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CURRENT PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES[1]

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The prevalence of certain important communicable diseases, as indicated by weekly telegraphic reports from State health departments to the United States Public Health Service, is summarized in this report. The underlying statistical data are published weekly in the PUBLIC HEALTH REPORTS, under the section entitled "Prevalence of Disease."

Typhoid fever.—The number of cases of typhoid fever reported for the current 4-week period was 706, as compared with 679, 717, and 883 for the corresponding period in the years 1932, 1931, and 1930, respectively. Regions reporting slight increases were the Middle Atlantic, East North Central, East South Central, and Mountain; those reporting decreases were the South Atlantic, West North Central, West South Central, and Pacific; the New England States reported the same number as last year for this period.

Scarlet fever.—The total number of reported cases of scarlet fever (21,144) represented a 10 percent decrease from last year's figure for the corresponding period. The incidence was approximately the same as in 1931 but was about 20 and 25 percent in excess of the corresponding period in the years 1930 and 1929, respectively. The disease seemed to be most prevalent in the East North Central region. The five States in that area reported 7,882 cases for the current period, which was the highest number reported from those States in the five years for which data are available. In other areas the incidence either closely approximated that of last year or was lower.

Measles.—For the whole reporting area the incidence of measles (67,856 cases) was less than for the corresponding period in any of the 3 preceding years, but was about 10 percent above the incidence in 1929. A comparison of geographic areas shows that within the individual areas there were wide variations. In the New England,

[1] From the Office of Statistical Investigations, U.S. Public Health Service. The numbers of States included for the various diseases are as follows: Typhoid fever, 48; poliomyelitis, 48; meningococcus meningitis, 48; smallpox, 48; measles, 48; diphtheria, 48; scarlet fever, 48; influenza, 38 States and New York City. The District of Columbia is counted as a State in these reports.

East North Central, and Mountain regions the numbers of cases reported for the current period were less than one-half the numbers reported for the same period last year, while in the West North Central States the number of cases (6,672) was 2.6 times that of last year, in the South Atlantic States 1.4 times last year's figure, in the East and West South Central groups 2.5 and 3.5 times last year's figure, and in the Pacific States there was a slight increase.

Diphtheria.—For the country as a whole the present year continues to maintain a record breaking low incidence of diphtheria. The number of cases reported for the 4 weeks ended May 20 was 2,033, or about 70 percent of last year's figure for the corresponding period and only about 50 percent of the average for recent years. A comparison of geographic areas shows that in all sections of the country, except the East and West South Central, the situation was similar to that described for the country as a whole. While the number of cases reported (377) from the South Central areas was not high, and very closely approximated that of last year for the same period, it was higher than in either of the 3 preceding years.

Meningococcus meningitis.—A decrease in the incidence of meningococcus meningitis was apparent in all geographic areas during the current 4-week period. The number of cases reported (230) was 83 percent of that for the corresponding period last year and less than 50 percent of the incidence in 1931. For this period in 1930 and 1929 the numbers of cases were 806 and 1,155, respectively.

A possible exception to the favorable situation was seen in the East and West North Central States, where an increase of 19 and 36 percent, respectively, over last year's figure was shown. While the incidence has not been unusually high in those regions, the number of cases for each 4-week period of the current year has been in excess of that for the corresponding period of last year.

Poliomyelitis.—Compared with previous years the poliomyelitis situation at the present is very favorable. The number of cases for the current period was approximately the same as that for the corresponding period last year and about 15 percent below the average for the last 5 years. For the current 4-week period the number of cases reported was 76, as against 54 for the preceding 4-week period. The greatest increases were reported from the South Atlantic and West South Central areas. The number of cases was not large in either area, 12 and 8 cases, respectively, but it represented a 50 percent increase in both regions over the preceding period.

Influenza.—For the current 4-week period the incidence of influenza (3,044 cases) was slightly below the level of the corresponding period in the years 1930 and 1929—fairly normal years in the incidence of the disease at this season. In 1932 and 1931 the minor epidemics of those years were still evident at this time, the number

of cases being 7,076 and 3,983, respectively. Each geographic area shared in the favorable situation for the current period. In the South Atlantic States, where an unusual number of cases is continuously reported, the incidence (930 cases, of which 843 were in South Carolina) was the lowest in the 5 years for which data are available.

Smallpox.—The current reported incidence (676 cases) of smallpox for the 4 weeks under report was about 56 percent of last year's reports. For the years 1931 and 1930 the numbers of cases totaled 3,423 and 5,512, respectively. The incidence remains at the relatively low level which it has maintained since the middle of the year 1930. Two geographic areas reported an excess over last year's incidence: In the Mountain region, Idaho reported 24 cases for the current 4-week period as against 4 for the same period last year; and in the Pacific area, California reported 131 for the current period as compared with 52 last year. Decreases in the various other areas averaged about 60 percent.

Mortality, all causes.—The average mortality from all causes in large cities, as reported by the Bureau of the Census, was the lowest in recent years. The rate for the 4-week period ended May 20 was 11.0 per thousand population (annual basis), as against 11.6, 11.9, and 12.5 for the corresponding period in the years 1932, 1931, and 1930, respectively. The average rate for this period for the years for which comparable data are available is 12.9.

THE SHWARTZMAN PHENOMENON: FACTORS COMPLICATING ITS USE IN THE TESTING OF ANTIMENINGOCOCCIC SERUM

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An interesting phenomenon reported by Shwartzman in 1928 may be briefly described as follows: Rabbits were given intracutaneous injections of filtrates of typhoid bacillus cultures. Twenty-four hours later they were given intravenous injections of the same material. Within four to five hours after the second injections many of these rabbits showed pronounced hemorrhagic necrosis at the site of the intracutaneous dose (1). Later, Doctor Shwartzman produced the same phenomenon with filtrates prepared from many other bacteria.

With many of these filtrates, including those from the meningococcus, neutralization of the reaction by immune sera was obtained (2).

In a subsequent report (3) the practical application of these facts to the evaluation of antimeningococcus serum was suggested, and

data were presented to show that serum neutralized the reacting factors of meningococcus filtrates.

It is generally agreed that evaluation of antimeningococcus serum is on an unsatisfactory basis, since the test-tube methods almost universally employed are completely arbitrary. A method involving the neutralization of some positive clinical sign in an animal would seem to be much more desirable. With the hope that this new method would give a better means of evaluating antimeningococcus serum than any of the strictly *in vitro* procedures in use, we applied this technique to a study of antimeningococcus sera in our laboratory. This paper describes some of our experiences with the Shwartzman phenomenon in regard both to the phenomenon itself and to its neutralization by serum.

EXPERIMENTAL WORK

(A) PREPARATION OF THE SHWARTZMAN ACTIVE AGENT

The technique described by Shwartzman (4) (5) was followed with very few deviations. The meningococci were grown on 1 percent glucose agar slants for 18 hours and suspended in 1 percent glucose broth, and 4 cc of the suspension was used as an inoculum for each Blake bottle of glucose agar. After four to five hours the bottles were inverted for the remainder of the 18 to 20 hours' incubation in order that the growth might not be washed off and autolyzed in the small amount of fluid in the bottle. After 18 to 20 hours this fluid was removed and the growth washed off with 0.87 NaCl solution containing 0.4 percent phenol, using 6 to 7 cc per bottle.

The pooled washings were at once centrifugated and filtered through Berkefeld V filters. After 48 hours' refrigerator storage, they were heated at 56° C. for 45 minutes to insure the death of any meningococci that might have passed through the filter. These antigens were made in amounts varying from 25 cc to 600 cc. Since 12 Blake bottles yield only about 50 cc of the finished product, the preparation of large quantities proved laborious. During our studies 31 lots were prepared, representing 15 chosen strains of meningococci.

(B) ACTIVITY OF SHWARTZMAN AGENT

These antigens were now tested for activity. The abdomens of large white rabbits were shaved and intracutaneous injections of 0.25 cc of 1 to 2 and of undiluted antigen were given. These injections were followed in 24 hours by intravenous injections (1 cc per kg of rabbit) of various dilutions of the same material. The results were recorded five hours later. Areas of hemorrhagic necrosis were read as + to + + + + according to the size of the area and intensity of the reaction.

The preparations varied widely in their activity. With some, pronounced hemorrhagic necrosis at the site of the intracutaneous injection was obtained with relatively high dilutions; with others, the activity was much less; still others showed no effect, even though undiluted material was used. This variation among different strains of meningococci was noted by Shwartzman (3) in his first paper on this subject. Such variation was found not only among different strains but also in various lots of antigen prepared from the same strain. Table 1 shows these differences.

TABLE 1.—*Variation in activity of Shwartzman antigens*

No.	Strain	Serological group	Source	Lot No.	Amount prepared	Date prepared	Date of titration	Highest dilutions in which reactions were produced
					Cc			
1	123	I	Rockefeller Institute, 1918.	A	50	Oct. 9, 1930	Oct. 14, 1930	1:2.
2				B	50	Oct. 18, 1930	Oct. 27, 1930	1:16.
3	198	I	Detroit, 1929	A	25	Nov. 14, 1931	Nov. 17, 1931	Inactive in all dilutions.
4	331	I	Memphis, 1930	A	50	Jan. 15, 1932	Jan. 18, 1932	1:200.
5				B	50	Jan. 22, 1932	Jan. 25, 1932	1:200.
6				D	50	Jan. 29, 1932	Feb. 1, 1932	1:400+.
7	267	I	New York State Laboratory. Received there from Rockefeller Institute, 1916.	A	200	Mar. 8, 1931	Mar. 10, 1931	1:320.
8				B	200	Mar. 14, 1931	Mar. 16, 1931	1:600+.
9				C	200	May 3, 1931	May 4, 1931	1:800.
10	55	II	Rockefeller Institute, 1917.	A	50	Oct. 9, 1930	Oct. 14, 1930	Inactive in all dilutions.
11				B	100	Oct. 18, 1930	Dec. 2, 1930	1:64+.
12				C	40	Apr. 12, 1932	Apr. 14, 1932	1:80+.
13	266	II	New York State Laboratory. Received there from Rockefeller Institute, 1916.	A	50	Jan. 15, 1932	Jan. 18, 1932	Inactive in all dilutions.
14				B	50	Jan. 22, 1932	Jan. 25, 1932	Do.
15				D	50	Jan. 29, 1932	Feb. 1, 1932	Do.
16	390	II	Original Gordon-Murray II, 1915.	A	50	Mar. 2, 1932	Apr. 14, 1932	1:20+.
17	173	II	Detroit, 1929	A	50	Apr. 10, 1932	Apr. 11, 1932	Inactive in all dilutions.
18	57	III	Rockefeller Institute, 1917.	A	50	Oct. 9, 1930	Oct. 14, 1930	Do.
19				B	50	Oct. 18, 1930	Mar. 10, 1931	1:160.
20	302	III	Indianapolis, 1930	A	50	Jan. 15, 1932	Jan. 18, 1932	1:400+.
21				B	50	Jan. 22, 1932	Jan. 25, 1932	1:400+.
22				D	50	Jan. 29, 1932	Feb. 1, 1932	1:400+.
23	60	IV	Rockefeller Institute, 1917.	A	50	Oct. 9, 1930	Oct. 14, 1930	1:2.
24				B	50	Jan. 15, 1932	Jan. 18, 1932	1:100.
25				C	50	Jan. 22, 1932	Jan. 25, 1932	1:100.
26				D	50	Jan. 29, 1932	Feb. 1, 1932	1:200.
27				E	50	Apr. 12, 1932	Apr. 14, 1932	1:20+.
28	395	IV	Gordon-Murray strain, 1915.	A	50	Mar. 2, 1932	do	1:20+.
29	425	IV	do	A	50	Apr. 10, 1932	do	1:20+.
30	158	IV	Chicago, 1928	A	50	do	do	1:20+.
31	155	N. f.*	do	A	50	Oct. 9, 1930	Oct. 14, 1930	Inactive in all dilutions.

