ELECTROCARDIOGRAPHIC ALTERATIONS IN ADULT RATS AS A RESULT OF ACUTE THIAMINE DEFICIENCY [1]

By JAMES M. HUNDLEY, *Senior Assistant Surgeon*, and W. H. SEBRELL, *Medical Director, United States Public Health Service*

Previous communications from this laboratory (*1, 2, 3, 4*) have described the pathologic and electrocardiographic abnormalities that develop in young rats in chronic thiamine deficiency.

It is the purpose of this paper to describe the electrocardiographic changes that develop in adult rats in acute thiamine deprivation.

METHODS

Two groups of rats were studied. One group consisted of 24 female rats approximately 9 months of age, which were being discarded from a breeding colony because of their age and reduced fertility. Litter records were not available. Twenty of these were of Buffalo strain and 4 of National Institute of Health strain. Their weights varied from 160 to 270 gm., averaging 195 gm. Eight were used as controls; the remainder were experimental. The animals were matched in control and experimental groups according to weight.

The second group was composed of 22 male rats approximately 9 months of age of Sprague-Dawley strain. They were not litter mates. Their weights varied from 370 to 450 gm. The 4 rats used as controls averaged 404 gm. in body weight; 18 experimental animals averaged 402 gm. An additional group of 12 rats of the same lot was continued on stock rations and served as further controls.

The same purified diet and crystalline vitamin supplements were used as in previous thiamine-heart experiments (*1, 2*). The diet was composed of casein, leached and alcohol-extracted, 18 percent; sucrose, 73 percent; cod liver oil, 2 percent; cottonseed oil, 3 percent; and salt mixture (O & M), 4 percent. The vitamin supplement was given

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

daily in the following amounts: Pyridoxine, 20 micrograms; ribo-flavin, 50 micrograms; calcium pantothenate, 50 micrograms; nicotinic acid, 1 mg.; and choline, 20 mg. Control animals received 100 micrograms of thiamine daily; the experimental groups received no thiamine.

Experimental rats were continued on this thiamine-deficient regimen until acute symptoms of deficiency developed, at which time they were treated with 50 to 100 micrograms of thiamine subcutaneously and then allowed to develop another acute episode.

Electrocardiograms were taken at the start of the experiment and at biweekly intervals thereafter until the signs of acute, severe thiamine deficiency began to appear. At this time electrocardiograms were taken at frequent intervals before and after therapy.

The apparatus and technique used to secure these electrocardiograms have been fully described in a previous paper (4). Briefly, the apparatus consisted of a radio amplifying unit attached to an ordinary string galvanometer electrocardiograph. The three standard leads were taken, using fine copper wires as electrodes. The sensitivity was 1 mv=2 cm., and the camera speed 75 mm. per second.

NORMAL RAT ELECTROCARDIOGRAM

The general contour of the tracings obtained from these rats was similar to that previously described for young rats (4). In other respects, however, they were different. The heart rate of the adult animals was slower; the PR and QRS intervals were longer. Furthermore, the male rats differed significantly from the female rats in these adult groups, the males having a slower heart action with longer electrical intervals. Table 1 summarizes the various measurements of the control animals at different ages. At least part of the gradual slowing of the heart rate was due to the lessened excitability of the animals as they became accustomed gradually to the mechanical procedure of taking the tracings. Figures 1 and 11 are normal electrocardiograms.

RESULTS

Body Weight.—After an average of 2 weeks, the females receiving no thiamine began to lose weight. In an average of 34 days (variation 25–40), when they developed their most acute deficiency, the females had lost from an average of 195 to 114 gm. Following treatment with 50 micrograms of thiamine they gained up to 129 gm. In an average of 19 days (variation 14–24) after treatment, they developed their second acute deficiency and had an average body weight of 95 gm. The few which were treated and survived after thiamine was given for this second deficiency gained up to an average of 107 gm. before they were killed at the termination of the experiment.
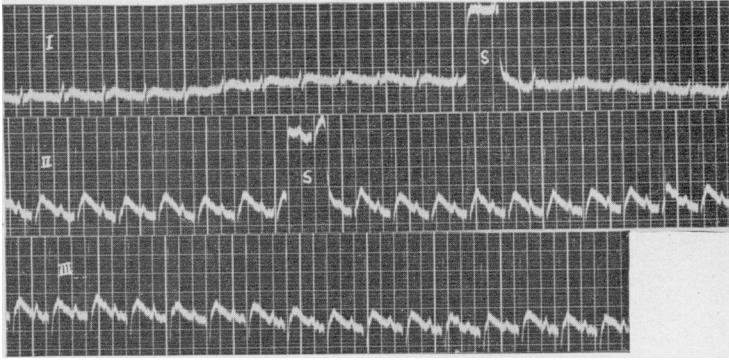
FIGURE 1.—Control rat (20785), 73 days, normal tracing, rate 510/min. PR 0.042 second; QRS 0.013 second; S1 and S3 are present. S=standardization; 1 mv=2 cm. I, II, and III refer to the respective leads.
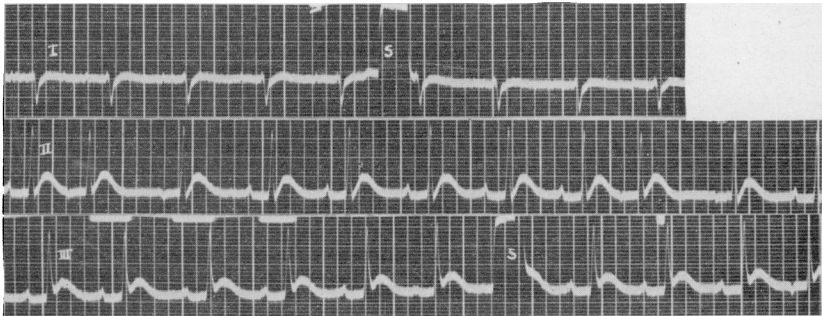
FIGURE 2.—Rat No. 20953 early in second acute deficiency; rate 280, PR 0.07, QRS 0.015. Moderate sinus arrhythmia. P waves variable in shape and direction in all leads. The second, third, ninth, and tenth beats in lead II are auricular ectopics.

FIGURE 3.—Rat No. 20953, 24 hours after tracing in figure 2. Second acute deficiency. Rate 213; PR 0.06–0.105; QRS 0.018–0.025. Note premature beats and altered P waves in lead II. Idioventricular rhythm starts in lead III. (Each of the spaces separated by the light vertical lines equals 0.04 seconds; each of the light horizontal lines equals 1 millimeter.
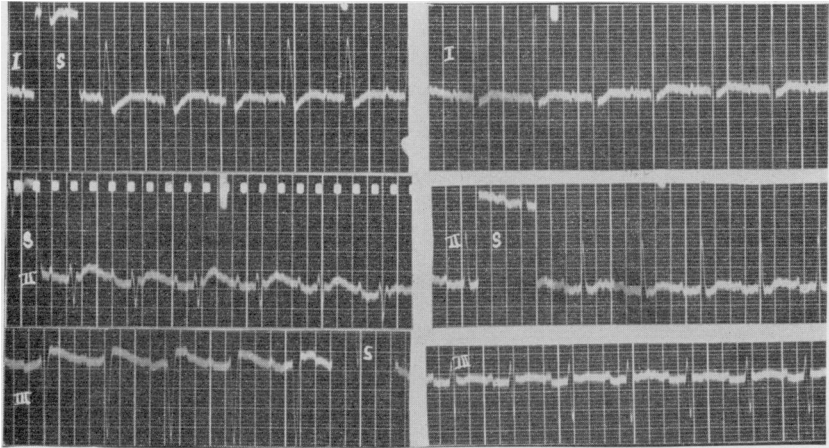
FIGURE 4.—Rat No. 20950. The above tracings were taken after 2 weeks on the thiamine-deficient diet. The tracings on the left show 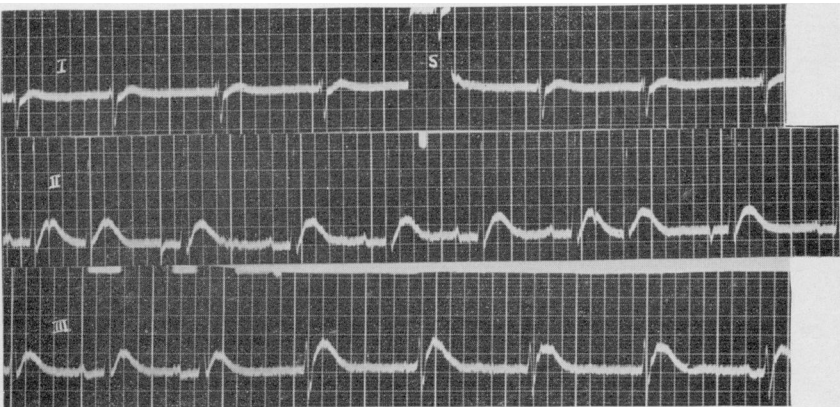an intraventricular conduction defect (QRS 0.02 second). The tracings on the right were taken a few minutes later, after the left vagus nerve had been cut. Note the change in the width and direction of the initial deflections.
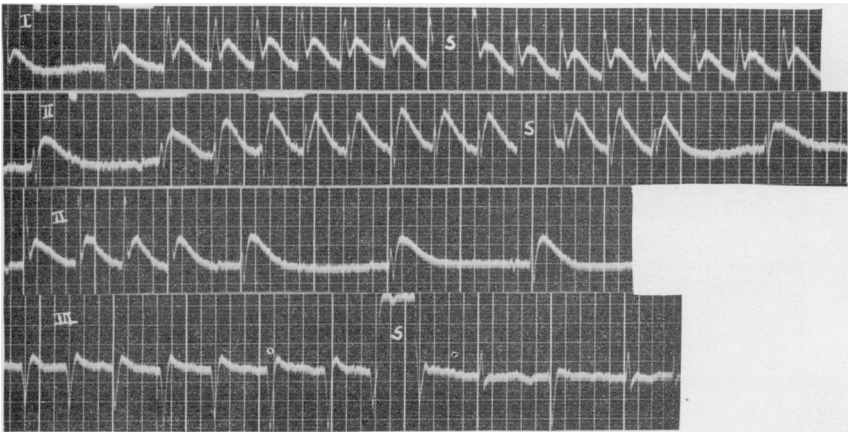
FIGURE 5.—Rat No. 20950 (same rat as above). First acute deficiency. There is a marked arrhythmia due to strings of A–V nodal ectopic beats with an intraventricular conduction defect. Note the variable initial deflections.
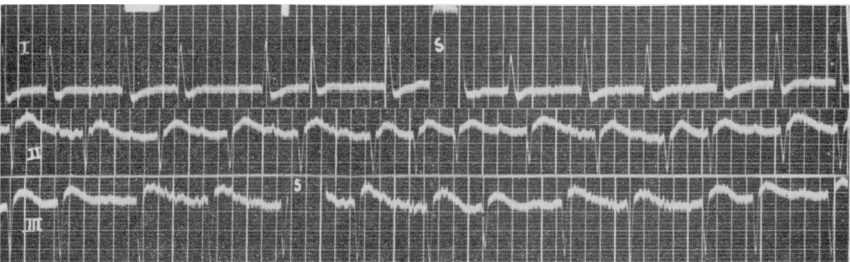
FIGURE 6.—Same rat as above, early in second acute deficiency. Initial deflection is somewhat wide and variable. Occasional distinct P waves can be seen. The rhythm is very irregular, due to A–V nodal ectopic beats.

