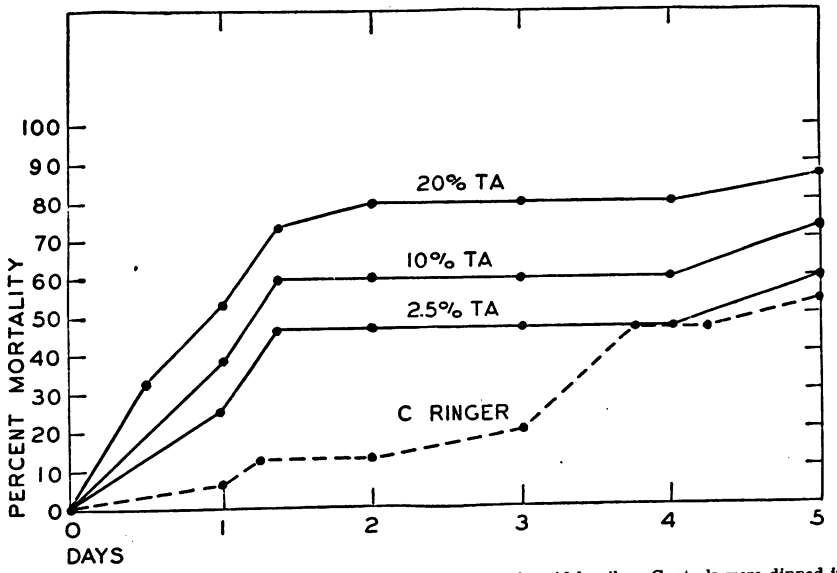

FIGURE 5.—One experiment with varying concentration of tannic acid locally. Controls were dipped in Ringer's solution. Fifteen mice were in each group.

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An ointment of 10 percent tannic acid and 5 percent sulfadiazine made according to a recommended formula[3] was employed in three experiments with 37 mice and 41 controls. The ointment was applied

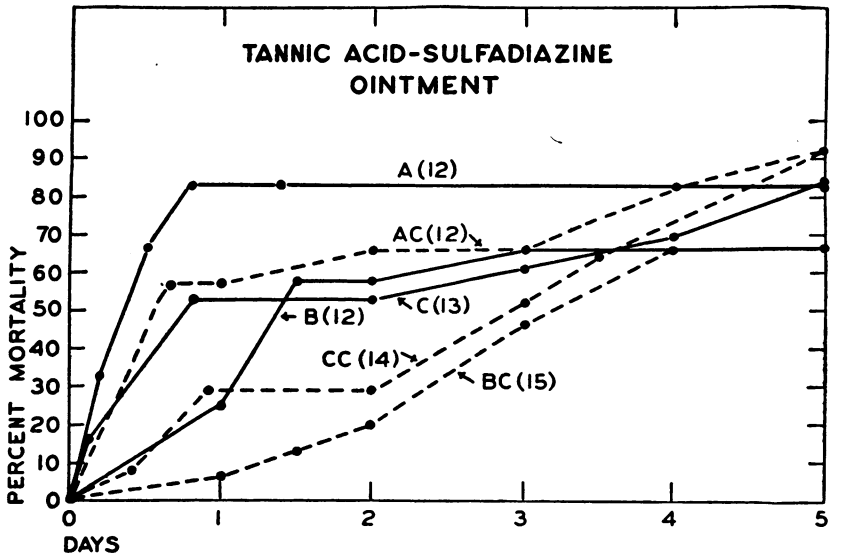

FIGURE 6.—Three experiments with an ointment containing 10 percent tannic acid and 5 percent sulfadiazine in a pectin base. A, B, and C are treated animals; AC, BC, and CC are the controls. The number of mice used is shown in parentheses.

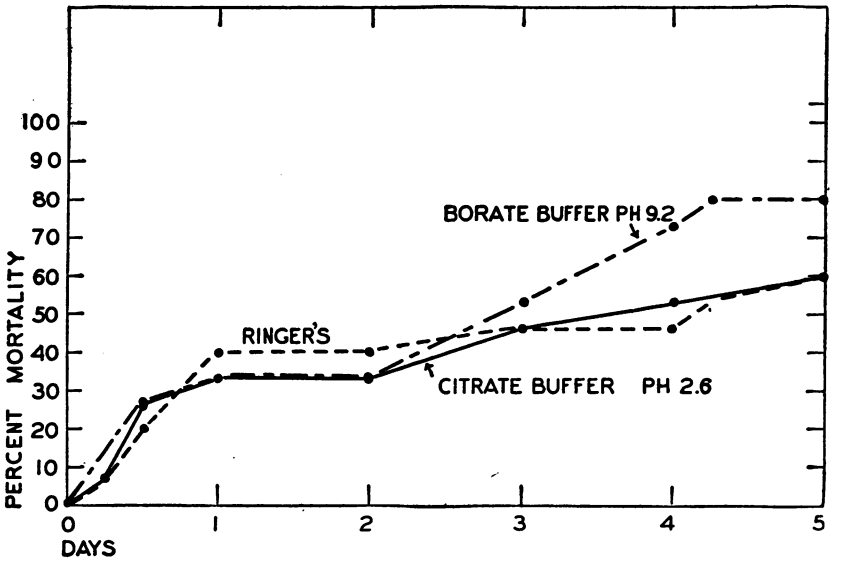

FIGURE 7.—The absence of effect of local applications of buffer solutions of pH 2.6 and 9.2 as compared with Ringer's solution, on early mortality following burns. Fifteen mice were in each group.

[3] Suggested in J. Am. Pharmaceut. Assoc., 3: 236 (1942). This ointment contained tannic acid 10 percent, sulfadiazine 5 percent, glycerin 12 percent, sodium sulfite 0.2 percent, Ringer's solution 67.6 percent, and pectin 5 percent.

within an hour of the burn and repeated in 24 hours. The mortality of the treated animals was 54.2 percent in 1 day and 70.5 percent in 3 days as compared to 29.2 and 53.6 percent among the controls (table 2, fig. 6). Later experiments revealed that sulfadiazine applications did not appreciably influence the mortality curve, and it is believed that the increase in mortality rate resulted from the tannic acid present. It remains to be established whether systemic absorption of tannic acid through the burned area contributed to this effect.

In order to eliminate the possibility that the increased mortality was due to the acidity of the preparations, a group of 15 mice were

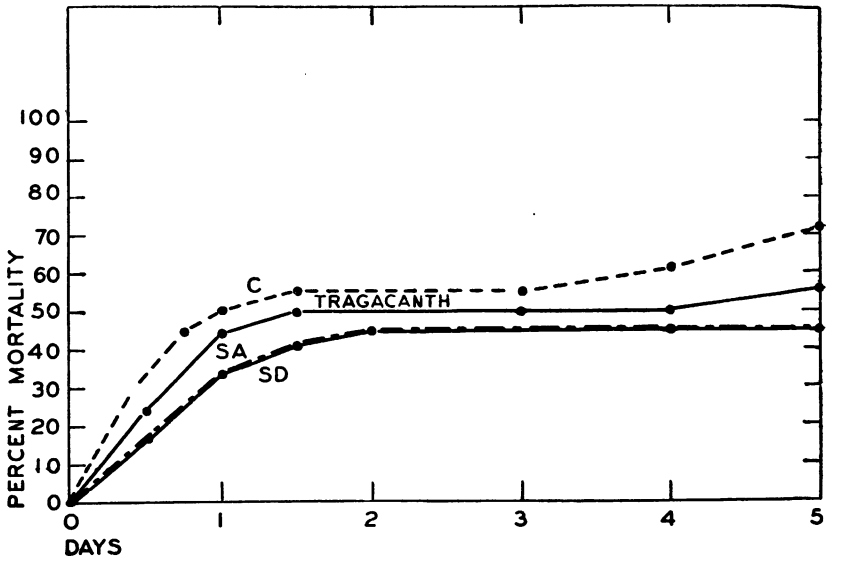

FIGURE 8.—The absence of effect of 5 percent sulfanilamide and 3 percent sulfadiazine jelly, and of tragacanth jelly alone, as compared to untreated animals. Fifteen mice were in each group.

immersed in McIlvaine's citrate-phosphate buffer of the same pH (2.6) as a 10 percent solution of tannic acid. As compared to immersion in Ringer's solution, no effect on the mortality curve was produced (fig. 7).

SULFANILAMIDE, SULFADIAZINE, AND SODIUM SULFADIAZINE

Because of the stickiness of the pectin ointment, a jelly of 2 percent tragacanth in water was used as a vehicle in these experiments. With 18 mice employed in each group it was found that three applications of the tragacanth jelly alone, the jelly with 5 percent sulfanilamide, and the jelly with 3 percent sulfadiazine did not appreciably affect the time-mortality curve (fig. 8).

Two experiments were made with a 5 percent aqueous solution of sodium sulfadiazine upon 29 mice. Mice were immersed in this solution at 1 hour, 5 hours, and 24 hours after the scald. The results compared with 30 controls showed approximately double the early mortality (table 2, fig. 9).

Since sulfadiazine jelly did not increase the mortality from shock while sodium sulfadiazine solutions did to a significant degree, experiments were conducted on the effect of pH, and also upon the systemic absorption of sodium sulfadiazine through normal and burned skin.

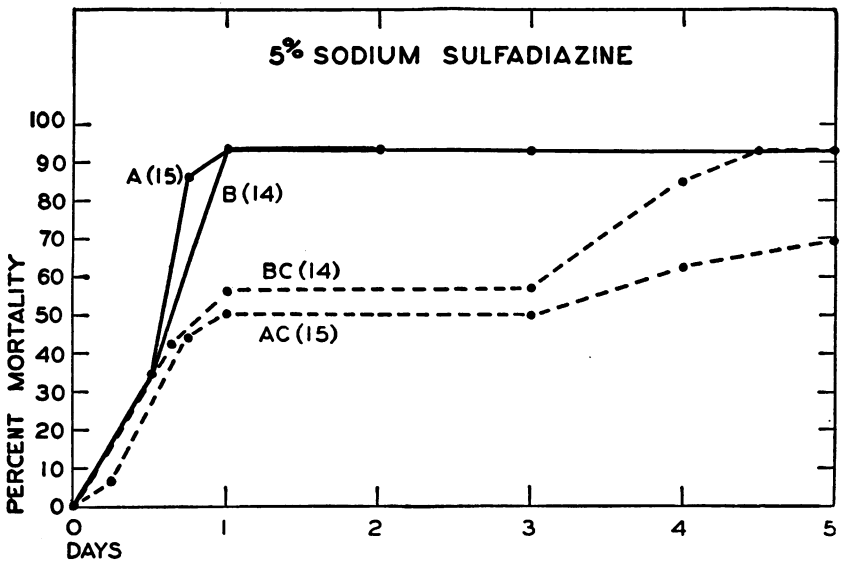

FIGURE 9.—Two experiments on early mortality of burned mice as influenced by local application of a 5 percent sodium sulfadiazine solution. A and B are treated animals, AC and BC are controls. The number of animals in each group is shown in parentheses.

A group of scalded mice were immersed in M/20 sodium borate solution the pH (9.2) of which was similar to that of a 5 percent sodium sulfadiazine solution. Immersion in borate buffer did not alter the time-mortality curve as compared to controls dipped in Ringer's solution (fig. 7).