* N. f. = *Neisseria flavescens.*

There seemed to be no relation between the activity of the antigens and serological grouping, though some individual strains regularly produced more active filtrates than others, e.g., 267 (see table 1).

The earlier preparations were made from strains which had been maintained in the laboratory for a number of years, i. e., strains 123, 55, 57, 60, 395, 425. It can be seen in table 1 that some of the anti-

gens made from these were not very active. There were also more recently isolated strains which were entirely inactive, e.g., 198, 173, 155. Recency of isolation did not seem to be an important factor in this respect. One of the most potent antigens was made from a fairly old strain, 267 (no. 8 of table 1).

(C) DIFFICULTIES MET IN TITRATION OF ACTIVE AGENT

The minimal amount of the active agent capable of causing a definite hemorrhagic reaction at the site of the previous intracutaneous injection in a rabbit was taken as an end-point in titration (5). With many preparations this end-point was very hard to find. Some of the factors responsible for this difficulty were as follows:

(1) There is a great variation in sensitiveness of rabbits to this agent. On one day 50 percent of the rabbits injected with given amounts of antigen would show definite reactions; 10 days later only 25 percent of a new set of rabbits might react to the same dilutions. Seventy-five percent of a third set of rabbits might show positive reactions. Thus, end-points found on several successive days often varied tremendously. Irregularity in reaction was often found in different parts of the same rabbit. Four seemingly identical intracutaneous injections on the abdomen of some rabbits would not react alike. Injections made into the skin of the inner surface of the ear showed similar variations in sensitivity. Occasionally a rabbit was found which gave positive reactions on both ears and abdomen, but more often the positive reactions would be irregularly distributed, some rabbits having positive ears and others positive abdomens. Sometimes one ear would be positive and the other negative. Although, on the whole, the skin of the ear reacted more often than that of the abdomen, the ear was nevertheless less satisfactory for routine use, because the end-point of the reaction was less definite. Some of the ear reactions did not develop until after 12 hours, and by that time earlier reactions were often less pronounced. On the other hand, after injections made on the abdomen, reactions were either positive or negative after five hours, and very rarely showed any subsequent change.

Several times an injection into the skin of one ear resulted in symmetrical areas of hemorrhagic necrosis on both, an observation which has also been made by Gratia and Linz (6).

(2) If the end-point in titrating these preparations be taken as the minimum amount that causes a reaction in one rabbit, it is obvious that, as pointed out by Shwartzman (5), several multiples of this amount must be used in order to produce a reaction regularly in a large proportion of animals. Thus it was necessary to use very large doses of the weaker preparations in order to obtain positive reactions with any regularity. This introduced other complications. These preparations, besides containing more or less of the Shwartzman

active agent, contain other things of uncertain nature in unknown quantities. It is difficult to titrate one active principle in the presence of such a mixture. Thus, some weak preparations were very toxic for rabbits, many of which died within a few hours after intravenous injection, though showing no positive Shwartzman reactions. The relation between the toxic agent and the Shwartzman active agent is not clear. They may be identical, but in these experiments there was no parallelism between them. Some of the most active preparations showed very little toxicity, whereas some of the weaker ones were so toxic that amounts large enough to produce Shwartzman reactions in the majority of rabbits would cause the death of the rabbits before the reactions could develop, e.g., antigens 57B and 395.

(3) The different preparations used in the experiments reported here varied widely in respect to stability. Some remained apparently unchanged over a period of five months (267–C); others showed deterioration after several weeks (331–D), and some actually seemed to increase in activity on storage.

Some of our experiments have indicated that the Shwartzman agent may be less heat stable than has been reported. Incubation for one hour at 37° C. has seemed to decrease activity in some preparations. Table 2 illustrates this effect of heating. It is possible that this heat susceptibility is only apparent and is due to variation in sensitiveness of the rabbits used; but the phenomenon has seemed to occur too frequently to be coincidental. Such a susceptibility to heat would have an important bearing upon the application of the Shwartzman phenomenon to serum testing; for incubating a serum and antigen mixture together at 37° C. before making intravenous injections might give an impression of serum neutralization that did not actually occur.

TABLE 2.—*Effect of heat on activity of Shwartzman agent*

Preparation	Activity of Shwartzman agent*	
	Heated filtrate	Unheated filtrate
	Percent	*Percent*
267–C	50	100
331–D	25	50
302–D	25	100
425	75	50
60–E	50	100
390	50	75

* Activity expressed by percentage of positive skin tests in rabbits.

(D) ACTUAL TITRATION OF SHWARTZMAN ACTIVE AGENT

Most of the factors discussed so far have been mentioned by Shwartzman in his series of papers. It was impossible to titrate many of the weaker antigens, i.e., to find an end-point sufficiently constant

for dependable reactions and sufficiently small so that several multiples did not kill the rabbits before the hemorrhagic necrosis had time to develop. Nevertheless, most of the stronger preparations were titrated quite satisfactorily.

Fully grown white rabbits were used. As a site for intracutaneous injection, the skin of the abdomen gave a more definite end-point than that of any other region. The abdomens were shaved, depilated with barium sulphide, or shorn with special electric clippers.

Injections of 0.25 cc of the undiluted antigen were made into the skin, only one such injection per rabbit being made, and in approximately the same location on each (these rabbits were divided into groups of four). Twenty-four hours later each received intravenously 1 cc per kilo of weight of a dilution of the antigen, each group receiving a different dilution. Both the highest dilution of antigen that could produce a reaction and the lowest dilution that failed to produce a reaction were determined, and the true end-point was taken to lie somewhere between them (5). For the most active preparations, this end-point was found to remain fairly constant for a time.

Shwartzman refers to the smallest amount of antigen that will produce a reaction as a "reacting unit" (5), and has advocated six units as a dose that should cause positive reactions in 75 percent of rabbits. With the active antigen for which a "reacting unit" could be satisfactorily determined, this rule was found to hold true, and our experiments entirely confirmed Doctor Shwartzman's observations on this point. Six reacting units could cause hemorrhagic necrosis in three of four rabbits in a group with a high degree of regularity. When this failed to occur, it usually meant that the antigen had lost some of its activity, and a re-titration was then necessary. The weaker antigens did not follow this rule.

(E) TECHNIQUE FOR STUDYING SERUM NEUTRALIZATION

The antigens that were chosen were made from the following strains: 267, 302, 331, 55, and 158. These were titrated in the manner previously described.

With these it became possible to study the neutralizing properties of antimeningococcic sera. Shwartzman's original methods (3) (7) were used. An intracutaneous injection of 0.25 cc of undiluted antigen was followed 24 hours later by an intravenous injection of a mixture of antigen and serum. This mixture was made up of diluted active agent containing six multiples of the end-point per cc and undiluted serum in the proportion of 4 to 1, i.e., four parts of filtrate to one part of serum. The dose was 1.25 cc per kilogram of rabbit. Unheated mixtures were used.

Each serum was tested on a group of four rabbits, and six or eight serums could be tested in one experiment. With each experiment

another group of four rabbits was given the Shwartzman agent without serum as a control on the activity of the antigen. The reactions were noted five hours after the intravenous injections. Satisfactory activity of the antigen was indicated by the production of positive reactions in at least 3 out of 4 rabbits in the control group. If all four rabbits which received a serum were negative, that serum was said to give "consistent neutralization" (CN) (7). If two or three out of four rabbits were negative, it was said to give "irregular neutralization" (IN). If no rabbits, or only one was negative, the serum was considered to give "no neutralization" (NN).

(F) VALENCY OF THE SHWARTZMAN ACTIVE AGENT

Since meningococci fall more or less into broad serological groups, as far as agglutination is concerned, it seemed important at this point to get additional information as to whether the Shwartzman active agent obtained from the principal groups of meningococci was the same or different. If it should be the same in all groups, the testing of antisera would be greatly simplified. If these preparations were group specific, the testing of polyvalent sera would, theoretically, require four times the number of animals, materials, and labor.

Monovalent group sera were obtained through the kindness of several manufacturers, as follows: Five of Group I, four of Group II, three of Group III, and four of Group IV, prepared in horses, sheep, goats, and rabbits. These were tested for neutralizing properties with a titrated antigen prepared from a representative member of each of the principal serological groups of meningococci. The results of these experiments indicate that the Shwartzman active agent shows no group specificity corresponding to the usual agglutination grouping

TABLE 3.—*Effect of monovalent sera on antigens of the same and of other serological groups*

Sera		Antigens			
Group	Source	Group I (331)	Group II (55)	Group III (302)	Group IV (158)
I	Sheep	IN[1]	IN	NN	IN
	Sheep	IN[1]	IN	IN	NN
	Horse	CN[1]	IN	NN	IN
	Rabbit	NN[1]	NN	NN	IN
	Rabbit	IN[1]	NN	NN	IN
II	Sheep	IN	IN[1]	NN	NN
	Sheep	NN	IN[1]	NN	IN
	Rabbit	IN	NN[1]	CN	IN
	Rabbit	IN	IN[1]	IN	IN
III	Sheep	NN	IN	NN[1]	CN
	Rabbit	IN	NN	NN[1]	IN
	Rabbit	IN	NN	IN[1]	CN
IV	Sheep	NN	IN	NN	NN[1]
	Sheep	NN	IN	NN	NN[1]
	Rabbit	CN	NN	NN	NN[1]
	Rabbit	CN	IN	NN	IN[1]

CN=Complete neutralization in all 4 rabbits tested.
IN=Neutralization in 2 or 3 of 4 rabbits tested.
NN=Neutralization in 1 or 0 of rabbits tested.
[1] Indicates serum of homologous group.

Table 3 shows the effect of the monovalent sera upon the group antigens. Only 1 serum out of 16 completely neutralized the active agent prepared from the homologous group, i.e., horse serum (Group I) neutralized antigen 331 (Group I). This antigen (331) was completely neutralized by two Group IV rabbit sera, although these Group IV sera had little effect on the antigen of their own group. The Group II antigen (55) was not neutralized completely by any serum. The Group III antigen (302) was completely neutralized by one Group II rabbit serum, but by no other; whereas the Group IV antigen (158) was neutralized completely by two Group III sera (one sheep and one rabbit) but by no others, and the Group IV antigen was neutralized completely by two Group III sera (one sheep and one rabbit) but by no others. Irregular neutralization occurred as often with the heterologous sera as with those of the same group. In interpreting the results obtained with these specific group sera, it is well to remember that they were prepared from different strains and in different animals which were under immunization for varying lengths of time. They were prepared as agglutinating sera, and the Shwartzman agent was not used as an antigen in any case. Results obtained with them are, nevertheless, not without significance and offer no evidence to show that there are differences in the Shwartzman agent which correspond to the agglutination groups of meningococci.

There is no indication that the use of several Shwartzman antigens in evaluating polyvalent therapeutic serum would serve as a measure of valency, in the usual sense, and in the following studies of the neutralizing potency of antimeningococcic serum only one antigen was employed. There is no proof that the Shwartzman agent obtained from all meningococci is the same; but if differences do exist, they do not seem to be correlated with agglutination.

(G) NEUTRALIZATION OF SHWARTZMAN ACTIVE AGENT BY THERAPEUTIC POLYVALENT ANTIMENINGOCOCCIC SERUM AND BY OTHER SERA

Strain 267 was chosen as a source of the antigen for routine use. This is a Group I strain which is "broad" enough agglutinogenically to overlap Groups II, III, and IV. Preparations of the Shwartzman active agent made from it have been more stable than those from any other strains used, the reactive unit of one lot remaining constant for more than five months. After this period, frequent retitrations were necessary.