FIGURE 7.—Rat No. 20950, in second acute deficiency. Rate 320 per minute. Initial deflections wide and quite variable. T3 is inverted. The rhythm is grossly irregular. No P waves are seen (muscular interference makes positive identification of P waves difficult). This tracing was interpreted as auricular fibrillation (see fig. 8).
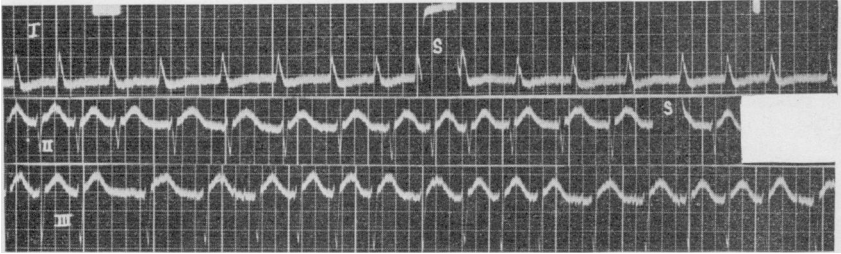
FIGURE 8.—Rat No. 20950 after treatment with 100 micrograms of thiamine daily for 10 days following second acute deficiency. Auricular fibrillation is still present. Heart rate has increased to 430 per minute. P waves absent. T3 now upright. Initial deflections are uniform but still wider than normal.
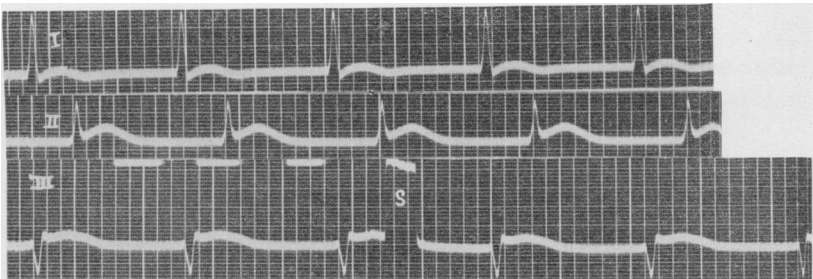
FIGURE 9.—Rat No. 20951, 24 hours after treatment for second acute deficiency. Auricular standstill with idioventricular rhythm (initial deflection 0.028 second). Note regular, very slow rhythm (138 per minute), and complete absence of P waves. This rat showed auricular fibrillation with an intraventricular conduction defect before thiamine therapy.
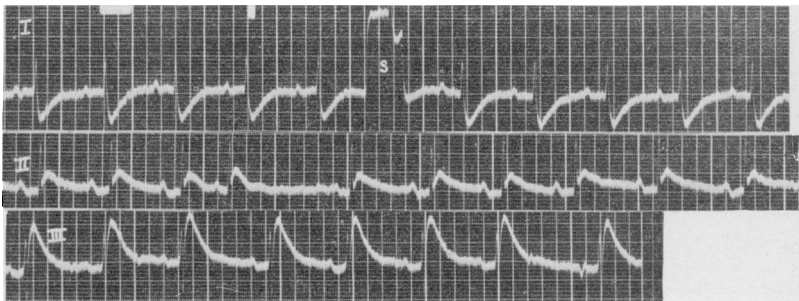
FIGURE 10.—Rat No. 20795. Second acute deficiency; rate 295 per minute; PR 0.06 second; QRS 0.011 second; T1 inverted and low take-off; T3 high take-off, P3 variable; auricular premature beat in lead II.
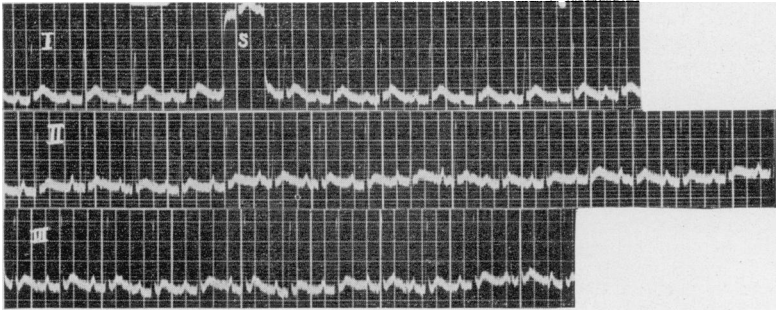
FIGURE 11.—Rat No. 20786 (control rat). Normal tracing. Regular, normal sinus rhythm. Rate 460 per minute ; PR 0.04 second ; QRS 0.009 second.
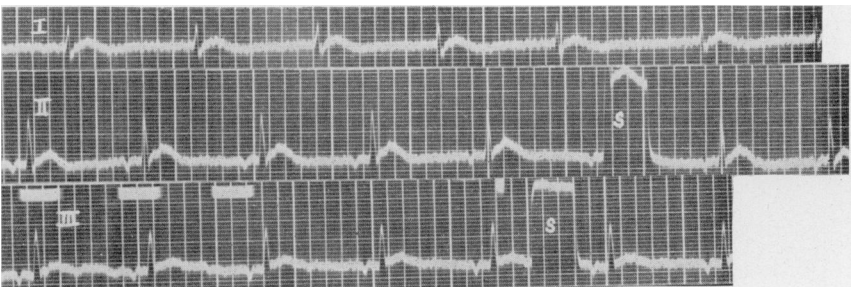
FIGURE 12.—Rat No. 20792. First acute deficiency. Moderate sinus arrhythmia. PR 0.05 second ; QRS 0.017 second ; P2, 3 inverted.
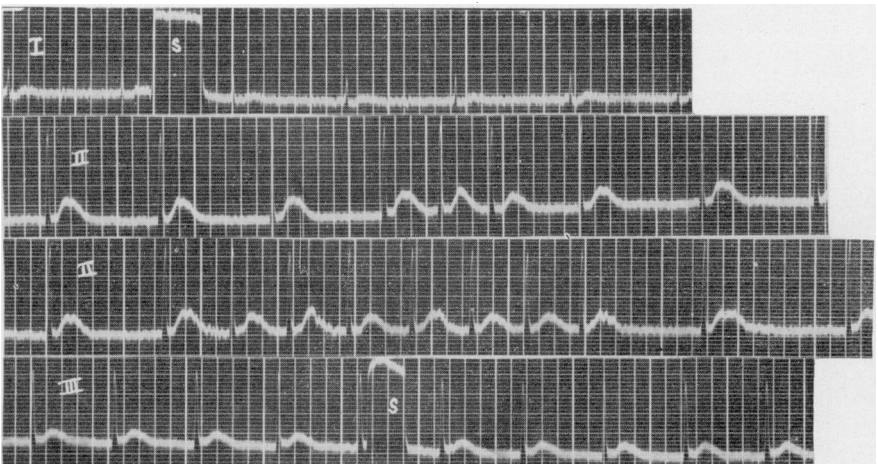
FIGURE 13.—Rat No. 20422. First acute deficiency. Auricular fibrillation. Heart rate 204–333 per minute. Occasional F waves can be seen. After thiamine therapy a normal sinus mechanism returned.

PLATE V
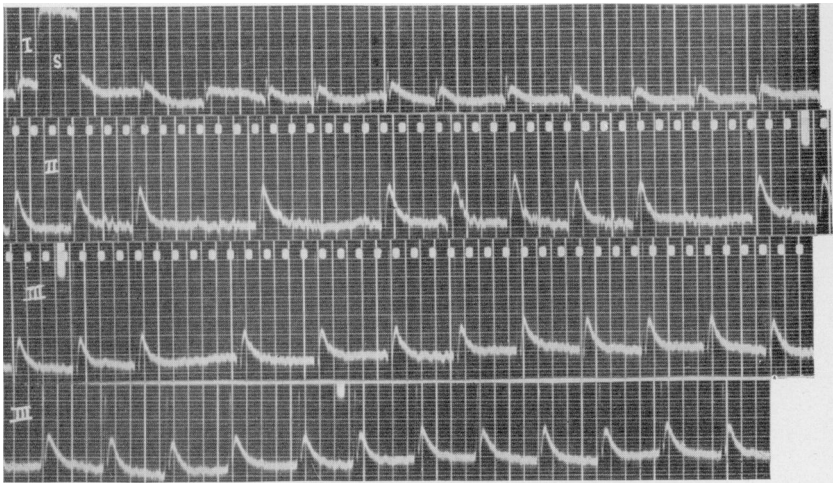
FIGURE 14.—Rat No. 20801 early in first acute deficiency. Auricular fibrillation. Heart rate (approximately) 350 per minute (see figure 15).
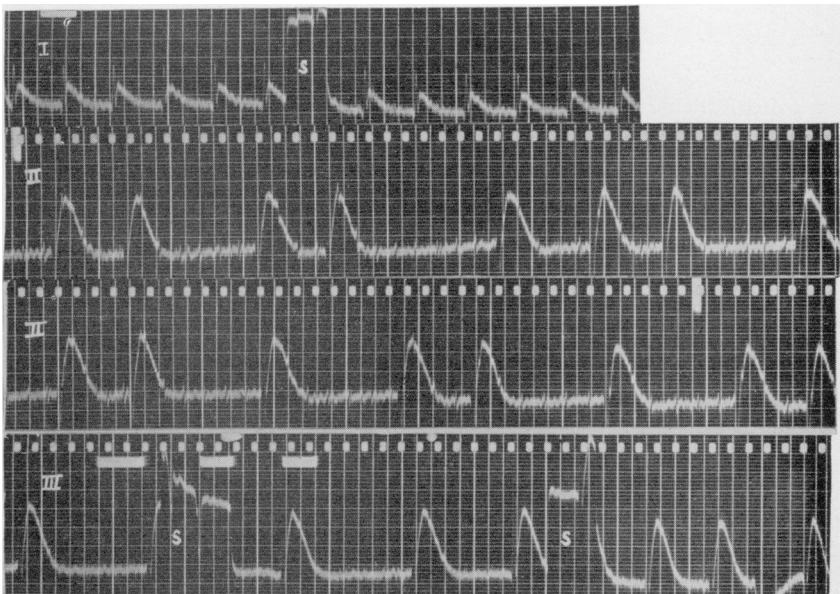
FIGURE 15.—Rat No. 20801. First acute deficiency, 24 hours after figure 14. Auricular fibrillation. Heart rate 200–430 per minute. Note tendency to coupling of beats. Most of the roughness in this tracing is due to muscle spasticity. After thiamine was given the rhythm became regular, with a first degree A–V block (PR 0.08 second), and inverted T1, 2, 3.