Ten shaved normal mice of 16 to 18 gm. in weight were immersed in a 5 percent solution of sodium sulfadiazine at 9 a. m. and at 1 p.m. During the day, each mouse was kept in a small wire cylinder fitting sufficiently close so that licking the skin was not possible. Between 4 and 5 p. m. the animals were anesthetized and heparinized blood drawn from the heart with a capillary pipette, using precautions to avoid contamination of the blood with the sulfadiazine on the skin. Determinations made upon 0.05 to 0.1 ml. of blood gave the following values for free sulfadiazine: 2.64, 2.7, 4.0, 4.2, 2.0, 4.5, 6.6, 9.0, 6.0, and 8.0 mg. percent (mean = 4.96 mg. percent).

This experiment was repeated with mice that had received the standard burn one-half hour before the first immersion in sodium sulfadiazine. Two mice died before completion of the experiment. The following values were found: 20, 21.6, 20, 20.1, 23, 18.8, 28, and 20 mg. percent (mean = 21.44).

The validity of the technique was checked by dipping three normal shaved mice into 5 percent sodium sulfadiazine and obtaining blood samples immediately after. No sulfadiazine was detected.

Our results indicate that sufficient sodium sulfadiazine can be absorbed from the burned area to contribute materially to the increased mortality observed under these conditions. Other experiments which we have done suggest that this absorption is greater in young mice than in old. That absorption of sulfadiazine can occur through burned human skin is also indicated by the report of Pickrell (*5*).[2] These findings are contradictory to the earlier experimental work of Underhill *(6)* on the absorption of strychnine. To demonstrate the absorption of strychnine through a burned area 5 mice were dipped in a 1 percent solution of strychnine sulfate one-half hour after the burn and placed in wire cyclinders as above. All died in convulsions within 10 minutes. Five normal mice similarly treated remained well during the 7 hours they were kept in the cylinders.

EPINEPHRINE, POSTERIOR PITUITARY EXTRACT, AND RINGER'S SOLUTION

In three experiments 48 mice were immersed in Ringer's solution or 0.8 percent saline four times during the first day and three times the second day following the burn. As compared to 48 controls a slight decrease in mortality rate was observed (table 2). The addition of 1-20,000 epinephrine to the solutions brought about an additional decrease in the mortality curve to an extent that is significant statistically (table 2, fig. 10). In two experiments there were 34 mice treated with epinephrine, 34 with saline, and 34 controls. The mortality in the epinephrine group was approximately half that of the controls during the first 48 hours.

In one of these experiments an additional group of 15 mice were treated with pituitary powder, 1-8,000. Results comparable to those with epinephrine were obtained. It cannot yet be stated whether the favorable effects of these hormones are due to their local action or to systemic effects as a possible result of absorption through the scalded area. It is of interest that Douglas (*7*) in 1923, on the basis of experimental work, advocated the use of epinephrine packs to produce local vasoconstriction in the therapy of burns.

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[2] The solution of sulfadiazine used by Pickrell does not contain the free compound but a salt formed with triethanolamine analogous to an ammonium salt.

COMMENT

It is believed that the methods outlined will be useful as a means of studying the effects of various agents upon mortality from shock. It must be strongly emphasized that results obtained in this study are not concerned with the effects upon the healing of local lesions. The treatment of burns has been dealt with only insofar as such treatment influences the early mortality from shock and is, therefore, of significance only in extensive and severe burns. That this is an important consideration is shown by the fact that, in man, from 60 to 80 percent

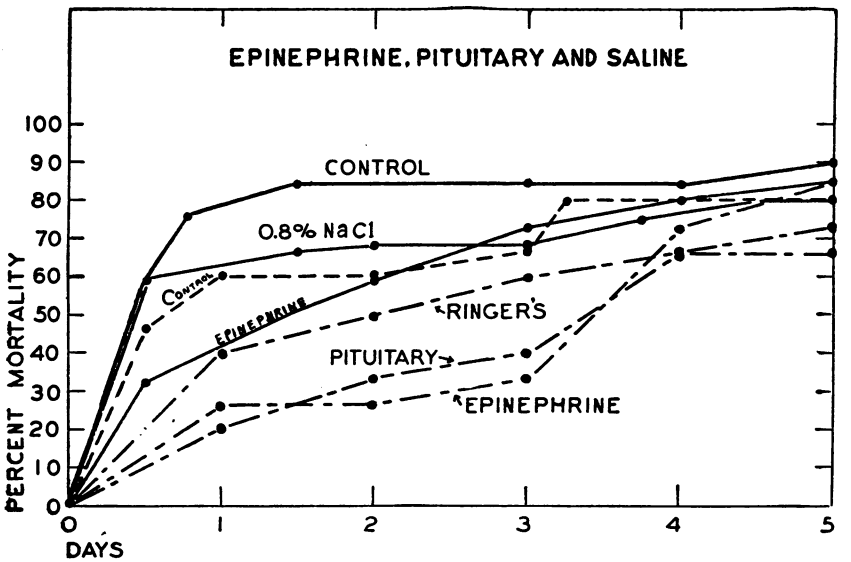

FIGURE 10.—The influence of 1-20,000 epinephrine and 1-8,000 posterior pituitary powder on the early mortality of burned mice as compared with Ringer's or saline applications and with untreated controls. Broken lines represent one experiment with 15 mice in each group; continuous lines, another experiment with 19 mice in each group.

of the deaths from extensive burns occur within the first few days as the result of shock (*8*).

It must also be pointed out that these experiments deal with mortality rates and not ultimate survival. Irrespective of early differences, the curves tended to approach each other after the third day.

This method of study should be useful in evaluating the effects of systemic therapy, including blood and blood substitutes, in shock and toxemia; work is being done on this problem. Whether or not with a modified procedure the time-mortality data will afford information of value in the investigation of the later stages of burns remains to be established.

SUMMARY

A procedure is described whereby a standardized burn is produced in mice. Conditions can be adjusted so that the majority of animals

succumb either during the first few days from shock or later from toxemia and secondary infection.

The age of the mouse and the environmental temperature have been found to influence appreciably the early mortality (within 3 days) following burns. Young mice were more susceptible than old. Any degree of cooling, as well as excessive heating of the room, exerted an unfavorable effect.

A study of agents commonly used in the local therapy of burns revealed that significant increases in early mortality were produced by cod liver oil, mineral oil, tannic acid solutions and ointment, and 5 percent sodium sulfadiazine when applied to a scalded area comprising approximately two-thirds of the body surface.

No significant effect upon early mortality was observed following the application of a 5 percent sulfanilamide jelly or 3 percent sulfadiazine jelly.

Ringer's solution or 0.8 percent saline lowered the early mortality slightly below control values. The addition of epinephrine or posterior pituitary extract to the solution caused a further decrease in mortality, believed to be a significant deviation from the control.

The present study bears upon burns only insofar as their relation to shock is concerned.

REFERENCES

(1) Wiggers, C. J.: The present status of the shock problem. Physiol. Rev., **22**: 74 (1942).
(2) Harkins, H. N.: The Treatment of Burns. Charles C. Thomas, Baltimore, 1942.
(3) Blalock, A., and Mason, M. F.: A comparison of the effects of heat and cold in the prevention and treatment of traumatic shock. Arch. of Surgery, **42**: 1054 (1941); **45**: 183 (1942); also Surgery, **11**: 356 (1942).
(4) Wiggers, C. J.: Applicability of experimental results to the shock problem in man. J. Am. Med. Assoc., **117**: 1143 (1941).
(5) Pickrell, K. L.: A new treatment for burns. Bull. Johns Hopkins Hosp., **69**: 217 (1941).
(6) Underhill, F. P., Kapsinow, R., and Fisk, M. E.: Studies on the mechanism of water exchange in the animal organism. Am. J. Physiol., **95**: 315 (1930).
(7) Douglas, B.: Restriction of rate of flow and interchange in the capillaries. J. Am. Med. Assoc., **81**: 1937 (1923).
(8) Rhoads, J. E., Wolff, W. A., and Lee, W. E.: The use of adrenal cortical extract in the treatment of traumatic shock of burns. Ann. Surg., **113**: 955 (1941).

OBSERVATIONS ON THE EPIDEMIOLOGY OF LEPROSY

By G. W. McCoy, *Medical Director (Retired), United States Public Health Service*

It is not sufficiently well known that at present in many parts of the world leprosy exhibits a tendency to disappear, or at least not to perpetuate itself, while in other parts of the world it tends to spread freely; that is, enough new cases develop to keep the number of lepers relatively constant. These facts are nowhere better illustrated

than in the United States where new cases develop to an extent that makes the disease a matter of public health concern only in certain States bordering on the Gulf of Mexico; elsewhere spread does not occur, or only so slightly as to be of no material importance to the community. These facts, though well known to those who have made a special study of the disease from the public health point of view generally, have not been taken into account in formulating public health laws and regulations of the various States.

Before analyzing in detail the situation in the United States it will be advisable to consider the problem as presented in Europe. There at present leprosy spreads appreciably only in the countries bordering on the Mediterranean and the Baltic. Elsewhere the few cases that develop are due to spread from imported cases and are not a remnant of the leprosy that was so prevalent in the Middle Ages. Endemic leprosy stemming from the widespread distribution of the disease in the Middle Ages is believed to have disappeared from the British Isles about the end of the eighteenth century. The very few cases developing there in the past century or more have been due to infection from imported cases. Many persons suffering from leprosy that was acquired in the colonial parts of the empire go to England either to secure treatment or because it is the home country and has a very liberal policy in dealing with this disease from the public health point of view.

It is well known that at present the tendency of leprosy to spread in the British Isles is so feeble that health authorities make no attempt to control it and that whatever measures are taken by patients are self imposed. This attitude is well justified by experience but it must not be assumed that leprosy never is transmitted in Britian. This is well illustrated by a report made by MacLeod (*1*) in 1925. He reported four cases in persons who never had been out of England; three acquired the disease in childhood, and one in adult life. All were close family contacts of adults who had acquired the disease in countries known to be foci of the infection.

That not everyone is comfortable about the leprosy situation in England is shown by the fact that the matter has come up in Parliament occasionally. In 1938 a short discussion of the subject took place in the House of Commons in which it developed that there were 60 to 100 lepers at large in the country, while there were special facilities for the care of only 12. The Ministry of Health reported that while leprosy was not required to be reported in England the authorities had information of 38 lepers and that in only 4 cases over a long period of years had infection occurred in England. Dr. MacLeod (*2*) has expressed the view that in some cases there is danger of infection and that supervision is advisable.

Flandin and Ragu (*3*) report on 95 cases of leprosy seen in recent years in Paris, France, and vicinity, but only 6 of these were regarded as infected in Paris and the surrounding region. Of these 6, 4 gave histories of close association with lepers, while 2 gave no such histories, but in each case there was mentioned possible sources of infection.

There appears to have been little published on leprosy in Germany except in relation to the outbreak in the Memel region in the latter part of the nineteenth century, so it may be taken for granted that local infections are very rare—indeed Cochrane (*4*), an English authority, remarks, "There is no endemic leprosy in Germany and only eight cases, all foreigners, mostly from Brazil."

Leprosy is endemic in parts of Italy, but cases developing in Rome and vicinity seem to be uncommon since but one example has been found in recent years in which infection probably occurred in that area (*5*).

Available information shows that leprosy which was so prevalent in Norway in the last century has declined so that now it practically has reached the vanishing point.

The communicability of leprosy in the United States has been a subject of much interest and some concern to physicians and health authorities. Better records are available for studying this subject in the city of New York than for any other place in the country, with few exceptions. Approximately half a dozen cases are reported to the health authorities there annually, and always a careful study is made of each one with the view of ascertaining probable contact with leprosy and the place of infection. Although these investigations have been made routinely on a total of at least 100 cases, not one could be discovered that could, beyond reasonable doubt, be attributed to infection acquired in New York, or indeed anywhere else in the northeastern part of the United States.