Ninety polyvalent antimeningococcic sera, prepared for therapeutic use by 11 different manufacturers, were tested for the property of neutralizing the Shwartzman active agent, using the technique previously described. All of these, except two, met the Federal requirements as to agglutinin content. The immunization periods of the horses used varied from 6 to 17 months. In addition to these specific

antimeningococcic sera, 25 heterologous immune sera were studied, as follows: 12 antipneumococcic sera, 6 antidysenteric sera, 3 antigonococcic sera, 2 antistreptococcic sera, and 2 diphtheria antitoxins. Four normal horse sera, 1 normal sheep serum, and 1 normal rabbit serum were included, as well as 2 normal human sera, 2 convalescent sera, and 1 serum from a person who had recovered from an attack of meningococcus meningitis several years previously.

Only 38 of the 90 specific antimeningococcic sera, or 42 percent, completely neutralized the Shwartzman active agent when tested by the technique already described. These 38 comprised sera from all 11 manufacturers. Irregular neutralization was obtained with 47 of the 90 sera, or 52 percent. Only five sera, or less than 6 percent, gave no neutralization at all. All five of these had a high agglutinin content. Four sera especially prepared by immunizing horses with the Shwartzman active agent directly were found to give irregular neutralization. The results of these experiments with polyvalent antimeningococcic sera are shown in table 4.

TABLE 4.—*Serum neutralization of Shwartzman active agent*

Sera	Number of sera tested	CN	IN	NN
Specific:				
Therapeutic polyvalent antimeningococcic sera	90	38 (42%)	47 (52%)	5 (5.5%)
Sera prepared from Shwartzman antigen	4	0	4	0
Nonspecific	25	2 (8%)	13 (52%)	10 (40%)
Antipneumococcic	12	1	5	6
Antidysenteric	6	0	5	1
Antistreptococcic	2	0	0	2
Diphtheria antitoxin	2	0	1	1
Antigonococcic	3	1	2	0
Normal:				
Horse	4	3 (75%)	1 (25%)	0
Sheep	1	0	0	1
Rabbit	1	0	1	0
Human	2	0	0	2
Convalescent (still in hospital)	2	0	0	2
Recovered case (14 years ago)	1	0	1	0

CN=Complete neutralization in all 4 rabbits tested.
IN=Neutralization in 2 or 3 out of 4 rabbits tested.
NN=Neutralization in 1 or 0 of 4 rabbits tested.

None of the 25 nonspecific immune sera studied contained agglutinins for meningococci. Nevertheless two—1 antipneumococcic and 1 antigonococcic—completely neutralized the active agent; while 5 antipneumococcic, 5 antidysenteric, 2 antigonococcic sera, and 1 diphtheria antitoxin gave irregular neutralization. The remaining 11 gave no neutralization. The results obtained with these nonspecific immune sera are included in table 4. They show that 60 percent of these heterologous sera neutralized the Shwartzman active agent as well as did 52 percent of the specific antimeningococcic sera, although only 8 percent gave complete neutralization as compared with 42 percent of the specific sera. One-half of all antipneumococcic

sera tested and 5 out of 6 antidysenteric sera gave this irregular neutralization. However, 40 percent of these nonspecific sera gave no neutralization, as contrasted with only 5.5 percent of the specific sera.

Table 4 also shows that three of the four normal horse sera tested completely neutralized the active agent; the remaining normal horse serum and the one normal rabbit serum gave irregular neutralization; the one normal sheep serum gave no neutralization. None of these normal sera contained agglutinins for meningococci.

Five human sera were also included in these studies—two from normal people, two from convalescent cases of meningitis, and one from an individual who had recovered from an acute attack of meningococcus meningitis several years before. Only this last serum gave an irregular neutralization. Both the normal and the convalescent sera gave entirely negative results. Results with these human sera are also included in table 4.

(H) IS THE APPARENT NONSPECIFICITY OF NEUTRALIZATION DUE TO THE CONCENTRATION OF THE SERUM TESTED?

The results obtained with the 143 sera, presented in table 4, indicated that neutralization of the Shwartzman active agent is not specific when the test is performed as has been outlined. It seemed possible that the neutralizing titer of the specific antimeningococcic sera might be found by employing dilutions sufficiently high to make the neutralization in low dilutions by nonspecific sera of little importance.

TABLE 5.—*Effect of dilution on serum neutralization of Shwartzman active agent*

Kind of serum	Percentage of neutralization given by—	
	Undiluted serum	1:20 dilution of serum
Polyvalent therapeutic antimeningococcic sera:	*Per cent*	*Per cent*
A	100	25
B	100	67
C	75	67
D	75	67
Nonspecific sera:		
Antipneumococcic sera (M)	100	0
Antigonococcic sera (P)	100	33
Normal sera:		
Horse (P)	100	0
Horse (M)	100	33

A group of specific and nonspecific sera were chosen for the study of this question—4 antimeningococcic sera, of which 2 had given complete neutralization and 2 irregular neutralization; 1 antipneumococcic and 1 antigonococcic, both giving complete neutralization; and two normal horse sera, both giving complete neutralization.

These were tested in dilutions of 1:5, 1:10, and 1:20, using the diluted sera in the same way in which the undiluted sera had been used. The results are shown in table 5. The difference in the amount of neutralization obtained with the antimeningococcic sera and with the nonspecific and normal sera was not very great, though the neutralizing titers of the nonspecific and normal sera were, on the whole, somewhat less than those of the specific antimeningococcic sera. There was better neutralization with the undiluted sera than with the diluted.

Some experiments were done to determine the presence of a "prezone" in some of the sera which failed to neutralize consistently. These experiments, while few, did not indicate that a "prezone" was present in any of the sera tested.

DISCUSSION

It is not easy to interpret the results of these studies on serum neutralization of the very interesting Shwartzman reaction. Little is known of its underlying mechanism, or of the nature of the active principle.

Although there is no proof that preparations of the active agent from all strains of meningococci are immunologically alike, there is considerable evidence to show that any differences which may exist do not follow the usually recognized serological groups. One antigen has been used in testing polyvalent and normal sera. This strain was very broad agglutinogenically, but it is possible that results might vary with other antigens.

Nearly all (94.5 percent) of the polyvalent antimeningococcic sera prepared by manufacturers have neutralized the Shwartzman active agent to some extent. Complete neutralization has been obtained, however, when tested by the technique originally described by Shwartzman, with only 42 percent. On the other hand, 60 percent of nonspecific immune sera neutralized as well as at least 52 percent of the specific sera. Five out of six sera from normal animals neutralized to a considerable degree, and in three of these, this neutralization was complete. These three were from horses.

From the data presented here it would be difficult to distinguish an efficient antimeningococcic serum from a normal horse or an antipneumococcic serum, even if complete neutralization be used as a criterion. If "irregular neutralization" be the criterion, it would be impossible to distinguish an antimeningococcic serum from an antidysenteric serum or a diphtheria antitoxin. Apparently serum neutralization of the Shwartzman meningococcus active agent is nonspecific to a marked degree. The occurrence of neutralizing antibodies in 3 of 4 sera from normal horses would seem to interfere

seriously with the significance of such a method of testing therapeutic sera for potency.

Nonspecific neutralization by certain immune sera has been noted by Shwartzman (8), who has made use of these "auxiliary antibodies" in titrating specific sera. The presence of such nonspecific antibodies has in our own experiments been a complication rather than a help. It is difficult to see how complete neutralization under these circumstances can be an indication of therapeutic value, when heterologous sera and normal horse sera show the same effect.

It is possible that this nonspecific neutralization is conspicuous merely because all therapeutic antimeningococcic sera are too low in content of Shwartzman antibodies to neutralize except in very low dilutions. Most of these sera have been prepared primarily with the object of producing agglutinins and not Shwartzman antibodies. Should very potent Shwartzman antibody serum be prepared, it is quite conceivable that the neutralizing titer could be so high that the nonspecific neutralization described in this paper would sink into insignificance. So far no such sera have been obtained, the four prepared with the Shwartzman active agent giving only irregular neutralization with the antigen used. Nevertheless, it may be possible to prepare such sera. Unless this can be done, it is difficult to see how this method of evaluating therapeutic antimeningococcic sera can be used practicably. Could sera of high neutralizing potency be obtained, the relation of the Shwartzman active agent to meningitis in man might be known.

The relation of the Shwartzman principle to the clinical manifestations of meningococcus meningitis is at present undetermined. Branham and Lillie (9) have produced fatal meningitis in rabbits by intracisternal injection of Shwartzman antigen. Such preparations are too complex in composition for the identity of the Shwartzman agent and the "toxin" to be assumed.

CONCLUSIONS

Serum neutralization of the Shwartzman phenomenon produced by filtered meningococcus washings is not restricted to antimeningococcic sera, but also occurs with antipneumococcic, antidysenteric, and antigonococcic sera and with diphtheria antitoxin, as well as with normal horse and rabbit sera. This nonspecific neutralization is so frequent and so marked that it seems to limit the usefulness of the Shwartzman phenomenon in the evaluation of therapeutic antimeningococcic sera.

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ENDEMIC GOITER IN SWITZERLAND

A REVIEW OF RECENT CONTRIBUTIONS TO ITS ETIOLOGY, INCIDENCE, AND PREVENTION

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Although there is still a great deal of speculation, as well as divergence of opinion among continental observers, as to the etiology of endemic goiter, the recommendations of the Swiss public health authorities for the prevention of this widespread affection are unequivocal. In an official publication,[1] the Swiss Department of Public Health has reviewed the findings of the Swiss Commission for the Prevention and Cure of Goiter, which body has strongly recommended the general consumption of iodized table salt, also known as "filled" salt, "full" salt, iodine salt, or salt containing iodine. In support of this recommendation, the Swiss Goiter Commission has presented statistics showing the considerable incidence of endemic goiter and has discussed the need for prophylactic measures.

[1] Bulletin No. 10, Mar. 7, 1931. Swiss Federal Department of Public Health. Translated into English by Margaret Lloyd Sanger, clerk at the American consulate, Zurich, Switzerland.

The Swiss Goiter Commission believes that the most efficacious means of preventing endemic goiter is by supplying the entire population with iodized salt, for cooking as well as for table use. However, it is not practicable, for many reasons, to regulate the supply of iodized salt by Federal laws. Consequently, the commission has urged the several Cantons to introduce and regulate the supply of iodized salt by cantonal decrees. In several cantons such decrees have been effective for some time.

In order that the need for goiter prophylaxis may be better appreciated, the goiter commission has commented upon the effects of the disease and has also presented statistics to show its incidence.

Effects of goiter.—It has long been known that endemic goiter, a simple but abnormal enlargement of the thyroid gland, prevails to an unusual extent in Switzerland. The disease can, either directly or by bringing about a general bodily deterioration, prove very harmful. Through pressure upon the blood vessels of the throat, it greatly impairs the circulation, and by pressing upon the windpipe it not only hinders respiration, but in acute cases may cause sudden death by suffocation. During the period 1911–20 goiter was the cause of 1,516 deaths in Switzerland, according to the death certificates issued, and during the same period 463 persons died of cancer of the thyroid gland. Approximately 3,000 patients with goiter undergo operation each year.

Of even more serious consequence to the Swiss people are the organic troubles caused by goiter. A large percentage of the serious disturbances occurring during the period of growth, such as dwarfism, mental deficiency, idiocy, deafness, and dumbness, are an outcome of endemic goiter as it occurs in Switzerland. These conditions constitute a serious menace not only to individuals but to the prosperity and welfare of the entire country as well.

Incidence of endemic goiter in Switzerland.—That there is considerable goiter in Switzerland is apparent even to inexperienced observers. Thyroid surveys by qualified physicians among selected groups, such as school children, have fully corroborated superficial impressions. The comment and statistics presented by Dr. Otto Stiner,[2] afford striking evidence of the frequency with which goiter is encountered among recruits for compulsory military service, as well as the need for prophylactic measures.