TABLE 1.—*Electrocardiographic values for control rats at various ages*

	Start (9 months)				9½ months				10 months				11 months				11½ months			
	Rate[1]	PR	QRS	QT	Rate	PR	QRS	QT	Rate	PR	QRS	QT	Rate	PR	QRS	QT	Rate	PR	QRS	QT
Females:																				
Average	509	0.041	0.011	0.061	491	0.044	0.012	0.064	473	0.043	0.012	0.064	-----	-----	-----	-----	484	0.043	0.011	0.064
Variation	{430–.560	.039–.041	.01–.013	.053–.07	470–.540	.04–.05	.011–.014	.06–.07	430–.510	.04–.05	.011–.013	.06–.067	-----	-----	-----	-----	450–.540	.039–.05	.009–.013	.06–.07
Males:																				
Average	450	.046	.012	.066	-----	-----	-----	-----	416	.046	.012	.075	435	0.044	0.012	0.07	-----	-----	-----	-----
Variation	{430–.500	.04–.055	.011–.013	.06–.07	-----	-----	-----	-----	400–.450	.041–.052	.011–.012	.068–.095	425–.440	.04–.05	.011–.013	.068–.073	-----	-----	-----	-----

[1] Heart rate is given in beats per minute. Other values are given in seconds.

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The males began to lose weight after 5 to 10 days on the thiamine-free regimen. After an average of 39 days (variation 33–43) at which time they were in acute, severe deficiency their average body weight had declined from 402 to 200 gm. After thiamine therapy (50–100 micrograms), the average weight rose to 257 gm., but 16 days (variation 15–19) later had fallen to 199 gm. at which time the animals were in their second acute deficiency. Treatment after this deficiency caused body weights to rise to 248 gm. (average).

Two animals, one male and one female, both in the control groups, developed extensive pneumonias and died (or were killed). The pneumonias were confirmed by autopsies. The remainder of both groups of controls maintained or gained in body weight, seemed healthy in all respects, and showed no gross pathology when autopsied at the conclusion of the experiment.

Signs of acute deficiency.—In order to bring out any cardiac changes that might be developing, it was hoped that these rats should become as thiamine-deficient as possible before treatment was given. With young rats acute polyneuropathy makes a convenient end point. With the older rats, however, polyneuropathy seldom occurred in their first deficiency. None of the male rats showed polyneuropathy (spasticity, ataxia, convulsions), and only 8 of the females were observed to develop polyneuropathy in their first deficiency. Consequently, it was necessary to judge the degree of deficiency by loss of body weight, heart rate, weakness, and changes in spontaneous activity. Unfortunately, these signs were neither consistent nor easily evaluated, and several animals died before electrocardiograms were taken; others became so deficient that they would not respond when thiamine was given, while others were treated too early.

In their second deficiencies nearly all the animals, both male and female, showed acute polyneuropathy, and thiamine therapy was more uniformly successful.

Heart rate.—The heart rates of the control animals are shown in table 1. Rates of both the experimental groups remained at approximately the control level until 7 to 10 days before acute deficiency occurred, even though the animals had been losing weight steadily for 2 weeks or more.

As the deficiency became more severe, the heart rates gradually slowed to an average of 285 (variation 155–346) in the females, and 263 (variation 187–346) in the males at the time thiamine therapy was given. Following treatment the heart rate responded well in most of the females, reaching an average of 457 (variation 390–580), 24 to 72 hours after therapy. Males responded less well, reaching an average of 371 (variation 246–450).

In their second deficiency the average heart rate of the females reached 294 (variation 250–360) and rose to 444 after thiamine was given. In the males the average heart rate in their second deficiency was 243 (variation 110–320), rising to 354 (variation 138–425) after treatment.

The heart rates as well as other data for individual animals are given in tables 2 and 3.

Rhythm.—The regular rhythm shown by these rats in the early part of the experiment became disturbed in a variety of ways as the deficiency progressed (see tables 2 and 3). Some degree of sinus arrhythmia was common (figs. 2 and 12). Marked sinus arrhythmia with 50–75 percent variations in successive PR intervals was seen in a few of the animals. Sinus arrest was also common, being followed by ectopic beats of auricular or A-V nodal origin. In a few animals premature beats of auricular or A-V nodal origin occurred (figs 2, 3, 5, 6, 10). Ectopic beats, sinus arrest, and sinus arrhythmia usually responded quickly and completely to thiamine administration.

Auricular fibrillation (figs. 13, 14, 15) occurred in two of the females in their first deficiency. Thiamine treatment converted one to a normal sinus mechanism, in the other the animal died in spite of treatment. In the males, auricular fibrillation was found three times (figs. 7, 8). In one rat (20965), this arrhythmia occurred in the first deficiency episode and the animal did not respond to thiamine treatment. Two males in their second deficiency (20950, 20951) showed auricular fibrillation. In one of these, thiamine was administered in 100-microgram doses daily for 10 days without altering this abnormal mechanism, although the heart rate increased considerably (fig. 8). In the other rat with auricular fibrillation, thiamine treatment apparently converted the auricular fibrillation to auricular standstill with an idioventricular rhythm (fig. 9).

Electrical axis.—No consistent axis deviation could be demonstrated in any stage of the deficiencies. Many of the male rats, both control and experimental, showed a tendency to left axis deviation.

PR interval.—An increase in the PR conduction time was noted in some animals early in their deficiency before their heart rates had declined appreciably. The figures given in parentheses in table 3 are values obtained after the rats had been on the thiamine-deficient regimen for only 2 weeks (fig. 4). It will be noted that several of the PR intervals are increased as compared to normal values for similar animals (table 1).

During the stage of acute deficiency, just before treatment was given, a marked delay in the PR interval of many of these rats was noted, the values ranging from 0.06 to 0.10 seconds (figs. 2, 3, 10).

TABLE 2.—*Summary of electrocardiographic findings in thiamine-deficient adult females*

Rat No.	During first deficiency [1]					During second deficiency				
	Heart rate	PR	QRS	Other findings	After treatment—findings	Heart rate	PR	QRS	Other findings	After treatment—findings
20790	290 (490)	0.055 (.043)	(?) (.011)	T2, 3 inverted	Died					
20791	333 (560)	.05 (.041)	0.015 (.01)	QRS wide	Rate 420; PR 0.051; QRS 0.011.	250	0.053	0.011	Sinus arrest; auricular and A-V nodular ectopic beats.	Rate 495; PR 0.039; normal sinus rhythm.
20792	195-321 (560)	.05 (.041)	.017 (.013)	Moderate sinus arrhythmia; P2, 3 inverted, QRS wide.	Died					
20793	321 (475)	.048 (.045)	.013 (.012)	T2, 3 inverted	Rate 410; PR 0.048; T2, 3 upright.	280	.042	.012	T2, 3 inverted	Rate 420; PR 0.042; T2, 3 upright.
20794	284 (450)	.045 (.04)	.012 (.013)	Normal	Rate 440; PR 0.041, normal.	280	.05	.012	T1, 2, 3 inverted	Died.
20795	340 (450)	.067 (.043)	.016 (.013)	Rhythm irregular; sinus arrest; P2, 3 variable shape and direction.	Rate 450; PR 0.055; QRS 0.008; P3 inverted; otherwise normal.	295	.06	.011	T1 inverted; T3 high; P3 variable; auricular premature beats.	Rate 375; PR 0.05, normal.
20800	250 (500)	.07 (.043)	.013 (.012)	P waves low, variable in shape and direction.	Rate 580; PR 0.05; P2 inverted.	300	.067	.013	T2, 3 inverted.	Died.
20801	200-430 (530)	(.047)	.011 (.013)	Auricular fibrillation, high T waves.	Rate 510; PR 0.08; T1, 2, 3 inverted, regular rhythm.				Died.	
20802	346 (495)	.055 (.041)	.015 (.013)	QRS wide.	Rate 470; PR 0.038, normal.					
20803	300 (430)	.042 (.042)	.013 (.01)	Normal	Rate 390; PR 0.043, normal.	265-310	.063	.012	Rhythm very irregular; strings of A-V nodal extrasystoles; P1, 2, 3±.	Rate 410; PR 0.052; regular rhythm; P waves still variable.
20422	204-333 (440)	(.053)	.013 (.013)	Auricular fibrillation	Died.				do	
20423	155 (500)	.095 (.05)	.02 (.013)	PR increase; QRS wide	...do...					
20425	290 (416)	.065 (.05)	.02 (.013)	Rhythm irregular; PR variable; strings of A-V nodal and auricular extrasystoles.	Rate 430; PR 0.043; QRS 0.013; regular rhythm.	360	(?)	(?)	Same as first deficiency	Died.

[1] The figures given in parentheses are normal values obtained at the start of the experiment from the same animal. Animals that died without electrocardiograms being obtained at the point of greatest deficiency are not included here.