So far as the records show, but two cases have been discovered in New York which by any impartial interpretation of the evidence might possibly have been infected there. These are as follows:

Case 1.—A young man who had in recent years lived in New York but whose earlier life had been spent in Canada was found to have leprosy. There was no history of association with lepers, either in Canada or in the United States. As the part of Canada in which this young man had lived never has been known to have produced a case of leprosy, the case has tentatively been assigned to New York with a full understanding that the evidence is unsatisfactory.

Case 2.—M. C., male, aged 32, born in New York and lived there nearly all of his life. He was found to have clearly marked nodular leprosy early in 1941. The manifestations were said to have been of 4 months' duration. Just prior to coming to New York where the disease was detected, he had lived in California—4 weeks in San Francisco, and about 3 years in Los Angeles. There was no known contact with leprosy either in California or in New York. The longest possible incubation period, assuming that infection occurred in California, would

be about 3 years—a period not to be given much weight in determining the source of infection. Furthermore, transmission of leprosy is very uncommon in California as will be shown later.

Obviously it is impossible to be certain of the source of infection in either of these interesting cases.

Doubtless the time will come when cases will be found clearly traceable to infection in New York just as it came about that persons infected in Minnesota were discovered, as will appear later. It may be confidently predicted, however, on the basis of many years of observation, that the experience will be the same as that of Minnesota, i. e., the infections occurring in the city of New York will be negligible in number, and always will be far less than are the imported cases—imported from other parts of the United States or from abroad.

To illustrate the probable sources of infection of lepers discovered in New York the following tabulation covers cases that have come to the attention of the city health authorities during a period of approximately 2 years ended April 30, 1941.

Initials of patient	*Place where disease probably was acquired*
D. E.	British West Indies.
S. M.	Dutch Guiana.
J. W.	California.
J. J.	Central and South America and Texas.
C. S.	Chile, Panama, Cuba, Brazil (South).
C. G.	Puerto Rico.
F. G. (sailor)	Sicily, Algiers, Russia.
L. W.	China.
W. S.	China.
H. B.	Hawaii, Philippines.
D. M.	Hawaii.
F. M.	Philippine Islands.
M. C. (case reported above).	California or New York.

Less complete data covering a longer period of time, and not including any cases referred to above, are shown in the following list. Probable sources of infection of lepers reported by the New York City Health Department, not included in the preceding table, are shown:

Birthplace	*Number of cases*
British West Indies	13
Virgin Islands	3
Russia	3
China	2
Cuba	2
Philippine Islands	2
Puerto Rico	2
Dutch Guiana	2
Italy	2

Birthplace	*Number of cases*
Florida	2
Georgia (Savannah)	1
Massachusetts (visited or lived in South America)	1
Texas	1
British Guiana	1
Canal Zone	1
Colombia, S. A	1
Cyprus	1
Germany (exposed in Peru, Brazil, and Argentina)	1
Greece	1
Haiti	1
Malta	1
Morocco	1
Poland	1
Santo Domingo	1
Sicily	1
Spain	1
Turkey	1
West Indies	1

The suggestion has been made that, possibly, persons acquiring the disease in New York do not come to the attention of the health authorities. This criticism appears to lose force when it is recalled that a number of lepers do come to the attention of the authorities annually and that upon investigation they can be traced to infection elsewhere with the exception of the two cases already mentioned. It would seem that cases infected locally would be as likely to be reported as those infected elsewhere.

By way of contrast let us turn our attention to cases of leprosy reported to the health authorities in the Gulf Coast States, especially Louisiana, Texas, and Florida. The lepers from Louisiana (259 cases in the period 1913 to 1937) and from Florida (65 in the period 1911 to 1937) are nearly all infected within those States, while a few contracted the disease in other well known foci. In Texas (159 cases from 1920 to 1937) the situation is a little different. While many persons are infected in the State, a number (at least 26 in the period mentioned) acquire the disease in the Mexican Republic. The figure given here for Texas is believed to be more of an understatement of the true number than that for the other States.

It is rather generally believed that there is a focus of leprosy in the central northwestern States, particularly Minnesota. The experience in Minnesota, the Dakotas, Iowa, and Wisconsin has been most instructive and interesting. During the last half of the nineteenth century there was a large immigration to this area from the Scandinavian countries, especially from Norway where at that time leprosy was rather prevalent. Among the immigrants were a number of persons with leprosy, and some persons who developed the disease soon

after arrival in America. The total number of lepers among the immigrants, including those in whom the disease was in the incubation stage, is uncertain but probably was between 100 and 200. More of the immigrants (and the lepers) settled in Minnesota than in any other State, indeed, probably more than in all other States mentioned. So far as is known, with the single possible exception of a case developing in an American-born person in Wisconsin, none of the States other than Minnesota furnished any indigenous cases. The number of lepers and persons in the incubation stage coming to Minnesota is not known but there is evidence that there were not less than 50 and probably not more than double that number. A rather careful study of the records indicates that the number was at least 72. So far as the available data warrant any opinion, one may say that the health authorities of Minnesota decided they would await developments and they refused to become alarmed over leprosy. Therefore, little or no attempt was made to control the spread of the disease in that State at that time.

Dr. Gronvald, the scientific advisor on leprosy for the Minnesota State Board of Health, a man thoroughly familiar with this disease from his experience abroad, wrote in 1894 in respect to a leper about whom he had been consulted: "Let the man live in peace, impressing on him of course the old precept of cleanliness first and cleanliness last—own bed and bedclothes, own table utensils, and, if possible, own room."

Bracken *(6)*, State health officer of Minnesota, reported in 1898 that among 78 children born to lepers no case of leprosy had developed among the children, nor had any leper infected his companion in wedlock.

For the first 40 or 50 years after the migration of lepers into the northwestern States, no infections locally acquired were reported; then over a period of 21 years (1895–1916) seven cases developed among American-born persons, chiefly in family contacts of foreign-born lepers; the first appeared in 1895, the last in 1916. It is a question whether any leprosy developed in the second generation of American birth. One case is recorded (1921), that of the daughter of an American-born leprous mother. The evidence with respect to this child is conflicting and there is no agreement as to whether she had leprosy; if she had, recovery was prompt and complete. To sum up the Minnesota experience it may be said that not less than 70 imported lepers gave rise to 7 contact cases and that these 7 gave rise to a doubtful one.

At least 13 lepers of Norwegian origin lived in Iowa between 1863 and 1899 but no case is known in which the disease was acquired in that State. Interest in leprosy in Iowa originated in 1883 when the Swedish health authorities sought to ascertain the status of this disease in the United States by communicating with the State Board

of Health of Iowa and the then National Board of Health at Washington, D. C.

The survey of the central northwestern States reveals that the whole tendency was towards automatic suppression with the result that leprosy has disappeared in that region.

The experience in California has been very similar to that in Minnesota and neighboring States but has attracted less attention than that of Minnesota. The lepers of California have come mainly from Mexico, the islands of the Pacific, and from China. The records of the State department of health show 475 cases of leprosy from 1913 to 1940. Deaths from 1906 to 1940 totaled 90 for the period, as shown by the following table:

Year	Cases	Deaths	Year	Cases	Deaths
1906 [1]		1	1925	24	4
1907 [1]		4	1926	18	1
1908 [1]		5	1927	23	5
1909 [1]		3	1928	19	1
1910 [1]		2	1929	23	2
1911 [1]		4	1930	19	1
1912 [1]		3	1931	19	1
1913	11		1932	20	2
1914	7	3	1933	11	2
1915	12	3	1934	14	2
1916	13		1935	16	
1917	21	6	1936	9	
1918	20	5	1937	13	2
1919	23	4	1938	15	
1920	20	4	1939	9	
1921	27	6	1940	6	1
1922	25	4			
1923	19	8	Total	475	90
1924	19	1			

[1] Morbidity records since 1913 only.

Of the 475 cases shown in this tabulation, not more than 14 probably were infected in the State. It seems clear from the histories in these cases that the disease could not have been acquired elsewhere. This figure, 14, includes two young leprous children of a Japanese leprous mother, discovered in 1941. The mother was reported to have been born in New Zealand and the source of her infection could not be ascertained. Also among the 14 was an American-born adult male, the place of whose infection could not be ascertained, but which probably was central California. Some years after this man's death from leprosy, one of his sons developed the disease. This is the only case with which the author is acquainted in which leprosy appeared in the second generation of a California-born person.

It will be noted that the number of cases and of deaths has declined in recent years. This probably is due to two factors: (1) examination of immigrants and exclusion of lepers and (2) the fact that from 1922 to 1938, inclusive, 165 California lepers have been committed to the United States Leprosarium at Carville, La., where their deaths would be recorded rather than in California. Data were available showing the birthplace of lepers reported in this State from 1920 to 1934. It is recognized that the place of birth is not necessarily the place in which the disease was acquired, but probably in the great majority of cases the two coincide.

Birthplace of lepers found in California

Place of birth	1920	1921	1922	1923	1924	1925	1926	1927	1928	1929	1930	1931	1932	1933	1934	Total
Mexico	13	15	12	8	9	16	11	14	12	14	10	11	7	3	10	165
Philippines		2	1	1	1	2	2		3	3	4	4	8	6		37
China	2	2	3	6	2	2	1		1	2			1	1		22
California					1	2	2	1	1		1	2	1			11
Hawaii				1		1	2	1		1	1	2				9
Spain					2	1			1							4
Japan												1			2	3
Puerto Rico										2					1	3
India		1					1									2
Tahiti								1			1					2
Greece		1											1			2
Ohio				1										1		2
Arizona	1														1	2
Syria							1								1	2
Korea	1															1
Cape Verde Islands		1														1
Portugal		1														1
Kansas			1													1
Alabama			1													1
Russia				1												1
England					1											1
Malta					1											1
Utah									1							1
Texas								1								1
Minnesota								1								1
Florida								1								1
Louisiana									1							1
West Indies													1			1
Montenegro													1			1
Poland															1	1
Hungary															1	1

There is a small area on the South Atlantic Coast where leprosy has occurred; somewhat accurate data are available for Charleston, S. C., only. There are records of deaths in Charleston almost continuously since 1821. Nine deaths from leprosy were reported from 1824–48 (25 years), while in the 25 years ended with 1937 there have been 10 deaths from the disease. The population of Charleston was approximately 30,000 in 1830 while it was approximately 68,000 in 1920. Obviously the tendency is toward a reduction of the number of cases in proportion to the population in this area. So far as the records show, all of the cases in Charleston have been infected locally with the exception of possibly two patients, one born in Scandinavia, and one born in Germany. So far as can be determined at this late date there never was any serious attempt made toward the control of leprosy in Charleston and it seems fair to assume that whatever reduction has occurred in the number of lepers has been due to the operation of natural causes.

The health records of Savannah, Ga., are less satisfactory but there have been three cases recognized in the period 1912–26; so far as one can judge, all of these persons were infected in Savannah or vicinity. The only other case certainly chargeable to Georgia was reported in 1924. The patient was a colored boy, 15 years old, who always had lived in a small community about 30 miles from Savannah. The source of his infection was not established, but a layman's description of a condition from which the patient's grandmother had died many years before was suggestive of leprosy.

When infections occur in Minnesota, California, or in the Gulf coast area, often direct contact with a leper can be established, or it can reasonably be assumed that there has been an unrecognized contact.