Method of examining thyroids of recruits.—As all males in Switzerland are required to present themselves for military service at the ages of 19 and 20 years, an admirable opportunity is afforded for determining the prevalence of goiter at this period. In order that all examinations

[2] Distribution of endemic goiter in Switzerland: Otto Stiner, secretary, Swiss Goiter Commission, and member of the Swiss Federal Department of Public Health. (Presented at the International Conference on Goiter, Aug. 24-26, 1927, in Berne, and published in the report of the conference. English translation, edited by Hans Huber, Berne, 1929.)

may be as nearly uniform as possible, regulations have been prepared and standards devised by the chief military physician in collaboration with the Swiss Goiter Commission for the guidance of the various sanitary investigation commissions. Because of the interesting method of making the thyroid examinations and recording the results, the procedure is here briefly described.

The examining physician first studies the neck and chest of the recruit from the front for evidence of venous dilation. Deeply seated goiters are sought by having the recruit swallow. The neck is then measured, the tape being placed at the napé of the neck above the vertebra prominens, encompassing the most protruding point of the base of the neck. This measurement is first taken during quiet breathing. The recruit is then asked to distend (bear down upon) the neck as much as possible while keeping the mouth closed, and the second measurement is taken in the same relative position.

When there is a difference of 2 centimeters or more between the two measurements, there is, according to the Swiss authorities, ground for suspecting the presence of a goiter rich in blood vessels or that there is some interference with venous circulation. This view is held even when inspection and palpation during quiet breathing indicate only an insignificant enlargement of the thyroid gland. However, an increase of less than 2 centimeters in the measurements does not preclude the existence of goiter.

Finally the medical examiner places his hands around the neck of the recruit in such a manner that his two thumbs rest on the nape of the neck while he palpates with the index and middle fingers of each hand. If the form of the thyroid can not be made out in this way, the palpation is repeated while the person examined swallows. In the presence of mensural enlargement it may be surmised that a goiter is present even though it can not be readily palpated and the external appearance of the neck is but little altered.

Recording the results of thyroid examinations.—For military purposes the following findings are regarded as normal:

1. When the groove between the trachea and the lateral cervical muscles is filled by a slightly perceptible cushion and the isthmus can be felt as a thin layer of tissue in front of the trachea.

2. When the thyroid gland can be easily palpated, but the contour of the neck is not perceptibly changed and the gland does not extend into the thorax. Moreover, the neck should not be distended more than 2 centimeters by personal effort, and there should be no difficulty in breathing after considerable exertion.

The presence of goiters is recorded under the following conditions:

1. When the thyroid is uniformly enlarged, and the shape of the neck is materially changed and greatly distended through personal pressure, the presence of a diffuse goiter is recorded.

2. When separate nodules can be definitely palpated in a normally sized or a diffusely enlarged thyroid gland, the condition is regarded as an adenomatous (nodular or lumpy) goiter.

However, fitness for military service depends upon the location of the goiter, its size, and its relation to the trachea and the great vessels of the neck. The presence of a deep goiter, tracheal compression (marked by difficulty in breathing during and after exertion), and great increase in the size and number of the cervical vessels are regarded as constituting unfitness for military service. Unless the nodules are small and noncompressible, the presence of an adenomatous goiter also constitutes unfitness. A recruit with a nodular goiter is not permanently debarred from military service, but is excused from such activities for one year when it is likely that medical or surgical treatment will prove effective and the man declares himself prepared to undergo the necessary régime.

General goiter incidence among recruits.—The percentages of goitrous affections among recruits from the various Swiss Cantons are shown in table 1, which represents in condensed form the more comprehensive information available from each district. A study of this table shows that the incidence of endemic goiter ranges from 5.7 percent in the Canton of Vaud to 82.4 percent in the Canton of Zurich. The percentage ranges of incidence, which show a wide variation within the same Cantons, are set forth in the third column of the table. In the fourth column are shown the mean percentages, which range between 12.4 percent, in the Canton of Tessin, to 67.4 percent, in the Canton of Appenzell A. Rh. Considering that this comparatively high incidence of goiter occurs among young male adults, in which age and sex group the disease is least frequent, the greater incidence of the malady in the general population can be more readily understood.

TABLE 1.—*Percentage range of incidence and mean percentage incidence of endemic goiter among recruits for military service in 25 Cantons in Switzerland* [1]

Canton	Number of districts reporting	Percentage range of incidence	Mean percentage incidence
Zurich	11	34.1–82.4	53.4
Berne	30	11.4–64.8	40.7
Lucerne	5	32.0–50.6	45.2
Uri			41.0
Obwalden			53.9
Nidwalden			37.4
Schwyz	6	15.4–24.8	15.5
Glarus			41.1
Zug			45.4
Fribourg	7	16.2–58.0	36.0
Solothurn	10	17.0–33.9	23.6
Baselstadt			20.6
Baselland	4	13.7–26.5	21.3
Schaffhausen	6	39.8–52.8	45.7
Appenzell I. Rh.	3	60.9–63.6	61.8
Appenzell A. Rh.			67.4
St. Gall	14	35.5–69.4	56.2

[1] From Bulletin No. 10, dated Mar. 7, 1931, issued by the Swiss Federal Department of Public Health.

TABLE 1.—*Percentage range of incidence and mean percentage incidence of endemic goiter among recruits for military service in 25 Cantons in Switzerland*—Contd.

Canton	Number of districts reporting	Percentage range of incidence	Mean percentage ihcidence
Grisons	14	22. 5–43. 1	30. 1
Aargau	11	22. 5–73. 0	34. 5
Thurgau	8	38. 1–69. 6	61. 3
Tessin	8	7. 7–27. 4	12. 4
Vaud	19	5. 7–29. 6	15. 6
Valais	13	9. 3–80. 4	51. 1
Neuchatel	6	12. 9–27. 3	22. 0
Geneva			25. 8

Nodular goiter.—In many of the Swiss Cantons nodular goiter, also known as lumpy goiter, struma nodosa, or adenomatous goiter, is encountered with comparative frequency among recruits. In table 2, which has been condensed from the original compilation, are shown the percentage ranges of incidence and the mean percentage incidences in each of the 25 Swiss Cantons. Only in 2 of the 186 districts was nodular goiter absent among the recruits examined in 1924 and 1925. The percentage range of incidence was between zero in the Cantons of Berne and Grisons and 49.2 percent in the Canton of St. Gall. The mean percentage incidence of struma nodosa, as shown in column 4 of table 2, ranged from 2.2 percent, in the Canton of Baselstadt, to 31.4 per cent, in the Canton of Uri.

TABLE 2.—*Percentage range of incidence and mean percentage incidence of nodular goiter among recruits for military service in 186 districts of 25 Cantons in Switzerland during the years 1924–25* [1]

Canton	Number of districts reporting	Percentage range of incidence	Mean percentage incidence
Zurich	11	9. 9–38. 4	19. 8
Berne	30	0 –33. 5	13. 7
Lucerne	5	17. 6–31. 6	22. 7
Uri	1		31. 4
Obwalden	1		15. 1
Nidwalden	1		6. 7
Schwyz	6	8. 3–19. 4	11. 2
Glarus	1		14. 8
Zug	1		14. 5
Fribourg	7	4. 4–20. 5	9. 3
Solothurn	10	1. 9–15. 6	7. 6
Baselstadt	1		2. 2
Baselland	4	2. 7– 7. 9	4. 1
Schaffhausen	6	5. 8–12. 9	10. 2
Appenzell I. Rh	1		23. 0
Appenzell A. Rh	3	20. 3–22. 8	21. 1
St. Gall	14	11. 2–49. 2	23. 3
Grisons	14	0 –21. 3	8. 1
Aargau	11	3. 9–33. 4	12. 7
Thurgau	8	11. 7–33. 8	20. 2
Tessin	8	. 8– 8. 8	3. 3
Vaud	19	. 6–14. 2	4. 0
Valais	13	1. 3–19. 5	7. 1
Neuchatel	6	1. 2– 4. 7	2. 7
Geneva	1		5. 0

[1] Condensed from Table IV accompanying the article, Distribution of endemic goiter in Switzerland, by Otto Stiner. Report of the International Conference on Goiter, Berne, Aug. 24–26, 1927, p. 384, edited by the Swiss Goiter Commission, 1929. (English translation, Hans Huber, editor.)

Rejections because of goiter.—When a comparison is made between the most recent statistics and former records of goiter incidence, it is apparent that there has been an increase in the number of cases. At the same time the intensity of the malady has decreased, as indicated by the smaller number of large and symptom-producing goiters encountered. In table 3 are shown the percentage ranges of rejections and the median percentages of rejections because of goiter among the recruits examined in the years 1924 and 1925. In 7 Cantons and in 83 districts out of 186 no recruits had to be released on account of goiter. In 24 other districts the number rejected did not reach 1 percent of those examined. On the average, 1.3 percent were found to be entirely or partially unfit on account of goiter, a low figure when compared with figures of former days; yet the requirements for military service have become more rigid. According to Hunziker, 11.4 percent of the recruits examined in 1886 and 8.7 percent of those examined in 1891 were declared unfit for service.

TABLE 3.—*Percentage range of rejections and mean percentage of rejections because of goiter, among recruits for military service in 186 districts of 25 Cantons in Switzerland, during the years 1924–25* [1]

Cantons	Number of districts reporting	Percentage range of rejections	Mean percentage rejections
Zurich	11	1.6–5.6	3.5
Berne	30	0 –5.3	2.3
Lucerne	5	4.5–8.8	5.8
Uri	1	----------	.7
Obwalden	1	----------	1.9
Nidwalden	1	----------	1.9
Schwyz	6	.9–3.3	2.2
Glarus	1	----------	.1
Zug	1	----------	1.7
Fribourg	7	0 –0	0
Solothurn	10	0 –2.3	.35
Baselstadt	1	----------	.2
Baselland	4	.2–2.3	1.1
Schaffhausen	6	0 –2.9	.7
Appenzell I. Rh.	1	----------	.7
Appenzell A. Rh.	3	.5–1.0	.8
St. Gall	14	0 –5.3	1.0
Grisons	14	0 –1.5	0
Aargau	11	.4–5.8	3.4
Thurgau	8	0 –.7	.25
Tessin	8	0 –0	0
Vaud	19	0 –0	0
Valais	13	0 –3.3	0
Neuchatel	6	0 –0	0
Geneva	1	----------	0

[1] Table II from the article, Distribution of endemic goiter in Switzerland, by Otto Stiner. Report of the International Conference on Goiter, Berne, Aug. 24–26, 1927, p. 382, edited by the Swiss Goiter Commission, 1929. (English translation, Hans Huber, editor.)

Peculiarities of goiter distribution in Switzerland.—In the high plateau lands goitrous conditions and rejections plainly run along parallel lines. Rejections were frequent in the following goiter sections of Switzerland:

1. Lucerne.
2. Southern part of the Canton of Aargau.
3. Southeastern part of the Canton of Zurich.
4. Western parts of the Cantons of Thurgau and St. Gall.

In the lower Alps the relationship between goiter and unfitness for military service is less pronounced. Thus, in the Cantons of Berne and Lucerne the percentages of sufferers from goiter run quite high, approximately up to 50 percent, and the number of those unfit for service is correspondingly high, up to 5.3 percent. In the Canton of Appenzell, on the contrary, where goiter prevails as high as 60.9 to 67.4 percent, the number of rejected recruits is relatively low, 0.5 to 1.0 percent. In certain regions there is a parallel course between goiter in general and nodular goiter. However, in other regions this fails to hold true. From the Canton of Uri 41.0 percent of the recruits were goitrous and 31.4 percent were adenomatous. In the Glenner district, which also lies at a high altitude in the mountains of the Canton of Grisons, 43.1 percent of the recruits had goiters, but only 7.8 percent were of the nodular variety. Likewise in the Valais district of Herens, where 51.1 percent of the recruits were goitrous, only 3.8 percent were presumably adenomatous.[3]

According to Stiner, the so-called "mountain goiter," characterized by abnormal shape and enormous size, is becoming rare. Of special interest is the fact that recruits rejected because of goiter do not necessarily come from the mountainous regions but rather from low-lying strips of land, and especially from the high plateau lands. The Rhone Valley, as far as Martingy and the Rhine Valley up to Lake Constance, are two striking examples of comparatively low regions in which goiter has apparently increased. According to Professor Galli-Valerio, goiter now seems to be more frequent in the mid and low land sections of Switzerland.