TABLE 3.—*Summary of electrocardiographic findings in thiamine-deficient adult males*

Rat No.	During first deficiency [1]					During second deficiency				
	Heart rate	PR	QRS	Other findings	After treatment—findings	Heart rate	PR	QRS	Other findings	After treatment—findings
20930	225 (440)	0.07 (.049)	0.018 (.013)	T1, 2, 3, inverted; ST1, 2 depressed.	Died					
20931	255 (440)	.069 (.055)	.012 (.013)	Normal except for PR increase.	---do---					
20932	250 (450)	.068 (.042)	.012 (.012)	T2 inverted; T3 diphasic.	---do---					
20940	144–220 (480)	.05–.06 (.042)	.012 (.011)	Marked sinus arrhythmia; A-V nodal premature beats; P2 variable±.	Killed					
20941	264 (450)	.063 (.05)	.013 (.013)	PR increase only.	Rate 430; PR 0.055 normal sinus rhythm.	290	0.07	0.011	Marked sinus arrhythmia; P2, 3 variable±.	Rate 425; PR .052 P2 still variable.
20942	231 (450)	.08–.10 (.048)	.025 (.016)	Bundle branch block; T1, 2 high; R3 notched.	Killed					Died.
20943	346 (460)	.046 (.045)	.011 (.012)	Normal sinus rhythm.	Rate 450; PR 0.05 normal sinus rhythm.	280	.058	.012	T1, 2, 3, inverted; ST1, 2, 3 depressed.	Died.
20950	166–460 (430)	.058 (.055)	.016–.018 (.02)	P2, 3 variable±; strings of A-V nodal premature beats.	Rate 421; QRS 0.018; P waves flat; R2, 3 variable and notched.	320		.019	Auricular fibrillation QRS variable in shape T3 inverted.	Rate 430; QRS 0.015; fast auricular fibrillation.
20951	281 (450)	.052 (.054)	.012 (.011)	Normal sinus rhythm.	Rate 246; moderate sinus arrhythmia; T3 variable±	110		.018	Auricular fibrillation ventricular conduction defect.	Rate 138; QRS 0.028 auricular standstill; Idioventricular rhythm.
20952	273 (445)	.052 (.045)	(?) (.01)	Normal sinus rhythm.	Died.					
20953	346 (440)	.066 (.06)	.016 (.015)	PR increase QRS wide.	Rate 410; PR 0.065; QRS 0.017.	213	.07–.10	.018–.025	Shifting pacemaker; P2, 3 variable; auricular premature beats; idioventricular rhythm lead 3.	Rate 420; PR 0.065 QRS 0.015; regular rhythm.
20964	273 (475)	Variable (.04)	.014 (.011)	PR 0.039 lead 2, 0.05 lead 3; P2 inverted.	Died.					
20965	250 (490)	---(.05)	.015 (.012)	Auricular fibrillation T2 diphasic, T3 inverted.	---do---					

[1] The figures in parentheses are values taken 2 weeks after the rats had been deprived of all thiamine. Only those animals which had electrocardiograms taken in their first acute deficiency episode are included here.

The upper limit of normal for the females was 0.05 seconds, and for the males 0.055 seconds. Higher degrees of blocking with dropped beats or complete block were not observed, however.

In a few rats, (20940, 20942, 20953, 20964), the PR intervals varied considerably from beat to beat.

Thiamine administration effected a prompt return of the PR interval to normal values in most instances.

QRS interval.—Three of the male rats showed increases in the width of their QRS complexes after only 2 weeks on experiment (see table 3, figures in parentheses), reaching values of 0.015, 0.016, and 0.02 seconds (upper limit of normal 0.013). In the rat with a QRS of 0.016 (20942), cutting the right vagus was without effect. In the rat with a QRS of 0.02 (20950), sectioning the left vagus decreased the QRS interval to 0.015 seconds (fig. 4). The rat with a QRS of 0.015 was not treated. Bilateral vagus sectioning was not done in these animals since it invariably has resulted in death from laryngeal collapse in other rats, and it was desired to save these rats to study their QRS pattern in more severe deficiencies.

Seven of the males and six of the females showed widened QRS complexes varying from 0.014 to 0.25 seconds at the time of their most acute deficiency (figs. 2, 3, 9, 12). In most instances there was only widening of the QRS complex, the direction and amplitude of the waves being normal. On one rat, however, 20942, which had shown a PR increase early in the experiment, the QRS interval increased to 0.025 seconds, R_1 increased from 3 mm. to 22 mm. in height, and R_3 changed from $+6$ mm. to -9 mm., indicating aberrant ventricular conduction. In two rats an idioventricular rhythm with widened QRS complexes developed (figs. 3, 9).

Thiamine administration had little effect on the widened QRS complexes in the males but returned those of the female rats to normal fairly consistently.

QT interval.—No changes in the QT intervals were noted except for those associated with changes in heart rate.

P waves.—P-wave abnormalities were observed in four of the females and six of the males (figs. 2, 3, 10, 12). Most often they were inverted, or varied from positive to negative, sometimes they were abnormally wide, or splintered and occasionally biphasic. Sometimes variable P waves were associated with variable PR intervals (fig. 3). Successive P waves often showed variation in their contour. One of the rats (20950) with variable P waves later developed auricular fibrillation. P-wave changes responded very irregularly to thiamine.

T waves.—Six of the females and seven of the males showed T-wave changes (figs. 7, 10, 15). Most of these were simple inversion or

diphasism. However, two of the females and one of the males showed abnormally high T waves in one or more leads, and one of the males showed a T_3 which was variably positive and negative after thiamine therapy. Most of the rats with T-wave changes died in spite of thiamine therapy. The T waves of the few that lived responded irregularly to thiamine.

The take-off of the T waves was abnormally low in two of the males. Both animals died after thiamine was given. No significance was attached to a moderately elevated T-wave take-off since normal rats often show this phenomenon.

<div align="center">DISCUSSION</div>

Most studies of thiamine deficiency in experimental animals have shown significant cardiac changes only when the animals were kept for relatively long periods on a low thiamine intake. Thus Weiss, Haynes, and Zoll (5, 6) accidentally produced a chronic partial deficiency followed by an acute deficiency when they failed initially to autoclave their yeast long enough to destroy the thiamine. These rats showed electrocardiographic changes. Swank, Porter, and Yeomans (7) found no cardiac failure in acutely deficient dogs but did find cardiac failure and electrocardiographic as well as pathologic changes in the myocardium of chronically deficient dogs. Swank and Bessey (8) found electrocardiographic changes and heart damage only in pigeons chronically deficient in thiamine. Wintrobe et al. (9, 10) found pathologic and electrocardiographic changes in both acute and chronic thiamine deficiency in swine. Lowry et al. (1) and Ashburn and Lowry (2) observed pronounced pathologic changes in rats chronically deficient in thiamine. Other reports from this laboratory on the electrocardiographic findings in thiamine deficiency (3, 4) utilized chronically deficient rats.

Each of the above studies used young or relatively young animals. The study reported here shows definitely that electrocardiographic changes do occur in acute thiamine deficiency and in older animals. On the average the changes are somewhat more severe and less amenable to treatment than the changes observed in chronically deficient young rats (4), although the basic characteristics of the electrocardiographic manifestations are the same.

It might be expected that the hearts of older rats would be more susceptible to the deleterious effects of thiamine deficiency since studies have shown that the rat's heart spontaneously acquires various types of cardiovascular diseases as the rat ages (11). This is quite similar to the picture in man where it is generally accepted that the heart becomes more susceptible to injury and disease with age.

The possible significance of the electrocardiographic abnormalities which occur in thiamine-deficient rats has been discussed in a previous publication (4).

SUMMARY

Nine-month-old rats developed a variety of electrocardiographic defects when they became acutely deficient in thiamine.

The changes included bradycardia, sinus arrhythmia, sinus arrest, ectopic beats, delayed auriculoventricular conduction (PR), widened QRS complexes, bundle branch block, auricular fibrillation, idioventricular rhythm, notched, inverted, or otherwise variable P waves, inverted or diphasic T waves, and depression or elevation of the T-wave take-off.

The response of these changes to thiamine therapy was variable. Some revert promptly and completely, others slowly or not at all.

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STUDIES OF THE ACUTE DIARRHEAL DISEASES

XVII. THE SULFONAMIDES IN SHIGELLOSIS [1]

By ALBERT V. HARDY, *Surgeon (R)*, *United States Public Health Service*

The relative activity of sulfonamides in shigellosis as observed in treated cases and carriers has been described in preceding reports (*1, 2*). In vitro studies of sulfonamide sensitivity and determination of sulfonamide levels in the blood and feces of some of the treated patients are discussed in the present report.

IN VITRO TESTS OF SULFONAMIDE SENSITIVITY

The test medium employed was a 1-percent tryptone broth. The sulfonamides were added in the following concentrations: 0.01, 0.05, 0.1, 0.15, 0.25, 0.5, 0.75, 1, 2, 3, 5, 7.5, 10, 15, 25, 50, 75, 100, 150, 200, 300, and 400 mg. per 100 ml. (The higher concentrations of the less soluble compounds were omitted.) An 18-hour broth culture was diluted to an estimated 1,000 organisms per milliliter and 0.1 ml. was used as the inoculum. The actual number of organisms in the inoculum was determined by plate counts. Tests were read after 24- and 48-hour incubation. The amount of growth at 24 hours was estimated by comparison with that in the sulfonamide-free control tube and was recorded as $4+, 3+, 2+, 1+,$ and $\pm$ (ranging from a turbidity equaling that in the control to a questionable beginning clouding). The turbidity standard which matched the control tube at the end of 24 hours was used as the basis of comparison in reading the tests at the end of 48 hours.