There is another small group of cases that is perhaps the most puzzling for the epidemiologist. These develop in areas in which the disease is not endemic and even the most searching investigation that has been practicable has failed to show any probable source of the infection. The author is acquainted with one case that must have been infected either in Maryland or Virginia, one in Missouri or Illinois, one in Pennsylvania, and two either in Illinois or Kansas. The latter cases deserve a little further explanation. A young woman was found to be suffering from leprosy while living in Chicago after having spent the early part of her life in Kansas. Her parents were from Virginia. A year or more after the daughter's infection was recognized her mother, who had been with the daughter, also developed leprosy. The most careful investigation of this case by the writer and others has failed to reveal a possible source of infection of the daughter, but the mother presumably was infected from her daughter.

SUMMARY

Leprosy has been introduced into different areas of the United States with very different consequences. In Louisiana, Florida, and Texas the presence of imported cases has resulted in the establishment of foci in which the disease shows a strong tendency to perpetuate itself, while in the central northwestern States and in California the reverse prevails and the disease has shown little tendency to become established. Elsewhere in the United States leprosy transmission occurs so rarely that it is negligible from the public health point of view.

The data presented refer to experiences with leprosy up to the present time. But in an age in which great changes, both social and economic, are occurring, no one can predict what unexpected influence these may have on the occurrence of leprosy as well as other diseases.

REFERENCES

(1) MacLeod, J. M. H.: Contact cases of leprosy in the British Isles. Brit. Med. J., 1: 107–108 (1925).
(2) Discussion on leprosy in England. Leprosy news and notes. Internat. J. Leprosy, 6: 561–563 (1938).
(3) Flandin, C., and Ragu, J.: Origine, mode de contagion, durée d'incubation de la lèpre dans 95 cas dont 6 contractés dans la région parisienne. Bull. acad. med., 117: 337–343 (1937).
(4) Cochrane, R. G.: Leprosy in Europe, the Middle and Near East, and Africa. World Dominion Press, London, 1928.
(5) Brocchieri, G.: Su un caso di lepra autoctona in provincia di Roma. Policlinico (Sez. Prat.), 45: 153–158 (1938).
(6) Bracken, H. M.: Leprosy in Minnesota. Philadelphia Med. J., 2: 1309–1311 (1898).

MARINE AMBULANCE SERVICE AT MIAMI, FLA.

Medical officers in the United States Public Health Service are charged with the duty of furnishing medical advice by radio to ships at sea. Certain radio stations on the Atlantic, Pacific, Gulf, and Great Lakes coasts, as well as in Hawaii, have been designated for convenience in radio communication.

The Public Health Service at Miami has received many requests for medical advice from ships that pass approximately 2 miles off shore. Radio consultations are not always satisfactory, and frequently the ship's captain requests that a doctor be sent to determine whether the patient could be treated on shipboard or should be removed to a hospital on shore.

On August 15, 1939, Surgeon General Thomas Parran inaugurated a marine ambulance service by authorizing the use of a quarantine vessel to make contact with ships passing off Miami and furnished medical supplies and equipment. Among items of equipment used is a specially constructed stretcher designed so that the patient being transferred from a large vessel to the small quarantine boat could be securely fastened within and which would safely float the patient if he should be dropped during the transfer.

A total of 113 marine ambulance runs have been made from Miami to the adjacent sea lane. Twenty-four seamen were found in such condition that it was feasible to furnish treatment and permit the men to continue with their ships. The condition of 89 seamen warranted immediate hospitalization. The delivery of these men to the hospital was expedited by sending a wireless request from the vessel to have an ambulance waiting at the dock. Delay to the vessel is minimized as the transfer of a patient is usually accomplished while his vessel proceeds on her course at reduced speed.

The patients removed included serious accident cases due to falls, crushing injuries, and extensive burns. The more common surgical emergencies such as appendicitis, gall bladder and kidney conditions, and intestinal obstruction have been encountered. Such medical emergencies as pneumonia and cardiac and mental diseases have been given prompt attention through the operation of the marine ambulance service.

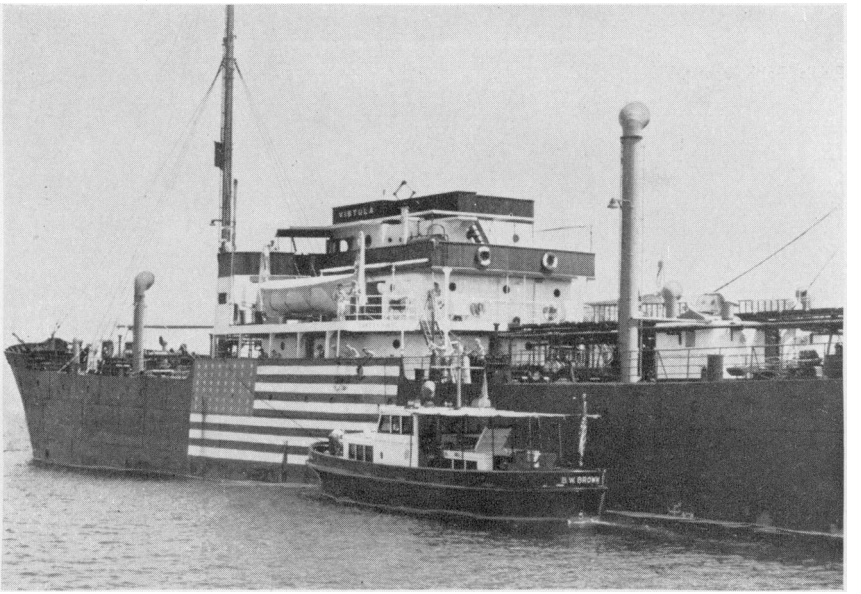

FIGURE 1.—Marine ambulance boat alongside a tank ship off Miami.

FIGURE 2.—Illustrating the ease of handling a patient when "secured" in the stretcher. With the canvas flap laced over the arms and up to the chin, a mentally upset patient can be transported without difficulty.

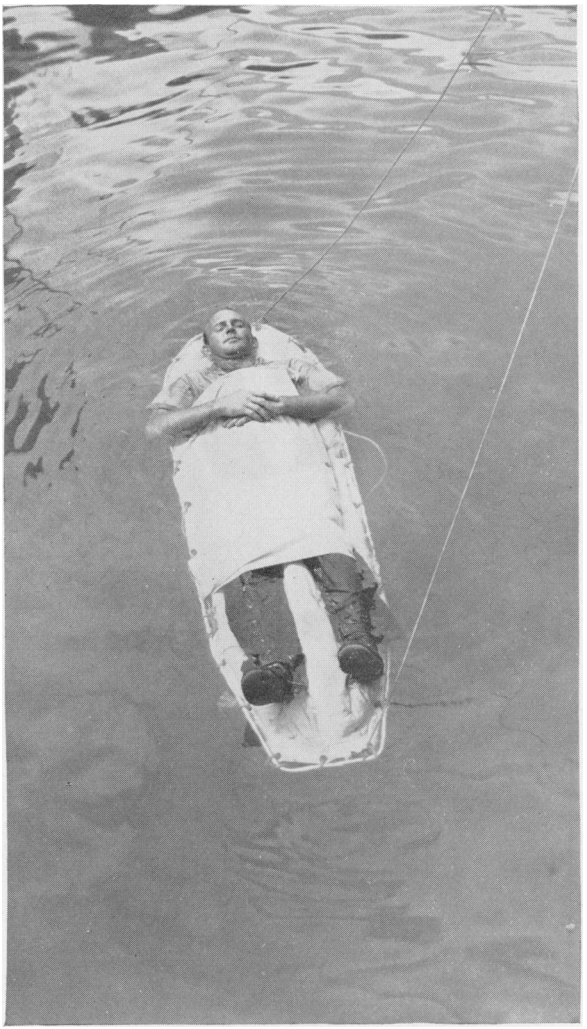

FIGURE 3.—The stretcher, fitted with a kapok-filled pad, to insure floating in the event of accident in transferring from ship to ship.

LOCATION AND MOVEMENT OF PHYSICIANS, 1923 AND 1938—EFFECT OF LOCAL FACTORS UPON LOCATION[1]

By JOSEPH W. MOUNTIN, *Assistant Surgeon General,* ELLIOTT H. PENNELL, *Statistician,* and VIRGINIA NICOLAY, *United States Public Health Service*

In earlier articles [2] [3] the authors of this series expressed quantitatively the uneven distribution of physicians. More specifically, there was revealed a paucity of provisions for professional services in States where the per capita income was low and where a large fraction of the population resided in rural areas. This situation was more serious in 1938 than in 1923, but seemed to be mitigated to a slight extent in those depressed areas which were favored by the presence of hospitals—especially those offering opportunities for intern training.

With the large and rapidly accelerating withdrawal of physicians from private practice to meet the exigencies caused by the war effort, data depicting the distribution of physicians assume particular significance in defining areas in which civilian health and morale would be least affected by further drafts. It may also be anticipated that the factors which determined the location and subsequent migration of physicians after the first World War will be operative in the period of readjustment following the current crisis unless some efforts are made to understand and direct the forces which have precipitated these distributional trends.

The large reservoir of information abstracted from data published in medical directories [4] makes possible analyses beyond those previously presented. To facilitate the tabulation of this material for local areas, county summary totals were prepared from the information concerning individual physicians and from other sources. These totals provide data which comprise the census population counts for counties in 1920, 1930, and 1940, the number of physicians located therein in 1923 and 1938, the distribution of physicians in 1923 and 1938 by 5-year age intervals,[5] the number of hospital beds in 1940,[6] and the total effective buying income in 1940.[7] Aggregate totals were then assembled for groups of counties classified on the basis of per capita income, metropolitan character, largest urban place and number of hospital beds in 1940, population trends from 1920 to 1930

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[1] Assistance in the preparation of these materials was furnished by the personnel of Work Projects Administration Official Project No. 65-2-23-356.

[2] Mountin, Joseph W., Pennell, Elliott H., and Nicolay, Virginia: Location and movement of physicians, 1923 and 1938—General observations. Pub. Health Rep., 57: 1363-1375 (1942).

[3] Mountin, Joseph W., Pennell, Elliott H., and Nicolay, Virginia: Location and movement of physicians, 1923 and 1938—Turnover as a factor affecting State totals. Pub. Health Rep., 57: 1752-1761 (1942).

[4] American Medical Directory, eighth, ninth, tenth, eleventh, twelfth, thirteenth, fourteenth, and fifteenth editions, 1923, 1925, 1927, 1929, 1931, 1934, 1936, and 1938. American Medical Association, Chicago.

[5] Physician totals were tabulated from information abstracted for individual physicians.

[6] Hospitals and other institutional facilities and services: 1939. U. S. Department of Commerce, Vital Statistics—Special Reports, Vol. 13.

[7] Sales management survey of buying power. Sales Management, Vol. 48, No. 8 April 10, 1941.

and from 1930 to 1940, and the number of physicians at the mid-period (1931).