Special observations in Fribourg and Vaud.—In order to clear up the much contested question of goiter incidence in the Fribourg-Vaud boundary, a special study was made by an investigator from the University of Berne. Formerly it was believed that goiter ceased at the western boundary of the Cantons of Fribourg and Vaud, which is supplied with salt from the Bex salt works, which salt is said to contain iodine in natural combination. On the contrary, the inhabitants of the Vaud enclave, which is entirely surrounded by Fribourg territory and are quite like the Fribourg people in their mode of living, are exceedingly goitrous. The Vaud members of the Swiss Goiter Commission have long declared that the Canton of Vaud is not free from goiter. Stiner believes that these findings tend to disprove the theory that goiter is due to a deficiency of iodine.

For his comparative investigations Th. von Fellenberg chose the Jura town of La Chaux-de-Fonds, which, in his opinion, was free from

[3] Inasmuch as the thyroid examinations of recruits were made by different physicians in the several Cantons, the possibility that the discrepancies mentioned by Stiner may have been due to variations in skill of the examiners and differences in applying the standards, should receive due consideration when these results are interpreted.

goiter, and the Emmental village of Signau, in which goiter was supposed to be quite prevalent. Furthermore, he selected the villages of Effingen, Hornussen, Kaisten, and Hunzenschwill in the Jura Mountains of the Canton of Aargau. Von Fellenberg assumed that there was scarcely any goiter in Effingen, while in the other villages the disease was believed to be present to a considerable extent. However, according to the data collected by the recruiting commission, the classification is not correct; at least it does not apply to young men in these villages who are 20 years of age. Chaux-de-Fonds is by no means free from goiter. On the other hand, the disease is not particularly prevalent in Signau. The two districts are classed in the same category, having between 20 and 30 percent of goiter. Effingen is not free from the disease. Stiner believes that if there is a relationship between endemic goiter and iodine deficiency, it is manifested in degenerative processes such as the formation of thyroid nodules rather than in the causation of endemic goiter. In support of this belief he cites the finding of 2.7 percent of nodular goiters in Chaux-de-Fonds and 10.7 percent of the same type in Signau.

Various views of the etiology of endemic goiter.—The conception that endemic goiter is due to a deficiency of iodine has not met with general acceptance. However, the failure to accept this theory, so widely supported by practical experimentation, application, and experience, is due in large part to an inadequate understanding of the underlying principles.[4]

At the International Conference on Goiter held in Berne in 1927 many views were advanced as to the etiology of endemic goiter. However, if the various etiological factors advanced at that time are carefully considered, it will be seen that practically all the ideas are included in the oft-repeated conception of the disease as set forth by Marine. However, because of the interest attached to some of the opinions concerning the etiology, a few may be mentioned here. All the statements quoted were made before the International Conference on Goiter, which was held in Berne in 1927.

[4] Endemic goiter is often called simple goiter; but it is simple in name only, for its causes are distinctly complex in character. In order that there may be no confusion or misunderstanding as to the relationship between iodine and endemic goiter as conceived by Marine, the American authority, his clear-cut explanations may here be restated. Marine has repeatedly emphasized that endemic goiter may be due either to an absolute or relative deficiency of iodine. (David Marine: The importance of our knowledge of thyroid physiology in the control of thyroid disease. Arch. of Int. Med., vol. 32, no. 6, pp. 811-827, December, 1923.) (Abs. Pub. Health Rep., vol. 39, no. 3, pp. 107-111, Jan. 18, 1924, Reprint No. 896.) In the case of absolute deficiency there is real shortage or actual lack of iodine in the food and water customarily available. In the case of relative deficiency, on the other hand, iodine may be available in sufficient quantities, but through some interference it may not be possible for the element to reach the gland and be utilized. Then, too, it may happen during adolescence, pregnancy, lactation, and the menopause, that the demand for iodine may be greater than the available supply, whereupon the gland undergoes hypertrophy. In all probability the majority of simple goiters are due to deficiencies of iodine which are relative and often complicated in character. Certain infections and intoxications, as well as the consumption of diets in which fat and proteids predominate, may likewise cause endemic goiter by interference with iodine intake.

Some theories of goiter etiology.—According to Dr. Maurice Freyss, of Strasburg-La Robertsau, endemic goiter is due to a variety of causes, including infestation of the intestinal canal with parasites.

Dr. E. Folley, of Paris, maintained that the condition is caused by the simultaneous presence of spirillæ and intestinal parasites. He claimed that the disease could be relieved by destroying the spirillæ with remedies containing arsenic and the parasites by the use of oil of chenopodium. He has abandoned the use of iodine.

According to Dr. Achille Marchesa Monneret, of Armeno, Italy, endemic goiter is associated with adverse economic conditions, poor hygiene, and improper food.

Dr. Andre Crotti, of Columbus, Ohio, stated that the following organisms are common both to endemic goiter and goitrigenous drinking water: (1) a gregarine; (2) a spirillum; (3) a flagellated infusorium; (4) a fungus.

By using iodine, which, according to Crotti, acts as a bactericide, the organisms are destroyed and the goiter is caused to disappear.

Dr. Robert McCarrison, of Coonoor, South India, believes that two theories have survived among the many advanced as the cause of endemic goiter: First, the iodine deficiency theory, which sees in the poverty of iodine in the water, soil, and foods grown on that soil, the essential cause of goiter; and, second, the infectious or toxic theory, which attributes the disease to some unknown pathogenic organism or its products. McCarrison believes that the truth lies in a judicious blend of both theories.

Dr. B. Galli-Valerio, of Lausanne, in commenting upon the many theories expounded during the conference, noted that a majority of the speakers favored drinking water as a cause of goiter. Thus, germs assumed to be in the water and chemical compounds absorbed from certain subsoils through which the water passed were frequently put forth as causes of the disease. However, the manner in which these various factors influenced thyroid enlargement was not clearly stated. Galli-Valerio was skeptical as to the influence of the factors mentioned, but was willing to admit that, as existing goiter may become aggravated through the use of contaminated drinking water, provision for potable water is a wise part of any campaign against goiter.

Prof. W. Kolle, of Frankfort-on-the-Main, regarded the iodine deficiency theory of goiter causation as the best established, particularly because of its practical success in reducing the incidence of thyroid enlargement among school children in endemic regions. Dr. Marcel Rhein, of Strasburg, was willing to accept the usefulness of iodine in preventing endemic goiter provided the treatment was supplemented by a diet rich in vitamins.

Prof. G. Pighini, of Reggio-Emelia, Italy, asserted that endemic goiter is due to the consumption of certain waters containing toxic chemical substances which enter into action with iodine compounds of the organism, more especially with the iodized hormones of the thyroid.

Stiner believes that nodular goiter is much more common among the German-Swiss, and ascribes the condition to the fact that these people have preserved their manners and mode of living, particularly their nutritional customs, through many generations. The vitamins are in part quite generally destroyed, owing to the habit of the housewives of adding soda to certain articles of food so that they will cook more quickly. It is well known, says Stiner, that the vitamins are very rapidly destroyed in an alkaline medium, even those which are heat proof in the presence of acids. The fundamental difference between the German-Swiss cuisine and that of Romance Switzerland lies in the fact that in the latter the foods containing iodine or vitamins are not subjected to improper cooking such as is prescribed in the cook books of the German-Swiss. Thus the mineral substances, including iodine, and the vitamins, are removed in great part before the foods are served at the table. Stiner believes that ultimate success in the prevention and cure of goiter depends more on a suitable revision of the cook book than on new scientific achievements. Coincident with the great economic boom, which Switzerland enjoyed at the beginning of the second decade of the present century, the severe varieties of goiter grew less and less in number. Thus in 1911 and 1912 only 2.9 percent of the recruits were unfit because of goiter. The explanation for this condition is to be seen directly in the betterment of standards of living.

Iodized salt for goiter prophylaxis.—In urging the general consumption of iodized table salt the Swiss Goiter Commission and the Federal Department of Public Health tacitly admit that this "silent" medication has definitely proved its efficiency in preventing endemic goiter, no matter what may be the underlying cause of the disease. The product used in Switzerland contains 5 milligrams of potassium iodide to each kilogram of sodium chloride, a proportion which is exceeded in some of the natural salt deposits. The iodizing is carried out in the salt works, 1 gram of potassium iodide being mixed with 200 kilograms of sodium chloride. According to the Goiter Commission 1 gram of iodide was often prescribed for a goiter patient in a single day. It is estimated that approximately 50 years would be required to consume 1 gram of potassium iodide mixed with ordinary table salt in the proportions recommended in Switzerland. In such small quantities iodine can not well prove harmful. If, in exceptional instances, a slight disturbance is occasioned by the use of iodized salt, rectification follows the return to the uniodized variety.

Consumption of iodized salt in Switzerland.—There has been a steady increase in the consumption of iodized salt in Switzerland since 1922. The percentage consumption of this product in the several Swiss Cantons during the period from 1922 to 1929 is shown in Table 4. It will be noted that in 1929 iodized salt was used exclusively in nine Cantons—Vaud, Nidwalden, Neuchatel, Schwyz, Schaffhausen, Obwalden, Zug, Uri, and Tessin. The annual consumption of iodized salt, in kilograms, from 1922 to 1929 is shown in table 5. It will be noted that the amounts have increased steadily and that in 1929 a large amount of this prepared salt was consumed.

TABLE 4.—*Percentage consumption of iodized table salt in the several Swiss Cantons during the period 1922–29* [1]

Canton	Year							
	1922	1923	1924	1925	1926	1927	1928	1929
Vaud	0	25	100	100	100	100	100	100
Nidwalden	0	47	100	100	100	100	100	100
Neuchatel	0	0	15	100	100	100	100	100
Schwyz	0	0.5	1	1	100	100	100	100
Schaffhausen	0	4	3	11	99	100	100	100
Obwalden	0	7	8	8	50	100	100	100
Zug	0	23	26	81	97	88	100	100
Uri	0	0	0	0.2	0.1	0.2	0.3	100
Tessin	0	0	0	0	0	0	0	100
Valais	0	0	33	63	65	75	78	80
Appenzell A. Rh	43	55	75	75	67	67	67	73
Appenzell I. Rh	0	34	50	50	48	46	53	54
St. Gall	0	12	24	27	25	26	27	47
Glarus	0	4	83	37	27	37	33	41
Thurgau	0	27	36	39	35	34	35	36
Grisons	0	3	6	9	9	13	16	18
Zurich	0	18	21	18	18	18	17	15
Baselstadt	0	5	10	12	12	13	14	15
Aargau	0	4	9	11	11	12	12	12
Baselland	0	2	5	5	11	12	12	10
Lucerne	0	5	3	4	6	6	6	7
Berne	0	0.8	1	4	4	4	4	5
Solothurn	0	1	2	2	2	3	3	3
Fribourg	0	0	0	2	2	2	2	2
Geneva	0	0.1	0.2	0.2	0.2	0.5	0.5	0.8

[1] The figures show the percentage of iodized salt consumed in comparison with the total amount of salt used. In those Cantons showing 100 percent the consumption of iodine-free salt amounts to less than one-half of 1 percent.