In testing the relative sensitivity of organisms isolated from different outbreaks, sulfadiazine only was used. Table 1 shows that the usual

TABLE 1.—*Lowest concentrations of sulfadiazine causing, in vitro, bacteriostasis of different strains of* Shigellae [1]

Sulfadiazine level in mg. per 100 ml.	Number and percent of strains with no growth or a questionable growth at 24 hours							
	Flexner		Sonne		Schmitz		Shiga	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
0.01–1	112	86	9	6	5	42	3	43
1.5–5	15	11	56	40	7	58	2	29
7.5–15	4	3	47	34	0	0	1	14
25–50	0	0	27	20	0	0	1	14
Total	131	100	139	100	12	100	7	100

[1] Flexner and Sonne strains isolated from cases or carriers which failed to respond to chemotherapy are excluded.

strains of Flexner organisms were highly sensitive. The growth of some was markedly inhibited by as little as 0.1 mg. of sulfadiazine per 100 ml. and 86 percent were inhibited by 1 mg. or less per 100 ml. The highest concentration of sulfadiazine required for marked bacteriostasis was 10 mg. per 100 ml.; in the 4 tests with growth at this level, the inoculum contained substantially more than the desired 100 organisms. The Sonne strains were notably more resistant. All but 6 percent required more than 1 mg., and 54 percent required more than 5 mg. per 100 ml. for the same degree of bacteriostasis. The Schmitz and Shiga strains were intermediate in their sensitivity as is shown in the table. In the treatment of cases and carriers, the usual Flexner (including Boyd 88) organisms were highly sensitive to sulfonamides, the Schmitz variety was a little more resistant; and Sonne strains clearly more resistant. We have not treated Shiga cases.

There was a wide difference between the usual strains and those isolated from cases or carriers which failed to respond to sulfonamide therapy. Ninety-eight of these resistant strains (31 Flexner and 67 Sonne) were tested. These grew with little if any inhibition in 100 mg. of sulfadiazine per 100 ml.

The relative activity of 8 sulfonamides against 20 *Shigellae* (7 Flexner, 6 Sonne, 4 Schmitz, 3 Shiga) was compared as follows: Identical series of dilutions of all sulfonamides were prepared and were inoculated similarly within the hour. The amount of growth at 24 and 48 hours, when present, was measured by comparison with the same turbidity standard. The response to sulfathiazole (the most active compound) was used in computing the relative levels which gave equal bacteriostasis. For example, if there was no growth at 24 hours and a 2+ growth at 48 hours at a level of 0.25 mg. of sulfathiazole and at a level of 0.5 mg. of sulfadiazine, then the relative levels giving equal bacteriostasis would be sulfathiazole 1 and sulfadiazine 2. The findings with the computed averages are given in table 2. This shows that an average of 1.8 times as much sulfadiazine, twice as much sulfamerazine, and 60 times as much sulfaguanidine as sulfathiazole was required for equal bacteriostasis of *Shigellae*. Since there were these demonstrable variations in bacteriostatic activity in vitro, the sulfonamide levels observed in patients may be weighted according to their relative potency. The factor used for this purpose is given in the last column of the table.

SULFONAMIDE LEVELS IN BLOOD AND FECES

The Bratton and Marshall procedure (*3*) was used for the sulfonamide determinations, with one modification for the tests on feces. It was noted that a turbid filtrate was obtained with the usual technique

TABLE 2.—*Relative in vitro bacteriostatic activity of sulfonamides against different varieties and strains of Shigellae*

Sulfonamide	Relative sulfonamide levels giving equal bacteriostasis of different varieties and strains of *Shigellae*																				Average	Reciprocal of average [1]
	Flexner							Sonne						Schmitz				Shiga				
	1	2	3	4	5	6	7	1	2	3	4	5	6	1	2	3	4	1	2	3		
Sulfathiazole	1.5	1.0	1.0	1.5	1.0	1.3	4.5	1.0	1.3	1.5	2.0	1.0	1.5	1.5	1.0	1.0	2.5	2.0	3.0	2.0	1.8	1.0
Sulfadiazine	1.0	2.0	1.5	1.0	1.0	2.5	1.5	.8	.9	1.0	1.5	.9	1.0	1.0	.5	3.0	1.2	2.0	1.0	1.0	1.2	.556
Sulfapyrazine	1.0	1.1	2.0	1.0	1.0	6.7	2.0	1.0	2.0	3.0	1.5	.9	1.5	1.2	1.0	1.5	2.5	4.0	1.2	3.0	2.0	.833
Sulfamerazine	1.0	2.0	2.0	1.0	1.2	1.2	6.0	1.5	2.3	6.7	4.0	2.7	6.0	5.0	7.5	2.8	3.0	4.0	3.0	3.0	2.5	.500
Sulfamethazine	1.0	3.0	3.0	3.3	2.8	6.7	6.0	1.5	2.3	10.0	10.0	6.7	6.0	10.0	7.5	2.8	3.0	5.0	3.0	3.0	3.5	.286
Sulfapyridine	9.0	21.0	15.0	5.2	5.2	6.7	6.0	5.0	10.0	10.0	10.0	6.7	6.0	10.0	7.5	2.8	3.0	5.0	3.0	3.0	9.1	.110
Sulfanilamide	35.0	100.0	20.0	50.0	33.3	66.7	100.0	50.0	50.0	100.0	75.0	----	50.0	20.0	10.0	75.0	100.0	100.0	50.0	50.0	70.8	.014
Sulfaguanidine	100.0	100.0	20.0	50.0	33.3	66.7	100.0	----	75.0	100.0	75.0	----	26.0	20.0	10.0	100.0	100.0	100.0	33.3	50.0	60.7	.016

[1] These factors are used to "weight" the blood and fecal levels according to potency as shown in table 3.

except in stools containing mucus. This led to the addition of egg (beaten yolk and white) to the saponin solution in sufficient amount to give a heavy floccular precipitate on the addition of the acid. The resulting filtrate was either clear or only slightly turbid. Any residual turbidity was measured on the colorimeter and final readings adjusted accordingly. This procedure accurately determined the amount of sulfonamide added to sulfonamide-free fluid feces.

We obtained for examination, not the relatively dry fecal residue, but the soft or fluid feces following a saline laxative. The specimens were collected by aspiration through a rectal tube, thus excluding any possibility of urinary contamination. The blood specimen was drawn shortly after the collection of the fecal specimen. Comparative levels were determined in 358 cases.

Routinely, each fecal specimen was tested in two ways designed to measure respectively the dissolved free and the total free sulfonamide. The first determination was obtained by examining the supernatant fluid of a centrifuged specimen; the second was obtained by using a portion of the whole mixed feces. The former is regarded as the measure of the amount of therapeutically active sulfonamide.

Differences in the sulfonamide levels in the centrifuged and in the whole fecal specimens are illustrated in figure 1. With the usual daily dosage of these drugs the amount of sulfonamide in the feces was such that only a portion was in solution. With four 1-gm. doses of sulfadiazine daily, the average fecal levels were 47.2 mg. per 100 ml. for the whole specimen and 29.4 for the centrifuged preparation.

SULFONAMIDE LEVELS IN CENTRIFUGED
AND WHOLE FECAL SPECIMENS
DISSOLVED AND TOTAL SULFONAMIDE COMPARED
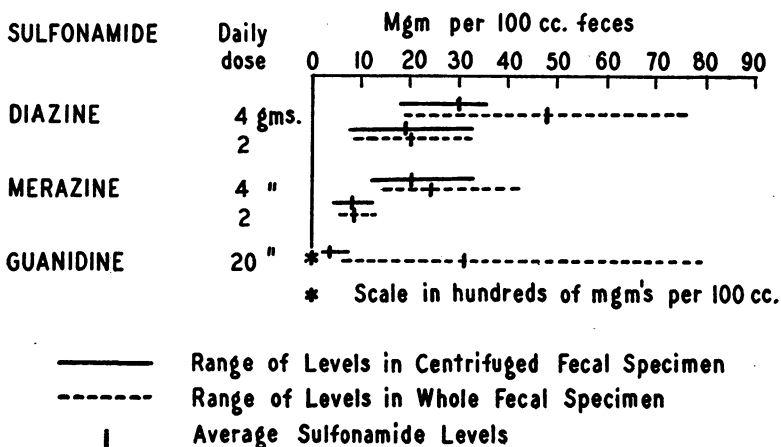
FIGURE. 1.

With the more soluble sulfamerazine, the variation was less. When the total daily dosage was low, all or almost all of these sulfonamides were in solution and the two determinations were closely similar. The sulfaguanidine levels were all high, but there was a wide difference between the total amount and the dissolved portion, as is shown.

The levels of dissolved sulfonamide in fluid feces are compared with the blood levels in figure 2. (The observations on five scattered pairs of specimens were substantially below the usual range in the series. These abberrant findings were excluded since they were thought to be related to the difficulties of administering medication to patients in mental hospitals.) The amount of sulfonamide in the blood varied with dosage in accordance with the known characteristics of the respective agents. The fecal levels were two to eight times higher than the blood levels when absorbable sulfonamides other than sulfanilamide had been given. There were wide differences in the blood and fecal levels when the poorly absorbable sulfonamides were used.

THE RANGE OF AND THE AVERAGE SULFONAMIDE LEVELS IN
BLOOD AND FLUID FECES
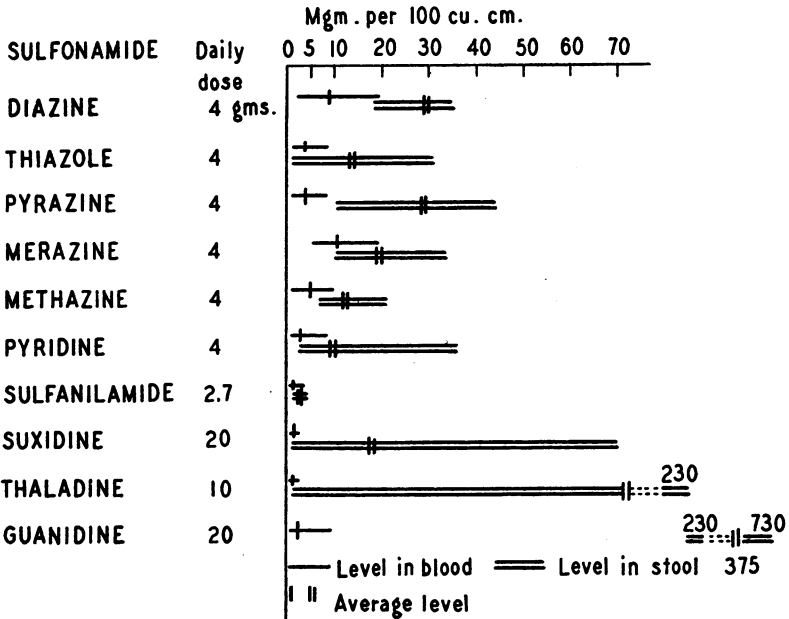
FIGURE 2.