It is recognized that scarcely any particular class of local political subdivision may be considered as representing a self-sufficient area in the provision of service such as medical care. On the other hand, a county usually encompasses a sufficiently large population group to permit the computation of reliable indices, and at the same time to provide community data which are essentially local in scope. Furthermore, individual peculiarities of aberrant counties tend to be merged in aggregate figures of the numerous units comprising broad categories. Tabulations from the county data reveal in dramatic fashion the

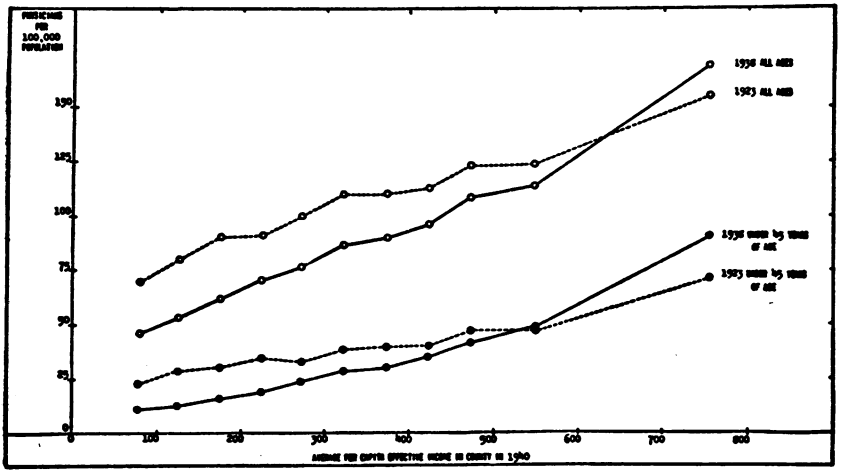

FIGURE 1.—Physician-population ratios in counties with different per capita incomes, 1938 and 1923.

influence of factors such as wealth, urban character, and medical facilities upon the ratio of physicians to population in local areas, and the trend in these ratios during the study period.

Among the several factors investigated, community wealth was found to be of paramount importance in determining the availability of physicians. The measure of wealth for classifying counties was obtained by dividing the 1940 effective buying income[8] in the county by the population. In 1938 the combined population in counties of different income classifications (table 1) varied from 1,244,000 in the group of poor counties with average per capita incomes of less than $100 to nearly 65,000,000 in counties where the per capita incomes averaged $600 or more. The number of physicians varied from less than 600 in the former to nearly 111,000 in the latter group of counties. When related to population (fig. 1) there was a marked progression

[8] Effective buying income, as presented in "Sales Management Survey of Buying Income (7)," is based upon money income from all sources plus an estimated nonmoney income of farmers and small-town residents within a State. The per capita figures referred to throughout this report have been obtained by dividing the county income totals by the 1940 U. S. Census population count.

from 46 physicians per 100,000 population in the poorest counties to 171 in counties with average incomes of $600 or more; physicians under 45 years of age represented 11 per 100,000 in the former and 86 per 100,000 in the latter group. Throughout the range of income, provisions in medical personnel increased in conformity with elevation of income. In counties with the highest per capita incomes the physician-population ratio was nearly four times as great as in the poorest counties; this ratio for physicians under 45 years of age was eight times as great. More than one-half of all physicians in the wealthy counties were under 45 years of age, but less than one-fourth were in this category in the poorest counties.

Comparable data for 1923 reveal a similar pattern with the exception that the contrast is less pronounced. The counties classified as most wealthy realized twice as many physicians per unit of population as did the poorest group of counties, while physicians under 45 years of age per unit of population were only slightly more than three times as numerous in the wealthy as in the poor counties. Thus it is apparent that the growing tendency for physicians to select wealthy and populous counties for the practice of medicine becomes more pronounced with each succeeding year.

TABLE 1.—*Distribution of population, total physicians, and physicians under 45 years of age in the continental United States in 1938 and in 1923 by income class of county*

Per capita income class of county	Average per capita effective buying income 1940	Estimated population (add 000)		Total physicians				Physicians under 45 years of age			
				Number		Per 100,000 population		Number		Per 100,000 population	
		1938	1923	1938	1923	1938	1923	1938	1923	1938	1923
All counties	563.3	130,104	111,526	169,594	146,344	130.4	131.2	76,763	61,101	59.0	54.8
Less than $100	80.1	1,244	1,149	567	797	45.6	69.4	135	261	10.9	22.7
$100–$149	126.9	3,960	3,735	2,127	2,984	53.7	79.9	487	1,048	12.3	28.1
$150–$199	175.8	6,216	5,769	3,826	5,230	61.6	90.7	968	1,739	15.6	30.1
$200–$249	225.7	6,447	6,067	4,525	5,611	70.2	92.5	1,238	2,080	19.2	34.3
$250–$299	273.9	5,584	5,081	4,266	5,116	76.4	100.7	1,338	1,673	24.0	32.9
$300–$349	323.8	5,408	5,080	4,697	5,658	86.9	111.4	1,545	1,975	28.6	38.9
$350–$399	374.6	6,080	5,724	5,511	6,367	90.6	111.2	1,851	2,294	30.4	40.1
$400–$449	426.0	6,078	5,563	5,902	6,361	97.1	114.3	2,125	2,258	35.0	40.6
$450–$499	474.9	7,677	6,695	8,395	8,337	109.4	124.5	3,225	3,199	42.0	47.8
$500–$599	552.1	16,667	13,759	19,187	17,144	115.1	124.6	8,089	6,497	48.5	47.2
$600 or more	758.5	64,743	52,904	110,591	82,739	170.8	156.4	55,762	38,077	86.1	72.0

As a matter of course, to maintain a fixed physician-population ratio over a period of time, the variation in the population counts must be balanced perforce by a corresponding proportionate variation in the number of physicians. Increased ratios may result in areas where the proportionate increase in physicians is greater than that for the population as a whole, or when decreased physician totals are associated with a population decline of greater proportions. The question naturally arises as to the degree of parity which has obtained between

population trends and physician migration trends during the period under study.

In the aggregate, the physician-population ratios for counties showing population increases in both intercensal periods 1920 to 1930 and 1930 to 1940 were high at the initial and terminal years of the study period and a moderate increase appeared during the interval. This was true of both total physicians and new registrants. On the other hand, decreased population totals in the intercensal periods were reflected in low and sharply declining ratios. In counties showing population gains in 1920 to 1930 followed by declines in the later decade, the ratios presented essentially the same picture as did those for counties with consistently increasing population totals throughout, whereas ratios for counties showing early population declines followed by increases in the last intercensal period pursued the pattern established by counties with consistently declining populations.

Further classification on the basis of income indicates that the high and increasing ratios in counties with expanding populations as revealed in the consolidated figures reflect the dominant influence of wealthy counties. In counties with average per capita incomes of less than $300, the ratios were low, regardless of population trend, and showed pronounced declines over the 15-year period covered by the study. At this income level, the ratios were slightly more favorable in counties where population declined in both intercensal periods than in counties where there was a consistently increasing population trend. In contrast, the ratios were relatively high in counties with average per capita incomes of $600 or more. Consistent population increase in counties of this classification indicated ratios markedly in excess of those for counties with population declines. Furthermore, counties in the former group realized considerable expansion in ratios from 1923 to 1938. Where population trends were consistently downward in wealthy counties, the ratios reached parity with those for all counties except wealthy ones with population increases, and these ratios were maintained at essentially the same level to the end of the study period.

These disclosures suggest that consistent population increase in areas reflects factors which serve to attract physicians, but only when the income in these areas is high. In counties of this class, the ratios both for total physicians and for those under 45 years of age were large, and the recruitment of physicians exceeded even the population increases so that expanded ratios occurred. Declining population totals in wealthy areas resulted in diminishing physician totals in almost the same degree so that the ratios varied only slightly during the study interval. In poorer counties, on the other hand, the provisions for care at a given time were not greatly affected by the population trends, but regardless of population trends the ratios declined in a consistent manner.

The lack of facilities for professional care of ill persons in counties may simply mean that those in nearby areas serve satisfactorily the residents of a county. However, generous facilities within a county may have developed because patients in large numbers are attracted thereto from adjoining or even from distant areas. The definition of counties, or groups of counties which represent both facilities and the population served thereby, is not strictly possible inasmuch as service areas are not always conformable to the boundaries of established political units. Exception may occur where special services are provided at public expense, and where residence within the areas has been established as a prerequisite in eligibility for care. To obviate at least in part the above difficulties, a special analysis has been prepared whereby counties are described in terms of their location with respect to metropolitan areas as defined by the United States Census Bureau in 1940. The classification of the Census Bureau [9] brings together people of urban centers of 50,000 or more inhabitants plus those residing in densely inhabited adjacent areas as single population units. For the purpose of this study this definition of metropolitan area has been extended to include all parts of the affected counties; all other counties are classified as nonmetropolitan. This classification of counties groups all populous and relatively wealthy urban counties plus a considerable number of contiguous counties which may be small either from the standpoint of area or population; the group of nonmetropolitan counties includes those essentially rural in character. The chief distinction between the two groups of counties is that residents of the former live within or at locations more or less accessible to large population centers, whereas residents of nonmetropolitan counties are removed by one or more counties from such concentrations of population.

The data in figure 2 reveal increasingly unequal provisions for physicians' services in the two types of counties during the 15-year period covered in the study. In 1923, there were 152 physicians per 100,000 population in the metropolitan counties as contrasted with 110 in the nonmetropolitan group. By 1938 the ratios in these two identical groups of counties had increased to 164 where the classification was metropolitan and had declined to 92 for the more rural group. It is thus apparent that the disparity in facilities for care, while large in 1923, was greatly expanded by the end of the period.

Wealth played an important part in the determination of facilities in both metropolitan and nonmetropolitan areas. In 1923 the physician-population ratio in the wealthiest metropolitan counties was more than twice as great as in the poorest group whereas it was only about 50 percent greater in nonmetropolitan counties. By

[9] Population and housing units in the metropolitan districts of the United States: 1940. Series PH-1: Summary. U. S. Department of Commerce, Bureau of the Census.

1938 the ratios had increased from 159 to 176 in wealthy metropolitan counties and had declined from 74 to 54 in the poor counties. Wealthy nonmetropolitan counties, on the other hand, nearly main-

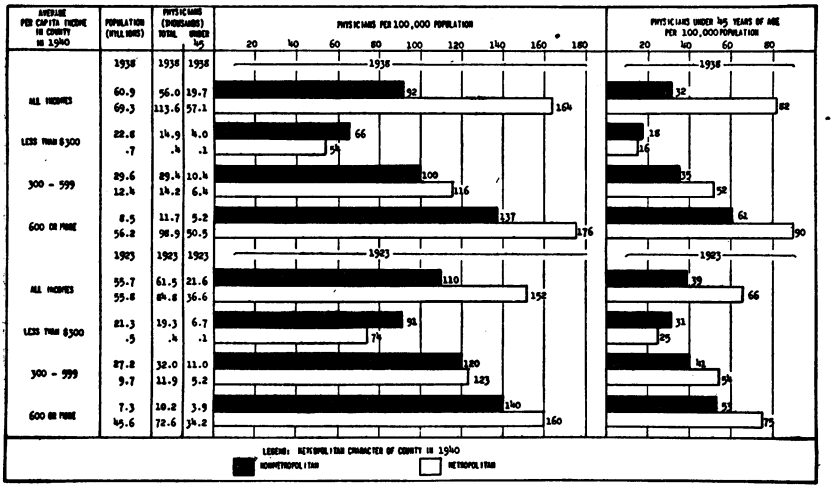

FIGURE 2.—Physician-population ratios in counties with different per capita incomes and of different metropolitan character, 1938 and 1923.