Table from Bulletin No. 10, dated Mar. 7, 1931, issued by the Swiss Federal Department of Public Health

TABLE 5.—*Amount (in kilograms) of iodized table salt consumed in Switzerland during the period 1921–29* [1]

Year	Kilograms of iodized salt	Year	Kilograms of iodized salt
1922	200, 000	1926	11, 800, 000
1923	3, 500, 000	1927	12, 800, 000
1924	7, 500, 000	1928	13, 100, 000
1925	10, 000, 000	1929	14, 482, 000

[1] From Bulletin No. 10, Mar. 7, 1931, issued by the Swiss Federal Department of Public Health.

When iodized salt is used in such large quantities two questions naturally arise: First, Is it actually efficient in preventing endemic goiter? and second, Is it harmful, particularly to individuals having

goiters? At the International Conference on Goiter in Berne there was considerable discussion on these points.

Does iodized salt prevent endemic goiter?—If iodized salt were the only goiter prophylactic used it would be comparatively simple to gage its effects. However, when goiter prevention is practiced it often takes the form of individual as well as general measures. In any event the situation is greatly complicated by the simultaneous administration of iodine in several forms. Despite this obvious handicap to accurate appraisal, a number of observers assert that the widespread consumption of iodized salt is beneficial. Thus Dr. H. Eggenberger, of Herisau, Switzerland, summarized the results of five years' use of iodized salt in the Canton of Appenzell, as follows:[5]

1. Operations for relief of goiter diminished 75 percent since 1923.
2. There was a total disappearance of congenital goiter.
3. The number of still-births and deaths among infants, due to thyrogenous debility, was diminished.
4. There was an average increase in weight at birth of 100 grams.
5. There was a disappearance of goiter among young school children.
6. There was a decrease in the incidence of goiter among adults.
7. No iodism due to the use of iodized salt was detected.

Prof. Wagner von Jauregg, of Vienna, cited a number of instances in which favorable results followed the use of iodized salt. In 1925 Zeller reported from Appenzell that 22 women who had used iodized salt during pregnancy gave birth to thyroid-normal infants. At the same time among 9 women who did not use such salt there were 7 thyroid-enlarged and only 2 thyroid-normal infants.

Bayard,[6] according to Wagner von Jauregg, demonstrated the ability of iodized salt to cause the disappearance of goiter, first among the members of five families and later in the populations of two villages. Dr. Hans Sepp, of Dietmannsried, has reported the observations in his sick-fund consultation practice which included persons beyond the school age in two regions, Kempten and Southofen, where iodized salt was used. During the six quarters prior to the use of the "full" salt between 15.4 and 22.6 percent of those applying for relief came because of goitrous conditions, while during the eight quarters following the introduction of iodized salt between 3 and 13.7 percent of his consultations were on account of goiter.

According to Eggenberger the measurements of thyroid areas of boys and girls entering the schools in Kempten in 1924, prior to the general use of iodized salt, were 23.8 and 22.1 square centimeters, respectively. Boys and girls entering school after iodized salt had

[5] These observations were reported in detail in vol. III, Handbuch der Innern Secretion, by H. Hirsch.
[6] O. Bayard: The goiter question. Schweiz. med. Wochen., vol. 53, pp. 701-724, July 26, 1923.

been supplied for two years had thyroid areas of 9.5 and 11.7 square centimeters, respectively.

Wahner-Jauregg also reported that goiter operations were fewer in number in Vienna following the use of iodized salt. There was also a decrease in the incidence of endemic goiter among the school children of Vienna between the years 1923 and 1927. However, this result could not be ascribed entirely to the consumption of iodized salt, for other prophylactics were used at the same time.

Dr. G. Maggia, of Sondrio, Italy, conceded the beneficial influence of iodine in the prophylaxis of endemic goiter but maintained that the measure is purely empirical. He believed improvement in the standards of living to be more important than the administration of iodine. Dr. Fr. Messerli, of Lausanne, who held a somewhat similar opinion, believed that the use of iodized salt was only a partial solution of the problem of prophylaxis. According to Messerli, iodides act upon the thyroid hypertrophy, which is a symption of goiter. The underlying cause of the goiter must be removed by proper hygiene, safe water, and suppression of infestation.

Alleged deleterious effects of iodized salt.—According to the Swiss Goiter Commission the greatest number of disturbances to the human system through the use of iodized salt have been reported in the United States of America, where a much larger quantity of iodine is used in the prepared table salt. In Switzerland injury to goiter patients through the use of iodine has been caused by so-called "wild" treatments; that is, by using the medication without competent guidance. The commission warns that all patent medicines used for the treatment of goiter contain iodine in excessive amounts, even when advertised as iodine free, and having such harmless names as "herb pills," etc. The commission feels that if the efforts to reduce the prevalence of endemic goiter by rational measures prove successful the uncontrolled use of iodine will steadily become lessened.

In order to determine whether iodized salt was responsible for the aggravation of existing goiters or harmful effects, Stiner circularized the 3,008 physicians in Switzerland. Among the 1,675 physicians who replied to the questionnaire, 79 reported a total of 167 cases in which it was thought that the use of iodized salt had been responsible for damage. Upon investigation it was found that only in 18 instances could the salt be held responsible. In this connection it is interesting to note that investigations by Eggenberger revealed the absence of iodine from much of the salt which was alleged to have been responsible for harmful effects.

Prof. L. Michaud, director of the medical clinic at Lausanne, reported that after the use of iodized salt in the Canton of Vaud over a period of three years not a single instance of iodism or of iodine-Basedow had been encountered either in private or hospital practice.

Professor Zollikofer, physician in chief of the department of internal medicine in the cantonal hospital of St. Gall, in which Canton a large number of iodized-salt disturbances were alleged to have occurred, is convinced that the advantages of iodized salt prophylaxis infinitely exceed the disadvantages. He declared that he had never observed any injurious effects from the use of iodized salt.

Doctor Roth, director of the cantonal hospital in Winterthur, has reported one case of iodism due to the use of iodized salt, but states that the condition cleared up promptly when uniodized salt was substituted.

Prof. A. Dieudonne, of Munich, stated that in Lindau, where cases of goiter were formerly of frequent occurrence among infants and small children, such cases are no longer observed since the introduction of "complete" salt. Furthermore, there have been no instances of health impairment which might be attributed to the consumption of iodized salt.

Despite these opinions as to the harmlessness of iodized salt, several Swiss physicians, namely, De Quervain and Bircher, have maintained from the beginning that this preparation has caused considerable harm. De Quervain [7] maintains that there have been no definite results following the addition of iodine to table salt. Furthermore, he has protested that iodine prophylaxis is too delicate a procedure to be carried out on such an extensive scale. The controlled sale of iodine and the exclusion of all hypersusceptible persons from treatment, he believes, are also necessary. More recently, however, de Quervain undertook personally the study of cases of illness apparently caused by the consumption of iodized salt. He concluded that hyperthyroidism may occur spontaneously without iodine consumption and that the number of cases reported is well within the range of spontaneous morbidity.

According to Bircher,[8] the uncontrolled use of iodine by the laity is to be condemned. All patients, he maintains, should be under the care of physicians. In 1920 Bircher saw 36 cases of thyropathy following the use of iodine, even in small doses. Furthermore, the physiology of the thyroid in normal and goitrous cases is so indefinite, the pharmacology of iodine so contradictory, and the experiences are so different, that it seems to Bircher a dangerous experiment to administer this effective poison to any great extent over a long period, either in food or otherwise. In Bircher's cases there was an impairment of health caused by the erroneous taking of iodized instead of plain salt. At the same time it should be mentioned that Eggenberger observed cases of thryotoxicosis in which careful investigation showed that the table salt was entirely free from iodine.

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[7] F. de Quervain: Iodine and Prophylaxis, Schweiz. med. Wochen., Aug. 31, 1922.

[8] E. Bircher: Iodine therapy of endemic goiter. Schweiz. med. Wochen., July 20, 1922.

SUMMARY

According to the Swiss Goiter Commission it has long been known that iodine, when employed in proper doses and under skillful direction in selected cases, will cause the disappearance of certain goiters. It has also been proved, experimentally and practically, that the administration of iodine in small doses will prevent endemic thyroid enlargement. After listening to the various papers in the Berne conference dealing with the etiology of endemic goiter, Kolle commented that such a typical disease as goiter could not possibly be due to so large a number of causes. He concluded that the etiological factors, such as altitude, improper diet, lack of vitamins, close blood relationship, heredity, injury to the nervous system, infectious diseases, intoxications, uncleanliness, improper hygiene, and other ascribed causes, are simply auxiliary factors which create the predisposition to thyroid enlargement. Kolle heard no convincing argument against the theory that the relative or absolute deficiency of iodine is the dominant cause of endemic goiter.

At the end of the conference on goiter, Dr. W. Silberschmidt, of Zurich, concluded that no effective arguments had been produced against the prophylaxis of endemic goiter by means of iodized table salt. Therefore, he felt that the action of the Swiss Goiter Commission in advocating this measure has been fully justified and confirmed.

COURT DECISION RELATING TO PUBLIC HEALTH

Marriage annulled where one party was venereally diseased at time of marriage.—(Delaware Superior Court; *Doe* v. *Doe*, 165 A. 156; decided Feb. 21, 1933.) Section 3004 of the Revised Code of Delaware, 1915, provided—

> A marriage may be annulled for any of the following causes existing at the time of the marriage: * * * (d) fraud, * * * at the suit of the innocent and injured party, unless the marriage has been confirmed by the acts of the injured party. * * *

Another law, section 2992 of the code, as amended by Laws 1921, chapter 182, contained the following:

> * * * It shall be unlawful for * * * a person who is venereally diseased, or a person who is suffering from any other communicable disease the nature of which is unknown to the other party to the proposed marriage, to marry.

The latter statute also provided that such a forbidden marriage should be voidable at the instance of the innocent party.

The plaintiff husband petitioned for an annulment of his marriage, basing his action on the grounds set forth in the above-quoted statutes. It was alleged in the petition that the defendant at the

time of the marriage had syphilis, that the plaintiff was ignorant of her condition until after the marriage, and that such marriage had not been confirmed by him after he learned the true facts. Medical testimony showed that the defendant at the time of the marriage had advanced syphilis and that she must, therefore, have known that she was afflicted with a serious venereal disease. The superior court granted annulment on both grounds, saying:

This court in *Williams* v. *Williams*, 2 W. W. Harr (32 Del.) 39, 118 A. 638, held that fraud, constituting a ground for annulling a marriage under the Revised Code of 1915, section 3004, paragraph d, must be fraud which went to the very essence of the marriage contract. In this case, the fraud alleged and proved by the plaintiff does go to the essence of the contract relation. The authorities are uniform in holding that the concealment of a venereal disease of a serious nature and incurable in character constitutes a valid ground for annulment of marriage on the ground of fraud. [Citations.]

2d. The petitioner has brought himself clearly within the provisions of the cited statute set forth as the second ground for annulment. It is true that the cause of action is not listed among the causes set forth in the divorce statute as a ground for annulment of marriage, but by act of the legislature it is expressly stated that it is unlawful for any person who is venereally diseased to marry and that the marriage at the instance of the innocent party is voidable. The plaintiff has been proven to have been the innocent party and the defendant is proven to have been afflicted at the time of the marriage with an incurable case of syphilis. Under the statute the marriage is voidable. While the statute is silent as to the form of action to be made use of by the injured party, I think it clear that annulment is the proper remedy.