The absorbable compounds with the highest average levels of dissolved sulfonamide in the feces were sulfadiazine and sulfapyrazine. Sulfamerazine, sulfathiazole, sulfamethazine, sulfapyridine, and sul-

fanilamide followed in the order named. The sulfadiazine levels were comparatively uniform from case to case, while there was a marked variation in the levels of sulfathiazole. Patients given sulfasuxidine had moderate but quite variable levels of the dissociated sulfathiazole. The readings were higher in patients who had received sulfathaladine, but these have uncertain significance since sulfathaladine may continue its dissociation in the passed feces. The minimum, average, and maximum fecal levels of sulfaguanidine were all high.

The observed levels need to be evaluated in the light of the demonstrated variations in the bacteriostatic potency of sulfonamides for *Shigellae*. Therefore, the amount of sulfathiazole which would have equal bacteriostatic activity was computed for each finding. The observed and weighted levels are given in the first columns of table 3.

TABLE 3.—*Relative effectiveness of sulfonamides in shigellosis, as indicated by weighted* [1] *sulfonamide levels and as determined in the treatment of cases and carriers*

Sulfonamide	Daily dosage in grams	Average sulfonamide levels in milligrams per 100 ml.						Order of effectiveness in shigellosis	
		Blood		Feces				As indicated by pharmacological findings	As determined in the treatment of cases and carriers
				Average		Minimum			
		Observed	Weighted	Observed	Weighted	Observed	Weighted		
Sulfathiazole	4.0	4.0	4.0	13.0	13.0	4.6	4.6	5	6
Sulfadiazine	4.0	8.0	4.4	29.4	16.3	18.5	10.3	2	2
Sulfapyrazine	4.0	4.2	3.5	29.2	24.3	12.2	10.2	1	1
Sulfamerazine	4.0	10.8	5.6	20.0	10.0	12.5	6.3	4	4
Sulfamethazine	4.0	6.6	1.9	12.7	3.6	7.2	2.1	7	5
Sulfapyridine	4.0	3.5	.4	10.8	1.2	2.0	.2	8	8
Sulfanilamide	2.5	2.7	.04	3.9	.05	2.7	.04	9	9
Sulfaguanidine	20.0	3.5	.06	375.0	6.0	230.0	3.7	6	7
Sulfasuxidine	20.0	.6	.6	19.4	19.4	3.6	3.6	3	3

[1] The factors used to weight the levels according to relative bacteriostatic potency for *Shigellae* are given in the last column of table 2.

Four sulfonamides (sulfapyrazine, sulfathiazole, sulfadiazine, and sulfamerazine) were present in the blood at average levels equivalent to 3.5 to 5.6 mg. of sulfathiazole per 100 ml. These circulating sulfonamides would be expected to be effective for *Shigellae* growing on or in the intestinal wall. It is reasonable to believe that organisms which grow directly on the surface of the mucous membrane, in the crypts, or while actually invading the tissues have major pathogenic importance. The absorbed sulfonamides will reach these through the blood stream and tissue fluids, and, more slowly, from the enteric tract. The sulfonamides with high average weighted levels in the feces were sulfapyrazine, sulfasuxidine, and sulfadiazine. The mini-

mum fecal levels must also be considered since a poor therapeutic response in some individuals could be related to an inadequate concentration of the drug. Sulfadiazine and sulfapyrazine stood above all others with minimum weighted levels above the equivalent of 10 mg. of sulfathiazole per 100 ml. Sulfathiazole, sulfaguanidine, and sulfasuxidine had undesirably low levels in some individuals. According to these data sulfapyrazine and sulfadiazine would be judged the most promising of the absorbable sulfonamides for *Shigella* infections. None of the poorly absorbed compounds had therapeutically effective levels in the blood stream, but sulfasuxidine had a high average fecal level. Sulfaguanidine, in comparison, had low average and minimum weighted fecal levels. The seeming advantage of this compound disappeared when the observed levels were considered in the light of the low activity of the drug.

The order of the effectiveness of the sulfonamides in the treatment of shigellosis is indicated in the last columns of table 3. The rating by the pharmacological findings is based on the total of the three weighted sulfonamide levels as given. The evaluation on cases and carriers was obtained through bacteriological observations on our 1,924 treated infections (*1*, *2*). The findings by these two types of study were in close agreement. Sulfapyrazine and sulfadiazine stand out as the best of the easily absorbed compounds, and sulfasuxidine as the best of the poorly absorbed preparations. Sulfamerazine is next in order of effectiveness while sulfathiazole, sulfamethazine, and sulfaguanidine are the least active of the preparations which might be recommended for shigellosis. Sulfapyridine and sulfanilamide had the lowest ratings.

The relative value of parenterally administered sulfonamides in shigellosis was investigated also. Ten patients were given sodium sulfadiazine and 10 were given sodium sulfapyrazine intravenously. Stool and blood specimens were collected after 4, 8, and 12 hours. At the end of 4 hours the fecal levels were about one-half the blood levels; at the end of 8 hours the blood levels had declined and the fecal levels increased, so all readings were within the same narrow range. At 12 hours the blood levels had declined substantially; the fecal levels only slightly. In only one case in each group did the fecal levels appreciably exceed the 4-hour blood levels. These findings were in marked contrast to a comparable series of cases similarly examined in which the drugs were given by mouth. In these, blood levels were lower and more variable. After 4 hours the concentration of dissolved sulfonamide in the feces varied from less than one-half to more than 5 times that of the blood. The stool levels at the end of 8 hours were all high, varying from 4 to 20 times those of the blood, and at 12 hours these continued to be high. It was evide t that the

high levels of sulfadiazine and sulfapyrazine in the enteric tract were due chiefly to incomplete absorption. Sodium sulfadiazine given parenterally was used in treating 24 children acutely ill with *Shigella* infection. There was a prompt clinical response. While this route of administration may be used to initiate treatment, oral administration with the resulting higher fecal levels is considered superior.

SULFONAMIDE-RESISTANT *SHIGELLAE*

During four of the outbreaks studied, some sulfonamide-resistant *Shigellae* were isolated. There was no apparent spread of the resistant infection in one outbreak and only limited spread in two. Careful isolation was required for control. The outcome was less favorable in the fourth outbreak. Flexner Z infection was first discovered by diagnostic cultures in cases occurring among 32 infants in an institution for the mentally defective. In December 1944, most of these children were ill with acute diarrhea. The symptoms subsided under sulfonamide treatment; the hospital physician discontinued medication on clinical recovery. Neither isolation nor follow-up laboratory examinations were employed. We obtained cultures on all children in the nursery during infrequent visits to the institution. Organisms isolated were examined for sulfonamide sensitivity (there was either marked inhibition by 1 mg. or less of sulfadiazine for 100 ml., or no apparent inhibition by 100 mg.). There were 13 positive cultures on the first survey (December 11, 1944); all organisms were sulfonamide sensitive. Three weeks later (January 5, 1945), 11 or 14 positive cultures were sensitive but 3 were resistant. In the next survey (January 31) 5 of the identified Flexner Z organisms were sensitive and 4 resistant. As of February 13, 1 positive culture was sensitive and 7 were resistant. Subsequently almost all organisms tested were highly resistant. Through transfer from patient to patient this infection was introduced to 3 other groups and spread freely. The sulfonamides were of no value in the treatment or control of this resistant infection. This unusual experience emphasizes the need for appropriate precautions in the use of sulfonamides in shigellosis.

CONCLUSIONS AND RECOMMENDATIONS

In the light of our experience with sulfonamides in shigellosis we have reached the following conclusions:

The sulfonamide of choice for the treatment of shigellosis is sulfadiazine. This drug is effective at a moderate dosage; it has low toxicity and is generally available. Sulfapyrazine also may be highly recommended when available. Of the poorly absorbed sulfonamides sulfasuxidine is the compound of choice.

The recommended dosage of sulfadiazine or sulfapyrazine for adults is 4 gm. daily; of sulfasuxidine 20 gm. daily. These should be given for a minimum period of 7 days, unless the individual has two consecutive negative cultural tests before the end of the period. Smaller doses for a shorter time are effective in most cases, but the larger amounts are recommended to minimize the risk of the development of sulfonamide-resistant organisms.

In acutely ill patients with troublesome vomiting, treatment may be initiated with sodium sulfadiazine given parenterally. Medication should be given by mouth as soon as it can be tolerated.

Where enteric infections spread readily, as among institutional inmates, effective isolation during and following treatment must be required. Patients with infections which fail to respond to sulfonamide treatment need to be isolated with particular care.

Cultures for *Shigellae* should be employed to guide treatment and to regulate isolation. Tests every other day during treatment, beginning on the third day, are adequate. The sulfadiazine treatment may be discontinued on the seventh day and the patient released from isolation when two consecutive cultures are reported as negative. If the cultural test on the seventh or ninth day is positive, a 7-day course of sulfasuxidine warrants trial.

Multiple negative cultures should be obtained before releasing a patient who has had a sulfonamide-resistant infection.

Sulfonamides may be used "prophylactically" in Flexner and Schmitz infections. When the prevalence of the infection is 10 percent or more, the treatment of all individuals is justified. The objective is to free the group of infection. One gram of sulfadiazine twice daily for 7 days is recommended. There should be at least two post-treatment surveys by the culture method, and any individual found positive should be isolated at that time and given full therapeutic doses of sulfonamide. Smaller doses may effectively suppress the development of clinical disease, but this should be recommended only in those emergencies which may occur in military practice.

Chemotherapy is to be used in acute diarrheal diseases with dehydration only when appropriate measures have been taken to restore and maintain normal hydration.

All individuals under treatment must be followed adequately for evidence of early signs of toxicity.

The sulfonamides have a high value in shigellosis, both in the treatment of clinical cases and in the control of the infection.