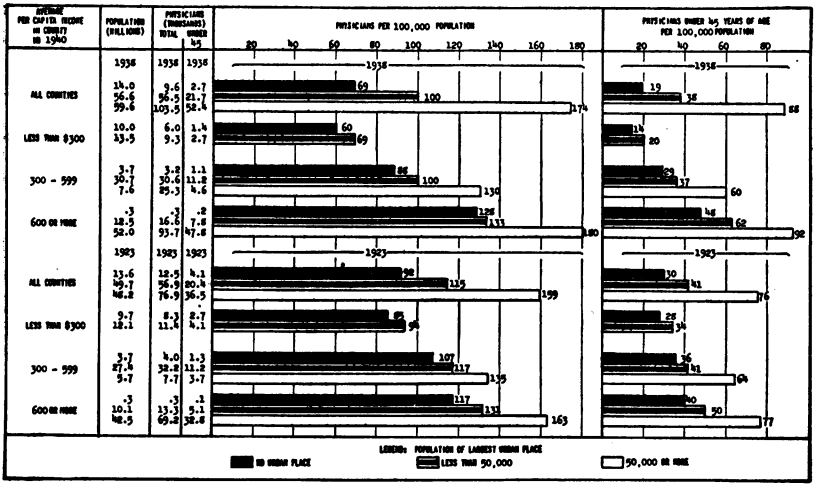

FIGURE 3.—Physician-population ratios in counties with different per capita incomes and of different urban character, 1938 and 1923.

tained the same provisions for care throughout the period as is indicated by the slight decline from 140 in 1923 to 137 in 1938. Poor counties in this group, while showing ratios which declined from 91 to 66, were able to maintain their facilities to about the same extent as did poor metropolitan counties. It should be noted that low rates in poor metropolitan counties do not present the same problem

as in the other group because these counties are adjacent to wealthy counties where large numbers of physicians reside.

A high degree of association between urban character of a county and the number of physicians located therein per unit of population is revealed in figure 3. In 1923 there were 92 physicians per 100,000 persons residing in strictly rural counties (no incorporated place of 2,500 or more inhabitants located therein). The presence of urban places of less than 50,000 inhabitants in counties was reflected by an average ratio of 115, and counties with cities of 50,000 or more people realized 159 physicians per 100,000 population. The corresponding ratios for 1938 were 69, 100, and 174. Only in the most urban counties was there evidence of more generous provisions for care in 1938 than in 1923.

Variations in urban character of counties resulted in important differences in the number of physicians at the three income levels studied. In counties reporting average per capita incomes of less than $300, the ratio in 1923 for strictly rural counties was 85; poor counties having one or more urban places of less than 50,000 inhabitants realized a ratio of 94. No counties with cities of 50,000 or more fell into this income group. The ratio for the two classes of poor counties showed large declines over the period. Essentially the same tendencies were displayed by counties with average per capita incomes from $300 to $599 except that the ratios were higher and the declines were less pronounced over the study period. Among the wealthiest counties, however, the ratios actually increased during the study period. For wealthy counties with cities of 50,000 or more inhabitants the ratio increased from 163 in 1923 to 180 in 1938, whereas the ratio in wealthy rural counties changed from 117 to 128. The ratio for young physicians (under 45 years of age) followed the same pattern as that established by all physicians.

In modern medical practice the hospital is an invaluable adjunct. Such an institution affords means for the accurate diagnosis and proper treatment of illness, and in many cases is essential for the care and recovery of patients. Absence of hospital facilities in a county may not always indicate a lack of accommodations accessible to those needing care, because one or more hospitals may exist in nearby areas. Nevertheless, relatively large numbers of hospital beds in counties are reflected in generous physician-population ratios. For the country as a whole there were in 1938 only 67 physicians per 100,000 population in counties without general or allied special hospitals as contrasted with 157 for counties in which there were 250 beds or more.

Figure 4 reveals a close association between hospital facilities and physician-population ratios. In wealthy counties with large numbers of hospital beds (250 or more) the ratios for both 1923 and 1938 exceeded by approximately 50 percent those for the corresponding group

of counties without hospitals. In counties of the lowest income classification such differences persisted but were not as great as in wealthy counties. This would indicate that, regardless of the income class of the county, the presence of large numbers of hospital beds reflected more attractive locations for physicians than did the limitation or absence of these facilities. This was especially true of physicians under 45 years of age. In all income classes the ratios of young physicians to population were twice as great where hospital beds were numerous as in counties without such facilities. The important contribution made by accessory facilities for medical care represented by large numbers of hospital beds upon the size of physician-population

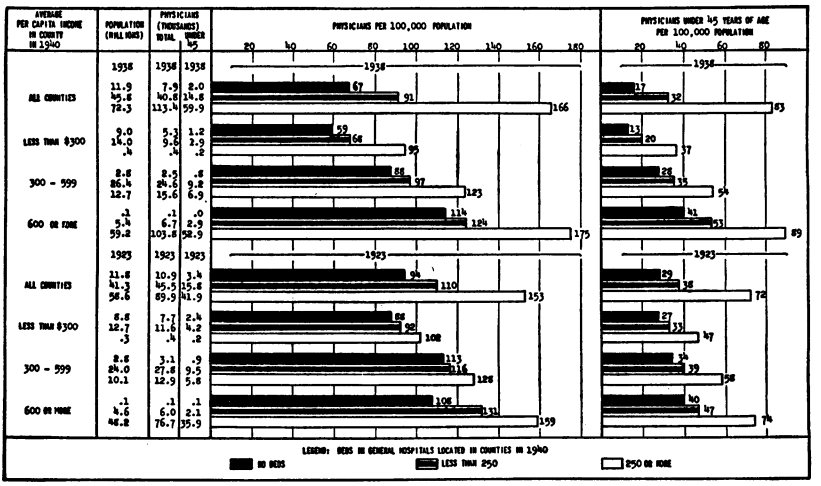

FIGURE 4.—Physician-population ratios in counties with different per capita incomes and with different facilities in general hospitals, 1938 and 1923.

ratios suggests that such facilities alone afford attraction for establishing medical practice apart from other factors such as wealth, population expansion, and urban character of counties.

Another important adjunct to the practice of modern medicine is the accessibility of professional associates for consultation and exchange of professional ideas. The opportunities for consultation and collaboration in the care of obscure conditions become greater as the number of physicians in an area is increased. Where the provisions for medical care in a county are limited to a small number of physicians the opportunities for interchange of professional ideas are fewer than in counties where the number of physicians is large. Analysis reveals that in counties where physicians in 1931 numbered less than 5 per county, there were only 69 physicians per 100,000 population in 1923 and the ratio had declined by 1938 to 57. Where there were 100 or more physicians per county in 1931 the ratio was 156 in 1923 and had

been elevated to 170 in 1938. At each income level large numbers of physicians were reflected in ratios markedly in excess of those for counties with small numbers of physicians. In 1938 the ratio of 44 physicians per 100,000 population in counties with few physicians and with average per capita incomes below $300 was only one-fourth as great as the 178 in wealthy counties with large physician totals. The number of physicians under 45 years of age showed even greater differences, the ratios varying from 12 in the poorest counties with few physicians to 91 in wealthy counties with 100 physicians or more.

SUMMARY

The data presented reveal much greater concentrations of physicians in some counties than in others. Throughout the comparison, wealth is a dominant factor in the maintenance of high physician-population ratios. Where wealth is high the ratios in 1923 were elevated and tended to remain fixed or even to expand by the termination of the 15-year period. In poor counties, on the other hand, the ratios in the initial year were low, and as a rule were seriously reduced by 1938. This phenomenon was revealed in an even more dramatic fashion for physicians under 45 years of age.

While metropolitan and urban character of counties, facilities as revealed in a count of total beds in general hospitals, and physicians in a county are closely allied with wealth, these factors in isolation also tend to induce larger physician-population ratios at all income levels.

Where income is high and the classification on the basis of other factors used to describe features making counties attractive to physicians is also high, the physician-population ratios are several times as great as in those counties falling lowest on the comparison scales. The number of physicians under 45 years of age per 100,000 population in these most favored classifications generally exceed the ratios based upon total physicians in the poorest counties. It is apparent that the great reservoir of physicians is in the large urban centers. If the ratio of physicians to population in the nonmetropolitan counties were taken as a base, one would find a marked excess of physicians in the larger urban centers and the counties immediately adjacent. Throughout the study period, which embraces a complete economic cycle, there has been a more pronounced tendency for physicians than for the general population to concentrate in urban areas.

DEATHS DURING WEEK ENDED DECEMBER 5, 1942

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

	Week ended Dec. 5, 1942	Corresponding week 1941
Data from 87 large cities of the United States:		
Total deaths	9,628	8,487
Average for 3 prior years	8,513	
Total deaths, first 48 weeks of year	401,720	398,807
Deaths per 1,000 population, first 48 weeks of year, annual rate	11.7	11.6
Deaths under 1 year of age	675	528
Average for 3 prior years	506	
Deaths under 1 year of age, first 48 weeks of year	27,671	25,201
Data from industrial insurance companies:		
Policies in force	65,292,593	64,696,204
Number of death claims	12,811	11,280
Death claims per 1,000 policies in force, annual rate	10.2	9.1
Death claims per 1,000 policies, first 48 weeks of year, annual rate	9.1	9.2

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

REPORTS FROM STATES FOR WEEK ENDED DECEMBER 12, 1942

Summary

The number of reported cases of influenza increased from 1,928 for the preceding week to 2,604 for the current week. The 5-year median (1937–41) is 2,995 cases. The current incidence, as well as the cumulative total to date, is below that for any previous year since 1938. While the number of reported cases for the country as a whole is below the median expectancy, the disease has been unusually prevalent in a few States in the South Atlantic and West South Central areas. Of the current total, Texas reported 732 cases, South Carolina 517, Virginia 371, Oklahoma 185, and Georgia 116—a total of 1,921 cases, or 74 percent, in these 5 States.

The incidence of meningococcic meningitis continues above the median expectancy, especially in some of the eastern States and the Pacific Coast area. The number of cases increased from 88 last week to 103 for the current week. The following named States reported the largest numbers: New York 11, Pennsylvania, Virginia, and Oregon 10 each, and Maine, Massachusetts, and California 6 each. No other State reported more than 5 cases. A total of 3,387 cases has been reported to date, which is the largest number reported for this period since 1937, when 5,146 cases had been reported.

The incidence of poliomyelitis declined from 79 to 66 cases, of which 22 occurred in Texas, 13 in California, and 5 in Pennsylvania. No other State reported more than 3 cases.

A total of 90 cases of endemic typhus fever was reported. To date, 3,509 cases have been reported—a larger number than for any previous entire year (2,998 in 1939 and 2,787 in 1941).

The death rate for the current week in 88 large cities in the United States is 13.0 per 1,000 population, as compared with 12.8 last week (excluding the Boston fire mortality), and a 3-year (1939–41) average of 11.9. The lack of accurate current urban population estimates and possible changes in the age distribution of these populations are as yet undetermined factors in these current death rates.