DEATHS DURING WEEK ENDED MAY 20, 1933

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

	Week ended May 20, 1933	Corresponding week, 1932
Data from 85 large cities of the United States:		
Total deaths	7,553	7,963
Deaths per 1,000 population, annual basis	10.6	11.4
Deaths under 1 year of age	497	673
Deaths under 1 year of age per 1,000 estimated live births [1]	41	56
Deaths per 1,000 population, annual basis, first 20 weeks of year	11.8	12.4
Data from industrial insurance companies:		
Policies in force	68,086,402	73,132,558
Number of death claims	12,658	13,796
Death claims per 1,000 policies in force, annual rate	9.7	9.9
Death claims per 1,000 policies, first 20 weeks of year, annual rate	10.8	10.5

[1] 81 cities.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

Reports for Weeks Ended May 27, 1933, and May 28, 1932

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended May 27, 1933, and May 28, 1932

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932
New England States:								
Maine	3	1	5	1	6	253	1	0
New Hampshire	1	2			100	21	1	0
Vermont	1				2	269	0	0
Massachusetts	20	34		4	736	1,232	2	1
Rhode Island	1	3			2	43	0	0
Connecticut	2	6	2	3	226	273	1	0
Middle Atlantic States:								
New York	80	92	[1] 9	[1] 13	2,597	2,720	6	4
New Jersey	26	40	2	5	1,419	1,120	3	0
Pennsylvania	34	78			1,348	1,578	3	6
East North Central States:								
Ohio	9	15	8	5	469	808	0	0
Indiana	16	23	17	26	272	208	4	2
Illinois	26	51	27	32	802	821	14	2
Michigan	26	9	1	11	930	3,326	2	2
Wisconsin	2	9	17	14	332	1,617	1	2
West North Central States:								
Minnesota [2]	3	6	2		588	46	0	2
Iowa	2	7			20	3	0	0
Missouri	20	23	1	3	305	78	3	0
North Dakota	3	6			113	115	0	0
South Dakota	2	4			17	8	0	1
Nebraska	3	13			171	1	1	1
Kansas	7	4	1	1	244	307	1	0
South Atlantic States:								
Delaware	2				15	2	0	0
Maryland [3]	7	10	5	4	63	41	0	0
District of Columbia		3			21	18	1	1
Virginia [2]	13				241		0	
West Virginia	7	10	1	11	136	436	1	3
North Carolina	8	12	21	25	600	703	0	3
South Carolina [4]	9	6	130	355	214	134	0	0
Georgia [4]	10	9		92	156	95	2	0
Florida	5	3	1	1	18	3	1	0
East South Central States:								
Kentucky	4	4	20	24	113	63	1	1
Tennessee		6	9	52	150	11	1	4
Alabama [4]	4	7	17	13	86	6	0	3
Mississippi	2	5					0	1

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended May 27, 1933, and May 28, 1932—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932
West South Central States:								
Arkansas	5	1	9	1	425		1	0
Louisiana	13	35	20	4	23	8	0	2
Oklahoma [5]	5	10	12	10	110	19	0	0
Texas [4]	43	16	56	13	624	30	0	0
Mountain States:								
Montana [2]				3	50	56	0	0
Idaho [2]		3		2	12	1	0	0
Wyoming [2]	1	1			6	37	0	0
Colorado [2]	5	5	23		7	68	0	1
New Mexico	9	5			12	25	0	0
Arizona	3	2		2	103		1	1
Utah [3]			2		31		0	1
Pacific States:								
Washington	8	9			64	232	3	0
Oregon [3]		2	20	19	57	210	0	2
California	31	57	22	40	1,255	550	2	1
Total	481	647	460	789	15,351	17,595	57	47

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932
New England States:								
Maine	0	0	25	21	0	0	1	2
New Hampshire	0	0	14	22	0	0	0	0
Vermont	0	0	13	6	0	0	0	0
Massachusetts	4	1	406	469	0	0	4	4
Rhode Island	0	0	18	45	0	0	0	0
Connecticut	0	0	85	127	2	0	1	2
Middle Atlantic States:								
New York	2	4	651	1,322	0	0	5	12
New Jersey	1	2	212	326	0	0	12	1
Pennsylvania	0	2	711	649	0	0	6	8
East North Central States:								
Ohio	1	0	416	143	6	8	9	3
Indiana	0	0	92	51	1	10	17	2
Illinois	0	2	419	294	7	7	14	11
Michigan	1	1	356	431	0	9	1	6
Wisconsin	2	1	128	66	3	1	3	2
West North Central States:								
Minnesota [2]	0	0	80	103	1	4	1	1
Iowa	0	0	24	34	54	16	3	4
Missouri	0	0	66	41	2	1	10	0
North Dakota	0	2	6	4	3	3	1	0
South Dakota	1	1	8	6	0	1	3	0
Nebraska	0	0	24	11	3	15	2	1
Kansas	0	0	31	31	2	5	0	6
South Atlantic States:								
Delaware	0	0	15	18	0	0	2	1
Maryland [5]	0	0	106	80	0	0	8	8
District of Columbia	0	0	10	17	2	0	0	0
Virginia [3]	0		82		0		8	
West Virginia	1	0	25	32	0	0	7	5
North Carolina	0	1	35	23	2	5	12	8
South Carolina [4]	0	1	2	3	2	1	21	12
Georgia [4]	0	2	1	8	1	0	16	37
Florida	1	0	3	2	0	1	2	4
East South Central States:								
Kentucky	1	0	60	38	4	7	26	8
Tennessee	0	1	17	7	0	7	4	14
Alabama [4]	0	0	5	4	0	13	12	5
Mississippi	0	1	3	5	0	11	7	8

See footnotes at end of table

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended May 27, 1933, and May 28, 1932—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932	Week ended May 27, 1933	Week ended May 28, 1932
West South Central States:								
Arkansas	0	0	1	1	0	6	8	3
Louisiana	0	0	7	13	1	0	21	20
Oklahoma[5]	0	1	7	6	22	34	9	1
Texas[4]	1	1	50	14	10	31	26	6
Mountain States:								
Montana[3]	0	1	35	21	0	2	2	0
Idaho[3]	0	0	0	1	5	0	1	0
Wyoming[3]	0	0	9	2	0	1	0	1
Colorado[3]	0	0	28	19	0	1	1	5
New Mexico	0	0	7	8	0	1	1	2
Arizona	0	0	6	4	0	0	1	0
Utah[3]	0	0	4	8	0	0	0	0
Pacific States:								
Washington	1	0	44	22	2	6	1	4
Oregon[3]	1	0	22	6	19	6	1	1
California	2	1	150	152	34	20	2	26
Total	20	26	4,469	4,716	188	233	292	244

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended May 27, 1933, 21 cases: 1 case in Minnesota, 2 cases in Virginia, 5 cases in Montana, 1 case in Idaho, 8 cases in Wyoming, 3 cases in Colorado, and 1 case in Oregon.
[3] Week ended Friday.
[4] Typhus fever, week ended May 27, 1933, 11 cases: 1 case in South Carolina, 3 cases in Georgia, 4 cases in Alabama, and 3 cases in Texas.
[5] Figures for 1933 are exclusive of Oklahoma City and Tulsa, and for 1932 are exclusive of Tulsa only.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
March 1933										
Hawaii Territory	1	11	55	----	3	----	0	2	0	53
April 1933										
California	13	194	158	5	5,141	7	9	660	171	25
Georgia	8	41	465	99	530	56	----	36	----	28
Kansas	2	35	4	----	1,421	----	1	212	6	7
Louisiana	2	40	42	23	159	17	1	40	1	61
Montana	1	4	33	----	161	----	0	60	2	10
Nevada	----	----	27	----	----	----	0	35	1	2
North Carolina	5	73	79	----	2,708	62	1	232	1	29
Oklahoma[1]	14	27	158	95	742	12	0	62	54	4
Texas	14	349	1,106	352	----	57	2	337	----	54
Washington	1	20	65	----	268	----	2	175	55	3
Wisconsin	5	22	218	----	2,192	----	3	667	31	15

[1] Exclusive of Oklahoma City and Tulsa.

March 1933

	Cases
Hawaii Territory:	
Chicken pox	110
Conjunctivitis, acute	13
Conjunctivitis, follicular	29
Dysentery, bacillary	2
Hookworm disease	51
Impetigo contagiosa	2
Leprosy	12
Mumps	23
Streptococcic sore throat	3
Tetanus	3
Trachoma	6
Undulant fever	1
Whooping cough	283

April 1933

	Cases
Actinomycosis:	
California	2
Botulism:	
Montana	3
Chicken pox:	
California	2,652
Georgia	231
Kansas	473
Louisiana	31
Montana	192
Nevada	6
North Carolina	579
Oklahoma [1]	126
Washington	647
Wisconsin	2,470
Conjunctivitis:	
Georgia	2
Oklahoma [1]	3
Dysentery:	
California (amebic)	19
California (bacillary)	10
Georgia	20
North Carolina	1
Oklahoma [1]	4
Food poisoning:	
California	11
German measles:	
California	64
Kansas	240
Montana	2
North Carolina	43
Washington	14
Wisconsin	21
Granuloma, coccidioidal:	
California	9
Hookworm disease:	
Georgia	394
Louisiana	12

April 1933—Continued

	Cases
Impetigo contagiosa	
Montana	7
Leprosy:	
California	2
Washington	1
Lethargic encephalitis:	
California	1
Kansas	2
Texas	4
Washington	2
Wisconsin	1
Mumps:	
California	1,237
Georgia	379
Kansas	710
Louisiana	3
Montana	15
Oklahoma [1]	31
Washington	326
Wisconsin	935
Ophthalmia neonatorum:	
California	4
Oklahoma [1]	1
Wisconsin	1
Paratyphoid fever:	
Georgia	2
Louisiana	3
Texas	8
Psittacosis:	
California	1
Puerperal septicemia:	
Washington	1
Rabies in animals:	
California	35
Louisiana	3
Washington	9
Rocky Mountain spotted fever:	
California	1
Montana	14
Nevada	3
Washington	2
Scabies:	
Kansas	3
Oklahoma [1]	16
Septic sore throat:	
California	12
Georgia	24
Kansas	4
Montana	1
North Carolina	5
Oklahoma [1]	29
Silicosis:	
Montana	1

April 1933—Continued

	Cases
Tetanus:	
California	7
Georgia	4
Louisiana	4
Washington	1
Tick paralysis:	
Montana	1
Trachoma:	
California	29
Georgia	15
Kansas	1
Montana	1
Oklahoma [1]	14
Wisconsin	1
Trichinosis:	
California	4
Tularæmia:	
California	1
Georgia	2
Kansas	2
Louisiana	2
Montana	4
Nevada	1
North Carolina	3
Oklahoma [1]	1
Typhus fever:	
Georgia	22
Louisiana	1
North Carolina	1
Undulant fever:	
California	13
Georgia	5
Kansas	2
Louisiana	1
North Carolina	1
Oklahoma [1]	4
Washington	3
Wisconsin	6
Vincent's angina:	
Kansas	1
Oklahoma [1]	8
Washington	1
Whooping cough:	
California	2,403
Georgia	252
Kansas	350
Louisiana	77
Montana	29
Nevada	4
North Carolina	576
Oklahoma [1]	44
Washington	31
Wisconsin	665

[1] Exclusive of Oklahoma City and Tulsa.