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FULL-TIME PUBLIC HEALTH POSITIONS IN LOCAL HEALTH DEPARTMENTS [1]

By MARION E. ALTENDERFER, *Associate Statistician, United States Public Health Service*

INTRODUCTION

The first local health departments in this country were in urban areas. As early as 1873, 134 cities had boards of health (1). Although most of these early organizations were not under the supervision of full-time health officers, many of the larger cities did have full-time units. This service was extended to the rural population for the first time in 1908 with the establishment of a full-time county health department in Jefferson County, Ky. Two more such departments began operation in 1911, one in Guilford County, N. C., and the other in Yakima County, Wash. (2). City-county units were soon organized and later two or more cities or counties were sometimes combined into district units. There has been a steady growth both in the number of local full-time health departments and in the number of political units served by such departments. By 1945, there were 1,160 full-time local health departments serving approximately 2,100 cities and counties (3). It has been estimated that about two-thirds of the people in the country live in communities served by full-time local health departments (4).

Health departments differ greatly both in the numbers and types of personnel employed. Studies have been made of the number of personnel of different types in all local health departments (4, 5) but no reports have been published on the pattern of individual health department organization and the relation between size of staff and population served. To study these aspects of the problem, use has been made of some of the data obtained in a survey of full-time professional and technical personnel in State and full-time local health departments sponsored by the Surgeon General's Committee on Postwar Training of Public Health Personnel in July 1945.

[1] From the Division of Public Health Methods.

MATERIAL AND METHOD

The survey questionnaire was sent to all local health departments listed in the 1945 Directory of Full-time Local Health Officers (6). Returns were received from 933 units or about 80 percent of all full-time local health departments. Table 1 shows the variation in the percent of returns from different parts of the country and from communities of different sizes. The variation was not great from one section to the other except for the Northeast. With regard to size, the variation was also small. On the whole, the returns seem to be fairly representative of all local full-time health departments with respect to location and size of community.

TABLE 1.—*Percent of local health departments returning questionnaires, by geographic area[1] and population size-group*

Population size-group	Total	Northeast	South	Central	West
Total	81	71	81	85	83
Under 25,000	79	62	87	82	67
25,000–49,999	82	65	82	88	86
50,000–99,999	79	73	79	84	81
100,000 and over	81	79	71	85	92

[1] Geographic areas include:
  Northeast: Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont.
  South: Alabama, Arkansas, Delaware, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, West Virginia, District of Columbia.
  Central: Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin.
  West: Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming.

The questionnaire asked for certain information about 17 specific types of professional and technical public health personnel and for any other types of technical personnel employed by the health departments. Of the information obtained, this report utilizes only the number of established positions of each type (both filled positions and those vacant at the time of the survey).

The data on the population served by each department are from the 1940 Census of Population. The per capita buying income data are for 1942 and were obtained from Sales Management, the Survey of Buying Power Number (7).

The returns show a total of over 17,000 full-time established positions in local health departments. If the 80 percent which returned questionnaires are representative of all full-time local health departments, the estimated number of established positions in all these units would be approximately 21,000.

FINDINGS

Distribution of health departments by size.—The 933 local health departments vary in size from one position (a health officer) to several hundred professional and technical positions (table 2). Almost one-fourth of the health departments have less than 5 positions; approximately one-half have less than 8 positions; and only 4 percent have 50 or more positions.

TABLE 2.—*Distribution of 933 local health departments by number of professional and technical positions on staff*

Number of positions on staff	Number of health departments	Percent distribution	Cumulative percent
All sizes	933	100	
1	11	1	1
2	30	3	4
3	81	9	13
4	104	11	24
5	83	9	33
6	104	11	44
7	61	7	51
8	56	6	57
9	53	6	63
10	47	5	68
11–14	96	10	78
15–19	54	6	84
20–29	67	7	91
30–39	32	3	94
40–49	14	2	96
50–99	21	2	98
100 and over	19	2	100

Ratio of population to total health department positions.—The ratio, population per full-time health department position, was calculated for each local health department, using the total number of established positions for all types of professional and technical personnel in the health department. Figure 1 shows the distribution of 186 city and 735 county units by this ratio. For 12 health departments it was not possible to determine the population served, therefore no ratios could be calculated for these units. The group of city health departments includes also city-county units where the city had a population of 100,000 or more in 1940. The group of county health departments includes also the rest of the city-county units and the State district and local district health departments.

It is apparent from the curves in the figure that the city health departments have more favorable ratios than the county health departments. The median for the city health departments is 4,200 and that for the county health departments is 5,800. For the city health departments, the middle 50 percent of the ratios lie between 3,100 and 5,400. For the county health departments this range extends from 4,100 to 8,300, making the interquartile range almost twice that of the city health departments. The highest ratio for the

city health departments is 23,500 persons per position, while for the county health departments the highest ratio is 137,700.

As might be expected, there is a definite relation between the ratio of population per health department position and the average per capita buying income of the population served. In general, the higher the income, the more favorable the ratio of population to health department positions (table 3). While 17 percent of the high-
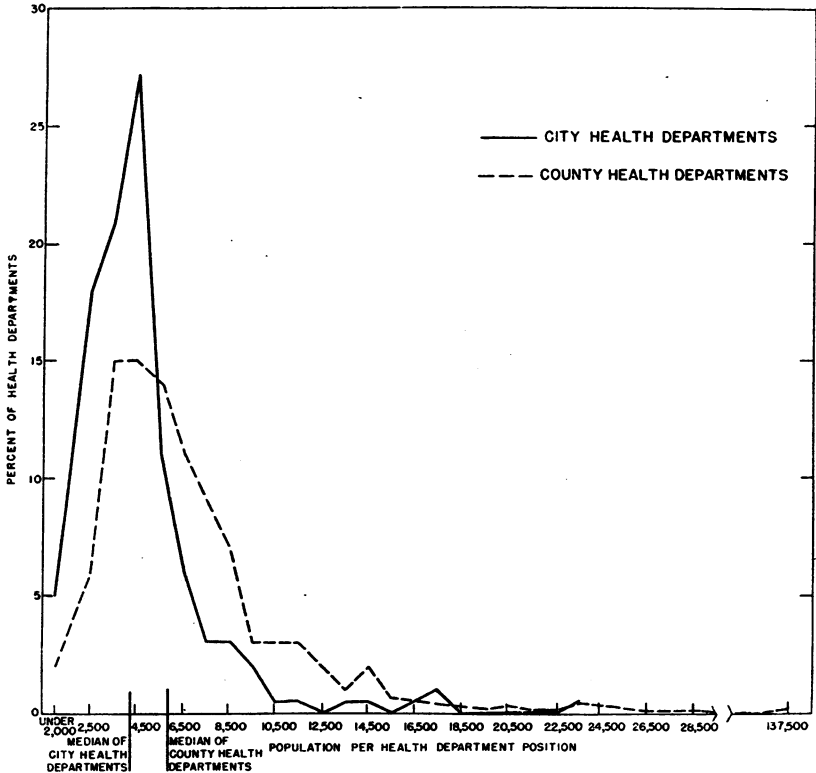
FIGURE 1.—Distribution of 186 city and 735 county health departments by ratio of population served to total professional and technical positions

est income group have ratios of better than 1 professional and technical position per 3,000 population, only 9 percent of the middle and 3 percent of the lowest income groups have such service. Also, 71 percent of the highest, 54 percent of the middle, and only 36 percent of the lowest income groups have ratios more favorable than 1 to 6,000. The median for the lowest income group (6,800) is almost half again as large as that for the highest income group (4,600). The minimum and maximum values show the great range of ratios in all 3 income groups.

Distribution of positions by type.—The 17,000 positions in the health departments returning questionnaires are divided among the different

TABLE 3.—*Distribution of 921 local health departments by ratio of population served to total professional and technical positions for three income groups*

| Population per professional and technical position | Annual per capita buying income of population served | | | | | |
| | Number of health departments | | | Cumulative percent | | |
	Under $300	$300–699	$700 and over	Under $300	$300–699	$700 and over
Total_____	183	340	398	_____	_____	_____
Under 2,000_____	1	11	15	1	3	4
2,000–2,999_____	5	19	52	3	9	17
3,000–3,999_____	15	56	81	11	25	37
4,000–4,999_____	20	56	87	22	41	58
5,000–5,999_____	27	47	52	36	54	71
6,000–6,999_____	31	29	30	53	63	79
7,000–7,999_____	23	27	20	66	71	84
8,000–8,999_____	14	31	11	74	80	87
9,000–9,999_____	11	9	8	80	83	89
10,000–10,999_____	11	9	4	86	86	90
11,000–11,999_____	4	9	10	88	89	93
12,000 and over_____	21	37	28	100	100	100
Median_____	6,800	5,600	4,600	_____	_____	_____
Minimum_____	1,500	1,000	1,000	_____	_____	_____
Maximum_____	33,300	90,300	137,700	_____	_____	_____

types of professional and technical positions as shown in table 4. Graduate nurse positions account for exactly half of all the positions. The next largest groups are: Sanitarians, 11 percent of the total; health officers and other medical positions, 10 percent; and inspectors, 10 percent. Several types of positions, epidemiologists, nutritionists, and chemists, each account for less than 1 percent of the total. It must be remembered that the figures shown do not include full-time positions in health departments under part-time health officers. This is especially important in regard to the number of graduate nurses.

TABLE 4.—*Established full-time positions for different types of public health personnel reported by 933 full-time local health departments*

Type of position	Number reported	Percent distribution
Total_____	17,192	100
Health officer_____	940	5
Epidemiologist_____	88	0.5
Other physician_____	843	5
Graduate nurse_____	8,526	50
Sanitary or public health engineer____	236	1
Sanitarian_____	1,810	11
Inspector_____	1,641	10
Veterinarian_____	318	2
Dentist_____	208	1
Statistician_____	220	1
Health educator_____	112	1
Nutritionist_____	60	0.3
Bacteriologist, serologist_____	436	3
Chemist_____	70	0.4
Laboratory technician_____	492	3
X-ray technician_____	108	1
Other technical_____	1,084	6

Although the survey questionnaire was meant to include only professional and technical personnel, it is felt that some health departments may have included clerical and other nonprofessional or nontechnical positions in their reports. For example, a number of statistical clerks may have been reported as statisticians.