Telegraphic morbidity reports from State health officers for the week ended December 12, 1942, and comparison with corresponding week of 1941 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median 1937–41	Week ended—		Median 1937–41	Week ended—		Median 1937–41	Week ended—		Median 1937–41
	Dec. 12, 1942	Dec. 13, 1941		Dec. 12, 1942	Dec. 13, 1941		Dec. 12, 1942	Dec. 13, 1941		Dec. 12, 1942	Dec. 13, 1941	
NEW ENG.												
Maine	1	0	1	-------	1	1	7	263	42	6	0	0
New Hampshire	1	2	1	-------	1	-------	58	4	4	0	0	0
Vermont	0	3	0	-------	-------	-------	109	0	21	0	0	0
Massachusetts	2	7	3	-------	-------	-------	539	143	192	6	1	2
Rhode Island	0	4	0	1	-------	-------	20	19	2	3	0	0
Connecticut	0	0	1	2	5	5	235	74	46	4	2	1
MID. ATL.												
New York	22	20	20	[1] 16	[1] 8	[1] 13	430	214	509	11	2	2
New Jersey	3	6	12	10	13	9	25	17	17	5	1	1
Pennsylvania	11	8	29	5	-------	-------	747	723	723	10	3	3
E. NO. CEN.												
Ohio	11	20	44	17	13	14	36	53	53	3	0	1
Indiana	4	3	20	7	12	12	38	33	14	0	1	1
Illinois	10	41	41	9	5	8	69	40	40	3	2	1
Michigan [2]	7	6	15	2	7	6	38	86	238	0	2	2
Wisconsin	1	0	1	34	34	34	140	129	129	2	1	0
W. NO. CEN.												
Minnesota	5	2	2	1	3	2	5	66	43	0	0	0
Iowa	4	0	3	-------	-------	3	40	44	44	0	1	0
Missouri	5	11	14	1	3	21	6	6	6	1	1	1
North Dakota	1	1	2	1	9	9	0	60	17	0	1	0
South Dakota	0	2	2	-------	-------	-------	55	3	3	1	0	0
Nebraska	4	2	2	21	-------	-------	83	4	4	0	0	0
Kansas	0	2	4	18	48	11	21	97	59	0	3	1
SO. ATL.												
Delaware	0	1	1	-------	-------	-------	1	3	3	0	0	0
Maryland [2]	2	9	9	8	9	12	4	115	14	4	1	1
Dist. of Col	2	0	2	7	1	2	3	2	1	0	0	0
Virginia	21	36	36	371	236	176	17	94	84	10	1	1
West Virginia	6	8	16	20	11	16	2	128	15	2	3	2
North Carolina	18	44	44	2	8	8	2	412	288	1	0	1
South Carolina	15	5	11	517	376	377	3	34	10	0	0	0
Georgia	5	22	14	116	80	99	3	57	8	1	1	0
Florida	7	14	12	1	11	9	0	3	3	2	1	0
E. SO. CEN.												
Kentucky	8	5	17	3	13	15	22	13	13	0	3	3
Tennessee	8	10	19	40	54	53	7	52	28	0	1	2
Alabama	12	23	23	80	98	124	2	41	35	3	0	1
Mississippi [2]	7	8	12	-------	-------	-------	-------	-------	-------	0	2	0
W. SO. CEN.												
Arkansas	15	24	18	87	150	116	22	50	23	0	0	0
Louisiana	7	4	11	13	1	12	3	4	1	0	1	1
Oklahoma	9	20	24	185	87	113	10	37	4	1	0	1
Texas	42	69	46	732	1,423	443	13	236	43	2	3	3
MOUNTAIN												
Montana	0	1	1	-------	12	12	95	28	19	1	0	0
Idaho	4	0	0	-------	-------	5	8	11	13	0	0	0
Wyoming	0	0	0	50	4	4	10	0	1	0	1	0
Colorado	10	14	11	46	25	25	12	244	44	2	0	0
New Mexico	2	0	4	-------	4	1	2	34	34	0	1	0
Arizona	1	8	8	110	110	110	4	22	3	0	0	0
Utah [2]	1	0	1	-------	9	28	658	45	45	1	0	0
Nevada	0	2	-------	-------	-------	-------	11	0	-------	0	0	-------
PACIFIC												
Washington	5	0	3	-------	12	-------	383	7	15	2	1	1
Oregon	9	3	1	16	15	23	221	68	26	10	0	0
California	23	16	20	55	84	52	66	607	134	6	2	1
Total	331	486	707	2,604	2,995	2,995	4,285	4,425	4,425	103	43	36
49 weeks	14,643	15,844	22,471	101,023	514,873	221,737	491,288	852,030	366,393	3,387	1,927	1,927

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended December 12, 1942, and comparison with corresponding week of 1941 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended--		Median 1937-41	Week ended—		Median 1937-41	Week ended—		Median 1937-41	Week ended—		Median 1937-41
	Dec. 12, 1942	Dec. 13, 1941		Dec. 12, 1942	Dec. 13, 1941		Dec. 12, 1942	Dec. 13, 1941		Dec. 12, 1942	Dec. 13, 1941	
NEW ENG.												
Maine	1	0	0	25	11	11	0	0	0	1	0	0
New Hampshire	0	1	0	9	13	10	0	0	0	1	0	0
Vermont	0	0	0	5	0	6	0	0	0	0	0	0
Massachusetts	0	1	0	293	274	169	0	0	0	2	3	3
Rhode Island	0	0	0	6	12	11	0	0	0	0	0	0
Connecticut	0	4	0	37	16	44	0	0	0	0	1	0
MID. ATL.												
New York	3	8	2	278	324	302	0	0	0	6	7	6
New Jersey	0	2	1	59	124	122	0	0	0	2	0	3
Pennsylvania	5	3	2	225	226	260	0	0	0	0	7	7
E. NO. CEN.												
Ohio	2	2	2	330	214	338	0	0	1	5	7	1
Indiana	1	3	1	55	87	138	6	0	3	0	4	3
Illinois	0	5	2	161	207	324	1	0	1	2	3	3
Michigan [2]	3	0	1	111	203	285	0	1	1	1	1	1
Wisconsin	1	0	2	178	147	149	1	1	6	1	1	1
W. NO. CEN.												
Minnesota	0	2	2	88	92	105	0	0	29	2	0	0
Iowa	2	0	1	68	55	62	0	2	10	0	1	1
Missouri	0	1	1	66	59	94	0	0	5	8	1	5
North Dakota	0	1	0	5	11	25	0	0	0	0	0	0
South Dakota	0	0	0	33	33	18	0	0	0	0	0	0
Nebraska	0	0	0	18	27	27	2	0	0	0	0	1
Kansas	3	1	1	67	70	128	0	0	0	0	1	1
SO. ATL.												
Delaware	0	1	0	5	23	12	0	0	0	1	0	0
Maryland [2]	1	0	0	54	47	51	0	0	0	2	13	5
Dist. of Col.	0	0	0	14	9	9	0	0	0	0	2	1
Virginia	0	1	1	45	65	65	0	0	0	0	11	4
West Virginia	0	0	0	47	48	52	0	0	0	1	1	5
North Carolina	0	4	1	81	78	78	0	0	0	2	4	4
South Carolina	1	1	1	21	12	12	0	0	0	0	1	1
Georgia	0	0	1	34	44	38	0	0	0	4	0	3
Florida	0	0	0	7	7	7	0	0	1	0	2	2
E. SO. CEN.												
Kentucky	0	1	1	39	77	77	0	1	0	2	1	3
Tennessee	1	5	1	74	60	60	0	0	0	8	0	3
Alabama	0	1	0	19	41	33	0	0	0	0	1	1
Mississippi [2]	0	1	2	20	16	13	1	0	0	0	1	0
W. SO. CEN.												
Arkansas	3	1	1	6	5	16	0	0	2	3	5	3
Louisiana	0	0	0	8	7	8	0	1	0	3	3	5
Oklahoma	0	1	1	26	27	27	0	2	4	1	3	7
Texas	22	1	1	39	54	63	1	0	2	10	4	11
MOUNTAIN												
Montana	0	0	0	10	24	24	0	0	1	0	0	0
Idaho	0	0	0	1	7	15	1	0	1	0	1	1
Wyoming	0	0	0	3	5	8	0	0	0	0	1	1
Colorado	2	0	0	47	26	31	0	0	22	2	1	2
New Mexico	0	0	1	9	8	16	0	0	0	2	1	3
Arizona	1	1	0	2	9	5	0	1	1	3	1	1
Utah [2]	1	0	1	53	13	18	0	0	0	0	0	0
Nevada	0	0	------	------	------	10	------	0	------	0	0	------
PACIFIC												
Washington	0	3	1	23	37	37	0	0	0	0	0	0
Oregon	0	0	1	19	5	31	0	0	2	0	1	1
California	13	2	3	144	131	191	0	0	2	4	10	6
Total	66	58	58	2,967	3,100	3,741	13	9	119	79	105	143
49 weeks	4,047	8,957	8,957	118,838	119,173	151,214	750	1,295	9,280	6,532	8,128	12,416

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended December 12, 1942—Continued

Division and State	Whooping cough		Week ended Dec. 12, 1942								
	Week ended—			Dysentery			Encephalitis, infectious	Leprosy	Rocky Mt. spotted fever	Tularemia	Typhus fever
	Dec. 12, 1942	Dec. 13, 1941	Anthrax	Amebic	Bacillary	Unspecified					
NEW ENG.											
Maine	124	45	0	0	0	0	0	0	0	0	0
New Hampshire	8	14	0	0	0	0	0	0	0	0	0
Vermont	51	8	0	0	0	0	0	0	0	0	0
Massachusetts	305	199	0	0	2	0	0	0	0	0	0
Rhode Island	48	34	0	0	0	0	0	0	0	0	0
Connecticut	84	44	0	0	1	0	0	0	0	0	0
MID. ATL.											
New York	450	640	0	0	9	0	0	0	0	0	0
New Jersey	212	261	0	0	0	0	0	0	0	0	0
Pennsylvania	347	216	1	0	0	0	1	0	0	0	0
E. NO. CEN.											
Ohio	152	223	0	0	0	0	0	0	0	1	0
Indiana	16	19	0	0	0	0	0	0	0	1	0
Illinois	176	225	0	0	0	0	0	0	0	5	0
Michigan [2]	321	352	0	0	9	0	0	0	0	1	0
Wisconsin	216	273	0	0	0	0	0	0	0	4	0
W. NO. CEN.											
Minnesota	42	27	0	0	0	0	0	0	0	0	0
Iowa	33	16	0	1	0	0	0	0	0	0	0
Missouri	8	20	0	0	0	1	0	0	0	0	0
North Dakota	20	13	0	0	0	0	0	0	0	0	0
South Dakota	2	2	0	0	0	0	0	0	0	0	0
Nebraska	1	3	0	0	0	0	0	0	0	0	0
Kansas	27	33	0	0	0	0	0	0	0	0	0
SO. ATL.											
Delaware	6	2	0	0	0	0	0	0	0	0	0
Maryland [2]	126	53	0	0	0	5	0	0	0	2	0
District of Columbia	17	10	0	0	0	0	0	0	0	1	0
Virginia	29	35	0	0	0	16	0	0	0	1	0
West Virginia	23	35	0	0	0	0	0	0	0	0	0
North Carolina	33	118	0	0	0	0	0	0	0	0	3
South Carolina	32	28	0	1	0	0	0	0	0	2	4
Georgia	9	13	0	1	2	0	0	0	0	0	29
Florida	3	10	0	0	0	0	0	0	0	1	4
E. SO. CEN.											
Kentucky	15	60	0	0	0	0	0	0	0	2	0
Tennessee	42	12	0	0	0	0	0	0	0	0	4
Alabama	5	6	0	0	0	0	0	0	0	1	15
Mississippi [2]			0	0	0	0	0	0	0	0	1
W. SO. CEN.											
Arkansas	20	2	0	2	1	0	0	0	0	0	0
Louisiana	4	1	0	1	7	0	0	1	0	1	3
Oklahoma	11	4	0	0	0	0	0	0	0	0	0
Texas	161	69	0	5	75	0	0	0	0	0	24
MOUNTAIN											
Montana	33	59	0	0	0	0	1	0	0	0	0
Idaho	0	3	0	0	0	0	0	0	0	0	0
Wyoming	2	15	0	0	0	0	0	0	0	0	0
Colorado	14	38	0	1	2	0	0	0	0	0	0
New Mexico	16	16	0	0	0	0	0	0	0	0	0
Arizona	32	60	0	0	0	40	0	0	0	0	0
Utah [2]	17	23	0	0	0	0	0	0	0	0	0
Nevada	4	5	0	0	0	0	0	0	0	1	0
PACIFIC											
Washington	22	103	0	0	0	0	0	0	0	0	0
Oregon	5	35	0	0	0	0	0	0	0	0	0
California	248	151	0	8	8	0	0	0	0	0	3
Total	3,572	3,633	1	20	116	62	2	1	0	24	90
49 weeks	169,469	199,305									

[1] New York City only.
[2] Period ended earlier than Saturday.