WEEKLY REPORTS FROM CITIES

City reports for week ended May 20, 1933

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Maine:											
Portland	0	------	0	0	0	3	0	0	0	1	18
New Hampshire:											
Concord	0	------	0	0	2	8	0	1	0	0	11
Manchester	0	------	0	0	0	0	0	1	0	0	6
Nashua	0	------	0	0	0	0	0	0	0	0	--------
Vermont:											
Barre	0	------	0	0	0	0	0	0	0	3	4
Burlington	1	------	0	0	0	1	0	0	0	0	5
Massachusetts:											
Boston	15	------	1	199	35	81	0	9	2	28	221
Fall River	0	1	1	0	1	4	0	1	0	7	--------
Springfield	0	------	1	7	0	11	0	0	0	7	35
Worcester	0	------	0	10	2	23	0	1	0	10	47
Rhode Island:											
Providence	2	------	0	0	4	72	0	3	0	13	65

City reports for week ended May 20, 1933—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Connecticut:											
Bridgeport	0	2	0	28	2	22	0	2	0	0	21
Hartford	0	------	0	4	0	28	0	2	9	4	26
New Haven	0	------	0	8	2	3	0	0	0	22	27
New York:											
Buffalo	9	------	1	52	15	39	0	6	0	35	133
New York	44	11	5	1,300	139	245	0	96	3	114	1,478
Rochester	0	------	0	3	4	26	0	0	0	6	59
Syracuse	0	------	0	3	1	10	0	0	0	11	40
New Jersey:											
Camden	4	------	0	19	1	13	0	0	0	0	18
Newark	0	1	0	161	7	13	0	10	0	48	80
Trenton	0	------	0	13	0	9	0	2	0	6	34
Pennsylvania:											
Philadelphia	4	2	2	443	28	114	0	26	1	5	464
Pittsburgh	0	2	2	16	9	85	0	5	0	34	124
Reading	0	------	0	15	2	8	0	0	1	6	26
Scranton	2	------	------	0	------	4	0	------	0	0	------
Ohio:											
Cincinnati	1	------	1	6	6	19	0	4	0	8	115
Cleveland	7	35	1	2	15	161	0	9	1	30	168
Columbus	0	2	2	9	5	27	0	4	1	0	58
Toledo	0	------	0	213	8	118	0	10	0	7	89
Indiana:											
Fort Wayne	6	------	0	1	2	6	0	0	0	0	28
Indianapolis	2	------	0	141	4	5	0	6	0	10	------
South Bend	0	------	0	3	2	1	0	1	0	2	17
Terre Haute	0	------	0	12	3	9	0	2	0	1	14
Illinois:											
Chicago	2	1	3	505	44	301	0	50	1	40	664
Cicero	0	------	0	5	0	4	0	0	0	0	3
Springfield	2	------	0	0	2	6	0	0	0	0	16
Michigan:											
Detroit	19	------	0	350	15	152	0	28	0	128	238
Flint	1	9	0	17	5	7	0	0	0	0	23
Grand Rapids	0	------	0	6	1	8	0	1	0	12	22
Wisconsin:											
Kenosha	0	------	0	0	0	2	0	0	0	33	2
Madison	0	------	------	87	------	1	0	------	0	3	------
Milwaukee	0	1	1	6	3	38	0	5	0	77	87
Racine	0	------	0	0	1	8	0	0	0	8	10
Superior	0	------	0	0	0	0	0	1	0	10	12
Minnesota:											
Duluth	0	------	1	9	2	0	0	3	0	60	29
Minneapolis	1	------	0	55	3	47	0	2	1	36	92
St. Paul	0	------	0	271	2	24	0	4	0	84	52
Iowa:											
Des Moines	2	------	------	0	------	9	5	------	0	0	18
Sioux City	0	------	------	4	------	1	0	------	0	5	------
Waterloo	0	------	------	1	------	1	0	------	0	0	------
Missouri:											
Kansas City	1	------	1	45	5	33	0	6	0	1	89
St. Joseph	0	------	0	27	6	0	0	2	0	1	43
St. Louis	12	------	1	128	5	12	0	6	3	12	149
North Dakota:											
Fargo	0	------	0	0	1	0	0	1	0	0	8
Grand Forks	0	------	0	0	0	1	0	0	0	0	------
South Dakota:											
Aberdeen	0	------	0	0	0	0	0	0	0	0	------
Nebraska:											
Omaha	1	------	0	134	5	2	0	1	0	9	50
Kansas:											
Topeka	0	------	0	113	1	2	0	1	0	2	17
Wichita	0	------	0	1	0	0	0	1	0	5	31
Delaware:											
Wilmington	0	------	0	6	2	4	0	1	0	1	25
Maryland:											
Baltimore	0	2	0	7	7	67	0	20	1	41	189
Cumberland	1	------	0	9	0	1	0	0	0	0	11
Frederick	0	------	0	0	0	0	0	0	0	1	1
District of Col.:											
Washington	0	------	0	19	8	8	0	9	1	9	129

City reports for week ended May 20, 1933—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Virginia:											
Lynchburg	2		0	28	0	0	0	1	0	6	14
Norfolk	0		0	8	2	2	0	1	0	2	20
Richmond	5		2	8	2	3	0	0	1	1	50
Roanoke	0		0	13	0	3	0	1	1	3	15
West Virginia:											
Charleston	0	1	0	0	0	0	0	0	0	0	17
Huntington	0			0		2	0		0	0	
Wheeling	0		0	5	4	2	0	0	0	7	16
North Carolina:											
Raleigh	0		0	3	1	0	0	0	0	2	5
Wilmington	0		0	31	3	0	0	0	0	0	13
Winston-Salem	0		0	16	0	2	1	0	0	2	11
South Carolina:											
Charleston	1	13	0	0	0	0	0	3	0	0	20
Columbia	0		0	0	2	0	0	1	0	0	14
Greenville	0		0	4	3	0	0	1	0	1	11
Georgia:											
Atlanta	0	12	1	44	5	0	0	3	2	27	72
Brunswick	0		0	0	0	0	0	0	0	0	3
Savannah	0	1	0	1	1	0	0	0	1	0	33
Florida:											
Miami	0		0	0	0	1	0	0	0	7	10
Tampa	2	1	1	0	2	0	0	2	0	1	18
Kentucky:											
Ashland	0		0	3	0	1	0	0	0	3	
Lexington	0		0	4	0	2	0	2	0	2	15
Louisville	0		0	13	8	9	0	2	0	9	80
Tennessee:											
Nashville	0		0	8	2	2	0	0	0	7	35
Alabama:											
Birmingham	1		0	1	4	3	0	4	0	3	51
Mobile	1		0	12	2	0	0	0	0	2	18
Montgomery	0			8		0	0		0	7	
Arkansas:											
Fort Smith	0			1		0	0		0	5	
Little Rock	1		0	141	0	0	[1] 2	2	0	0	2
Louisiana:											
New Orleans	5	3	5	12	6	6	0	6	1	8	125
Shreveport	0		0	3	0	0	0	1	0	0	33
Oklahoma:											
Oklahoma City	0	26	0	36	4	0	0	4	0	0	33
Tulsa	0			44		1	2		0	13	
Texas:											
Dallas	2		0		5	6	0	1	1	13	59
Fort Worth	1		1	5	3	1	0	1	0	0	38
Galveston	0		0	0	2	2	0	1	0	0	13
Houston	2		0	3	7	1	0	7	0	4	62
San Antonio	1		0	25	3	1	0	6	0	1	80
Montana:											
Billings	0		0	1	0	0	0	0	0	0	5
Great Falls	0		0	0	0	0	0	0	0	4	5
Helena	0		0	0	0	0	0	0	0	0	3
Missoula	0		0	26	0	1	0	0	0	0	4
Idaho:											
Boise	0		0	3	0	0	1	0	0	1	3
Colorado:											
Denver	1	23	0	2	6	21	0	4	0	1	68
Pueblo	0		0	0	1	0	0	0	0	0	10
New Mexico:											
Albuquerque	0		0	0	0	0	0	2	1	9	8
Utah:											
Salt Lake City	1		0	16	1	2	0	0	0	15	26
Nevada:											
Reno	1		0	0	1	0	0	1	0	0	3
Washington:											
Seattle	1			11		23	0		0	6	
Spokane	0			4		0	1		0	0	
Tacoma	0		0	1	2	6	1	2	0	0	33
Oregon:											
Portland	1		0	1	1	7	4	2	0	4	66
Salem	0	2		5		0	0		0	0	
California:											
Los Angeles	24	12	0	519	15	52	14	25	0	80	292
Sacramento	0		0	2	0	0	0	3	2	55	24
San Francisco	1		1	4	11	7	0	6	6	94	152

[1] Nonresident.

City reports for week ended May 20, 1933—Continued

State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths	
New York:			
New York	4	2	1
Pennsylvania:			
Pittsburgh	0	1	0
Indiana:			
Indianapolis	1	0	0
Illinois:			
Chicago	13	6	0
Michigan:			
Detroit	1	1	0
Wisconsin:			
Milwaukee	3	0	0
Minnesota:			
St. Paul	1	1	0
Missouri:			
St. Joseph	0	1	0
St. Louis	2	1	0
Delaware:			
Wilmington	0	0	1
South Carolina:			
Columbia	0	1	0
Louisiana:			
New Orleans	0	0	1
Oklahoma:			
Oklahoma City	2	0	0
California:			
Los Angeles	1	0	3
San Francisco	1	1	0

Lethargic encephalitis.—Cases: Philadelphia, 1; Milwaukee, 1; Wichita, 1; Birmingham, 1.
Pellagra.—Cases: Winston-Salem, 2; Charleston, S.C., 3; Savannah, 2; Lexington, 1; New Orleans, 1.
Typhus fever.—Cases: Springfield, Ill., 1; Savannah, 1.

FOREIGN AND INSULAR

CUBA

Habana—Communicable diseases—Four weeks ended May 20, 1933.—During the 4 weeks ended May 20, 1933, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	20	8	Scarlet fever	9	2
Leprosy	1	1	Tuberculosis	23	6
Malaria	5		Typhoid fever	11	7
Rabies	2	2			

Provinces—Communicable diseases—Four weeks ended April 1, 1933.—During the 4 weeks ended April 1, 1933, cases of certain communicable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Chicken pox		4	1	2	1		8
Diphtheria		6	2	4		2	14
Malaria	46	10	283	41	42	31	453
Measles		1	2	9			12
Scarlet fever		1					1
Tuberculosis	3	14	5	14	7	3	46
Typhoid fever	1	16	7	20	8	17	69

CZECHOSLOVAKIA

Communicable diseases—March 1933.—During the month of March 1933 certain communicable diseases were reported in Czechoslovakia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	5	3	Paratyphoid fever	23	1
Cerebrospinal meningitis	13	6	Poliomyelitis	7	1
Chicken pox	389		Puerperal fever	52	21
Diphtheria	2,669	133	Scarlet fever	1,885	20
Dysentery	6		Trachoma	155	
Influenza	2,722	45	Typhoid fever	371	27
Lethargic encephalitis	2	2	Typhus fever	9	1
Malaria	12				

SWITZERLAND

Vital statistics—Years 1931 and 1932.—The following table shows the number of births and deaths, together with deaths from certain diseases, reported in Switzerland during the years 1931 and 1932.

	1931	1932		1931	1932
Number of births	68,249	68,644	Number of deaths from—Contd.		
Number of deaths	49,414	49,910	Influenza	1,855	1,924
Number of deaths under 1 year of age	3,374	3,499	Measles	84	87
Number of deaths from—			Pneumonia	3,127	2,853
Arteriosclerosis	5,004	5,402	Puerperal fever	118	140
Cancer	5,671	5,837	Scarlatina	34	31
Diphtheria	127	92	Tuberculosis, pulmonary	3,768	3,528
Enteritis	380	395	Tuberculosis, other forms	1,206	1,219
Heart disease	6,209	6,045	Typhoid fever	32	27
			Whooping cough	88	185

NOTE.—The population of Switzerland, according to the 1930 census, was 4,077,000.

YUGOSLAVIA

Communicable diseases—April 1933.—During the month of April 1933 certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	24		Poliomyelitis	2	2
Cerebrospinal meningitis	15	10	Scarlet fever	154	9
Diphtheria and croup	483	63	Sepsis	12	4
Dysentery	17	1	Tetanus	27	14
Erysipelas	136	10	Typhoid fever	182	30
Measles	494	12	Typhus fever	31	6
Paratyphoid fever	1				

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

(NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for May 26, 1933, pp. 586-596. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued June 30, 1933, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Cholera

Philippine Islands.—During the week ended May 27, 1933, cholera was reported in the Philippine Islands as follows: Leyte Province, 4 cases, 4 deaths; Bohol Province, 15 cases, 12 deaths; Pampanga Province, 1 case, 1 death.

Yellow Fever

Gold Coast.—During the week ended May 20, 1933, a case of yellow fever was reported at Oda, Gold Coast.