WEEKLY REPORTS FROM CITIES

City reports for week ended November 28, 1942

This table lists the reports from 89 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Atlanta, Ga	1	0	1	0	0	0	7	0	7	0	0	0
Baltimore, Md	2	0	3	1	2	5	14	0	15	0	0	64
Barre, Vt	0	0	------	0	48	0	0	0	0	0	0	0
Billings, Mont	0	0	------	0	0	0	0	0	0	0	0	0
Birmingham, Ala	1	0	1	1	1	0	3	0	4	0	0	5
Boise, Idaho	0	0	------	0	0	0	0	0	0	0	0	0
Boston, Mass	0	0	------	1	1	1	13	0	59	0	0	42
Bridgeport, Conn	0	0	------	0	0	0	0	0	2	0	0	3
Brunswick, Ga	1	0	------	0	0	0	0	0	1	0	0	0
Buffalo, N. Y	0	0	------	0	34	0	9	0	4	0	0	17
Camden, N. J	1	0	------	0	0	0	2	0	0	0	0	4
Charleston, S. C	1	0	45	0	0	0	0	0	2	0	1	0
Charleston, W. Va	0	0	------	0	0	0	0	0	2	0	0	0
Chicago, Ill	6	0	5	2	19	0	19	0	57	0	1	87
Cincinnati, Ohio	3	0	------	0	9	0	5	0	18	0	0	9
Cleveland, Ohio	3	0	4	1	1	1	10	0	44	0	0	63
Columbus, Ohio	0	0	1	1	0	0	4	0	17	0	0	2
Concord, N. H	0	0	------	0	0	0	1	0	3	0	0	0
Cumberland, Md	0	0	------	0	0	0	0	0	0	0	0	0
Dallas, Texas	0	0	1	1	0	0	2	0	2	0	0	4
Denver, Colo	2	0	12	0	3	0	7	0	5	0	0	5
Detroit, Mich	1	0	------	2	1	1	20	1	28	0	1	60
Duluth, Minn	0	0	------	0	0	0	2	0	1	0	0	2
Fargo, N. Dak	0	0	------	0	0	0	0	0	0	0	0	0
Flint, Mich	2	0	------	0	0	0	5	0	2	0	0	8
Fort Wayne, Ind	0	0	------	0	0	0	1	0	0	0	0	0
Frederick, Md	0	0	------	0	0	0	0	0	0	0	0	0
Galveston, Texas	1	0	------	0	0	0	1	0	1	0	0	0
Grand Rapids, Mich	0	0	------	0	1	0	1	0	1	0	0	3
Great Falls, Mont	0	0	------	0	0	0	1	0	6	0	0	3
Hartford, Conn	0	0	------	0	1	0	4	0	2	0	0	7
Helena, Mont	0	0	------	0	0	0	0	0	0	0	0	1
Houston, Tex	1	0	------	0	0	0	3	1	1	0	0	2
Indianapolis, Ind	1	0	------	1	2	0	6	0	8	0	0	10
Kansas City, Mo	1	0	------	0	0	0	4	0	2i	0	0	1
Kenosha, Wis	0	0	------	0	1	0	0	0	3	0	0	0
Little Rock, Ark	0	0	6	0	0	0	3	0	0	0	1	0
Los Angeles, Calif	18	0	24	1	5	2	7	4	18	0	0	14
Lynchburg, Va	1	0	------	0	0	0	1	0	0	0	0	0
Memphis, Tenn	1	0	1	1	1	0	5	0	4	0	0	20
Milwaukee, Wis	0	0	------	0	14	0	6	0	45	0	0	31
Minneapolis, Minn	0	0	------	1	1	0	4	2	22	0	1	8
Missoula, Mont	0	0	------	0	0	0	0	0	0	0	0	0
Mobile, Ala	6	0	1	1	1	0	4	0	2	0	0	0
Nashville, Tenn	0	0	------	0	0	0	5	0	6	0	0	0
Newark, N. J	0	0	2	0	2	1	6	0	9	0	0	12
New Haven, Conn	0	0	------	0	0	0	0	0	5	0	0	12
New Orleans, La	0	0	1	1	0	0	5	0	3	0	1	0
New York, N. Y	13	0	9	2	13	4	77	0	114	0	2	81
Omaha, Nebr	2	0	------	0	0	0	6	0	7	0	0	1
Philadelphia, Pa	0	0	4	1	309	3	17	0	27	0	4	128
Pittsburgh, Pa	3	0	2	3	0	1	5	0	9	0	0	36
Portland, Maine	0	0	------	0	0	8	2	0	0	0	0	26
Providence, R. I	0	0	------	0	0	0	6	0	3	0	0	18
Pueblo, Colo	0	0	------	0	2	0	0	0	3	0	0	0
Racine, Wis	0	0	------	0	9	0	0	0	8	0	0	0
Raleigh, N. C	0	0	------	0	3	0	0	0	4	0	0	0
Reading, Pa	0	0	------	0	2	0	1	0	2	0	0	2
Richmond, Va	0	0	------	0	1	0	5	0	1	0	0	7

City reports for week ended November 28, 1942

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
Roanoke, Va	0	0	------	0	0	0	1	0	0	0	0	1
Rochester, N. Y	0	0	------	0	2	0	9	0	4	0	0	22
Sacramento, Calif	3	0	2	0	2	1	0	0	5	0	1	6
Saint Joseph, Mo	0	0	------	0	0	0	1	0	0	0	0	0
Saint Louis, Mo	2	0	2	1	1	0	10	1	14	0	0	8
Saint Paul, Minn	0	0	------	0	1	0	3	0	6	0	0	9
Salt Lake City, Utah	0	0	------	0	110	0	2	0	3	0	0	4
San Antonio, Tex	3	0	2	0	1	0	10	9	1	0	0	2
San Francisco, Calif	2	0	1	1	4	0	15	2	17	0	1	4
Savannah, Ga	0	0	2	2	0	0	1	0	1	0	0	4
Seattle, Wash	0	0	------	0	10	1	3	0	3	0	1	14
Shreveport, La	2	0	------	0	0	0	6	0	0	0	0	0
South Bend, Ind	0	0	------	0	0	0	0	0	0	0	0	0
Spokane, Wash	0	0	1	1	28	2	1	0	7	0	0	0
Springfield, Ill	0	0	------	0	0	0	0	1	1	2	0	13
Springfield, Mass	0	0	------	0	2	0	6	0	58	0	0	1
Superior, Wisc	0	0	------	0	1	0	0	0	2	0	0	2
Syracuse, N. Y	0	0	------	0	1	1	1	3	1	0	0	14
Tacoma, Wash	1	0	------	0	94	0	3	0	1	0	0	2
Tampa, Fla	0	0	------	0	0	0	2	0	0	0	1	0
Terre Haute, Ind	0	0	------	0	0	0	1	0	1	0	0	0
Topeka, Kans	0	0	------	0	2	0	5	0	2	0	0	1
Trenton, N. J	0	1	------	0	1	1	1	0	1	0	0	3
Washington, D. C	1	0	1	0	2	3	5	0	21	0	1	20
Wheeling, W. Va	0	0	------	0	0	0	2	0	0	0	0	0
Wichita, Kans	0	0	------	0	1	0	5	0	2	0	0	9
Wilmington, Del	0	0	------	0	0	0	3	0	0	0	0	13
Wilmington, N. C	1	0	------	0	1	0	0	0	1	0	0	3
Winston-Salem, N. C	0	0	------	0	0	0	0	0	1	0	0	1
Worcester, Mass	0	0	------	0	1	0	8	0	5	0	0	11

Dysentery, amebic.—Cases: New Haven, 1; New York, 5; San Francisco, 1.
Dysentery, bacillary.—Cases: Detroit, 4; Los Angeles, 11; New York, 8; Philadelphia, 2; Richmond, 3; San Francisco, 1; Syracuse, 1.
Tularemia.—Cases: Pittsburgh, 2.
Typhus fever.—Cases: Atlanta, 1; Baltimore, 1; Charleston, S. C., 2; Dallas, 1; Houston, 1; Little Rock, 1; Nashville, 2; New Orleans, 1; New York, 1; Savannah, 1; Shreveport, 1; Winston-Salem, 1.

Rates (annual basis) per 100,000 population for the group of 89 cities included in the preceding table (estimated population, 1942, 34,018,770)

Period	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
		Cases	Deaths						
Week ended Nov. 28, 1942	13.34	20.54	4.14	115.26	63.30	117.41	0.31	2.61	147.91
Average for week 1937–41	13.74	14.90	[1] 2.62	[2] 106.73	[1] 53.97	88.23	.73	2.83	118.76

[1] 3-year average, 1939–41.
[2] 5-year median.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended November 14, 1942.—During the week ended November 14, 1942, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis	1				4				6	11
Chickenpox		10		181	291	65	39	27	52	665
Diphtheria		26	1	36	2	9	2			76
Dysentery				126		1				127
Encephalomyelitis							1			1
German measles				5	8		4		9	26
Influenza		4			8				117	129
Lethargic encephalitis						1				1
Measles		15		28	44	11	43	3	7	151
Mumps		29	1	135	356	25	44	17	206	813
Pneumonia		2			5	1			20	28
Poliomyelitis				1	1				2	4
Scarlet fever		12	41	62	86	12	32	35	61	341
Tuberculosis	3	7	6	53	51	9	35	7	22	193
Typhoid and paratyphoid fever		1	2	8	1	1				13
Undulant fever					1					1
Whooping cough		3	1	163	120	24	2	13	18	344
Other communicable diseases		6		2	251	34	2	5	2	302

CUBA

Habana—Communicable diseases—4 weeks ended November 14, 1942.—During the 4 weeks ended November 14, 1942, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	25		Poliomyelitis	5	
Malaria	12		Tuberculosis	4	
Measles	5		Typhoid fever	15	2

Provinces—Notifiable diseases—4 weeks ended November 7, 1942.—During the 4 weeks ended November 7, 1942, cases of certain notifiable diseases were reported in the Provinces of Cuba, as follows:

Disease	Pinar del Rio	Habana[1]	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	2	1	5	11		5	24
Diphtheria	2	32	2	1	9	1	47
Leprosy				1		2	3
Malaria	365	14		57	8	221	665
Measles		2				11	13
Poliomyelitis	9	9	7	22	10	13	70
Rabies		1					1
Tuberculosis	10	16	27	38	2	39	132
Typhoid fever	8	27	18	33	3	23	112
Typhus fever						1	1
Yaws						1	1

[1] Includes the city of Habana.

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Except in cases of unusual prevalence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

(Few reports are available from the invaded countries of Europe and other nations in war zones.)

Plague

Peru—Lima Department—Barranca.—During the month of October 1942, 2 cases of plague were reported in Barranca, Lima Department, Peru.

Typhus Fever

Irish Free State—Galway County.—During the week ended November 21, 1942, 8 cases of typhus fever were reported in the rural district of Galway County, Irish Free State.

Rumania.—For the week ended November 21, 1942, 24 cases of typhus fever were reported in Rumania.

Tunisia.—For the period October 11–20, 1942, 38 cases of typhus fever were reported in Tunisia.

Turkey.—For the week ended November 21, 1942, 8 cases of typhus fever were reported in Turkey.

Yellow Fever

Colombia—Intendencia of Meta.—On October 31, 1942, 1 death from yellow fever was reported in Intendencia of Meta, Colombia.

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