Composition of health departments.—The total number of employees in a health department does not give the complete picture. Health departments with the same number of employees may differ both in the types of positions included and in the number of each type included. Aside from the health officer, the types of positions most frequently found are those for graduate nurses and sanitation personnel. Few health departments with less than 6 positions have any other type of position than these 3. In spite of the great variation in the composition of health department staffs, it is possible to determine typical patterns of composition for different sizes of health

TABLE 5.—*Percent of health departments of different sizes with specific types of positions*

| Number of positions in health department | Number of health departments | Percent of health departments in size-group with one or more positions for— | | | | | | | | |
		Health officer	Graduate nurse	Sanitation personnel [1]	Medical officer [2]	Laboratory personnel [3]	Statistician	Dentist	Health educator	Nutritionist
1	11	100								
2	30	100	77	20	3					
3	81	100	96	74	1	2				
4	104	100	98	95	7	3	4	3		
5	83	100	96	98	7	1	11	4		
6–9	274	100	99	95	12	16	5	8	6	3
10–19	197	100	99	98	34	42	15	10	10	3
20–29	67	100	99	97	61	73	30	36	10	6
30–39	32	100	100	97	81	88	34	28	31	3
40–49	14	100	100	100	93	79	43	43	43	21
50 and over	40	100	100	100	95	90	68	38	58	40
All sizes	933	100	97	91	25	27	13	11	9	4

[1] Includes sanitary engineers, sanitarians, inspectors and veterinarians.
[2] Includes epidemiologists and other physicians.
[3] Includes bacteriologists, serologists, chemists, and laboratory technicians.

departments. For example, the typical health department with 5 positions consists of a health officer, a sanitation position and 3 nurse positions. The typical 10-position health department has a health officer, 2 sanitation positions, 6 nurse positions, and a laboratory position.

As the size of health department increases, the number of different types of positions also increases (table 5). With a few exceptions, the proportion of health departments with a particular type of position also increases with size. The proportion of health departments with laboratory positions varies from 0 percent in the departments with only 2 positions to 90 percent in those with 50 or more positions. As was stated before, the data on statisticians must be used cautiously

because of the possible inclusion of subprofessional personnel in this group.

Since nurses and sanitation personnel constitute a large part of the staffs of health departments of all sizes and are practically the only type of personnel, in addition to the health officer, employed in small health departments, the ratios of population to these positions are important measures of the adequacy of health department staffs. Therefore, these two ratios were computed for all health departments for which the population served is known.

Ratio of population to graduate nurse positions.—The distribution of health departments by this ratio is shown in table 6. It will be

TABLE 6.—*Distribution of 921 local health departments by ratio of population served to graduate nurse positions*

Population per graduate nurse position	Number of health departments	Percent distribution
Total with known population	921	100
Under 4,000	36	4
4,000–5,999	108	12
6,000–7,999	132	14
8,000–9,999	142	16
10,000–11,999	121	13
12,000–13,999	84	9
14,000–15,999	47	5
16,000–17,999	41	4
18,000–19,999	25	3
20,000 and over	156	17
No graduate nurse positions	29	3
Median	10,800	-----------
Minimum	1,700	-----------
Maximum	413,000	-----------

seen that 29 health departments or 3 percent of the total have no graduate nurse positions. Another 17 percent have ratios of 20,000 persons or more per nurse position. The median of the distribution is 10,800. The whole distribution is considerably skewed in the direction of the higher ratios. This skewness is caused by a small number of health departments with extremely large ratios.

Ratio of population to sanitation positions.—Table 7 shows the distribution of health departments by the ratio of population to total sanitation positions. Included are both professional sanitation positions (engineers and veterinarians) and nonprofessional positions (sanitarians and inspectors). Nine percent of the health departments have no positions for sanitation personnel. Twelve percent have ratios of 50,000 or more persons per sanitation position. On the other hand, 8 percent have ratios of less than 10,000 persons. The median is 24,800. As with the distribution of graduate nurse positions, the distribution is skewed in the direction of the higher ratios.

TABLE 7.—*Distribution of 921 local health departments by ratio of population served to sanitation positions*

Population per sanitation position	Number of health departments	Percent distribution
Total with known population	921	100
Under 10,000	74	8
10,000–14,999	142	16
15,000–19,999	129	14
20,000–24,999	121	13
25,000–29,999	95	10
30,000–34,999	66	7
35,000–39,999	48	5
40,000–44,999	31	3
45,000–49,999	26	3
50,000 and over	111	12
No sanitation positions	78	9
Median	24,800	----------
Minimum	2,700	----------
Maximum	413,000	----------

In fact, the two distributions are quite similar in character although most of the graduate nurse ratios are much smaller than the sanitation personnel ratios.

SUMMARY

Use has been made of data obtained from a survey of full-time professional and technical public health personnel by the Surgeon General's Committee on Postwar Training of Public Health Personnel in 1945 to study the pattern of local health department organization and the relation between size of staff and the population served. The most important points brought out by the analysis are:

1. Approximately one-half of the local health departments in the survey have less than eight full-time professional and technical positions.

2. City health departments have more favorable ratios of population to total health department positions than do county health departments.

3. There is a definite relation between the ratio of population per health department position and the average per capita buying income of the population served; higher incomes being associated with more favorable ratios.

4. Half of the full-time professional and technical positions in local health departments are for graduate nurses.

5. The majority of small health departments employ only a health officer, a sanitation worker, and one or more graduate nurses.

6. The median ratio of population to graduate nurse positions is 10,800.

7. The median ratio of population to sanitation positions is 24,800.

DISCUSSION

The data presented in this report show that there is great variation in the composition of health department staffs. While some health departments seem to have adequate staffs, others are extremely under-staffed in relation to the population served. It is of interest in this connection to compare the actual distributions of the ratios of population to various types of positions with the standards suggested by the Committee on Local Health Units of the American Public Health Association in a recent report (4).

This Committee suggests as a minimum staff, for a health department serving 50,000 persons: A health officer, a trained sanitary or public health engineer, a sanitarian, and 10 public health nurses. This would mean a ratio of 25,000 persons per sanitation position. The median of the actual distribution of health departments by this ratio is 24,800. Therefore, half the health departments have more favorable ratios than the standard suggested by the Committee and half have less favorable ratios.

The number of public health nurses recommended by the Committee would mean a ratio of 5,000 persons per nurse position. The survey questionnaire asked for all graduate nurse positions and not for public health nurses only, therefore the survey data are not quite comparable to the Committee's standard. However, the average ratio of population to graduate nurse positions found in this survey is over twice as great as that recommended by the Committee.

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TULAREMIA

FIRST CASE TO BE REPORTED IN ALASKA

By RALPH B. WILLIAMS, *Director, Division of Public Health Laboratories, Juneau, Alaska*

A period of 8 years has elapsed since Philip and Parker (1) and Philip (2) isolated *Pasteurella tularensis* from naturally infected rabbit ticks, *Haemaphysalis leporis-palustris*, collected from various hares (*Lepus americanus*) taken in the vicinity of Fairbanks, Alaska. The first group, collected July 19, 1937, consisted of 150 ticks of all stages, from 5 hares. The second group consisted of 6 adults, 48 nymphs, and 76 larvae collected on July 26 from 1 hare which appeared sluggish when shot. The third group comprised 27 nymphs from 1 hare. The collections were triturated with physiologic saline solution. Each suspension was equally divided and inoculated into 2 guinea pigs. The necropsy findings in these guinea pigs were characteristic of tularemia, and pure cultures of *P. tularensis* were isolated and confirmed at the Rocky Mountain Laboratory, Hamilton, Mont. This was the first evidence of the presence of tularemia in the Territory of Alaska.

On May 12, 1939, Mr. J. W. Warwick, field assistant of the United States Bureau of Biological Survey, collected 73 ticks of all stages from 2 hares "in good condition" near Fairbanks, Alaska. Two guinea pigs were inoculated with suspensions of these ticks at the Rocky Mountain Laboratory; both animals succumbed and presented typical pathological lesions of tularemia. A pure culture of *P. tularensis* was isolated.

On June 25, 1945, a blood specimen was received at the Juneau Laboratory, Territorial Department of Health, from Capt. John H. Pinson, Jr., United States Army Medical Corps, for an agglutination test for tularemia. The blood serum was positive in a dilution of 1:1,280. On August 25, 1945, Captain Pinson reported a definite case of tularemia, clinically typical of the ulceroglandular type.

Patient.—J. O.: Age 31 years, meteorologist, Northway, Alaska, was taken ill on June 5 with typical symptoms of influenza. There was an epidemic of a mild type of influenza in the area at that time. His symptoms were headache, orbital pain, generalized aching, fever of 104° F., and a dry bronchial cough. He was confined to his bed at home on June 7. The patient developed swollen, tender axillary and epitrochlear lymph nodes in his left arm. There was a small encrusted lesion on the back of the left middle finger which he stated had been there about a week. The red blood cell count was 4.56 million; white blood cell count, 8,050; hemoglobin, 85 percent (Talquist); differential count—polymorphonuclears, 50 percent; lympho-

cytes, 40 percent; monocytes, 7 percent; stab cells, 2 percent; and juvenile cells, 1 percent. Urinalysis was negative for sugar and albumin. Attempts were negative to culture organisms from the lesion on the finger and from material aspirated from the lymph nodes on egg yolk at the United States Army Air Force Base, Army Transport Command (using a homemade incubator). A blood sample taken June 20, 1945, showed agglutinins against *P. tularensis* to a titer of 1:1,280 when tested at the Juneau Territorial Laboratory.

The patient was treated symptomatically and rapid recovery was made without complications. The lymph nodes gradually receded over a period of 10 days.

The patient gave a history of having skinned numerous muskrats for about 6 weeks prior to onset of symptoms. He paid no particular attention to the livers, although one of his companions thought he had noticed some mottled discoloration in them, but was not sure. No muskrats, beavers, mice, or rabbits were found dead by the patient in the area of trapping operations.

Additional agglutination tests were made on blood specimens collected from the patient on August 25, 1945. The Rocky Mountain Laboratory, Hamilton, Mont., reported that the blood specimen forwarded to the laboratory agglutinated *P. tularensis* completely at 1:640 and partially at 1:1,280.

This is the first recognized human case of tularemia to be reported in the Territory of Alaska. The Division of Public Health Methods of the United States Public Health Service informs us that cases of tularemia have been recognized in 47 States and the District of Columbia (all States except Vermont), and in Japan (1925), Russia (1928), Norway (1929), Canada (1930), Sweden (1931), Austria (1935), Turkey, Czechoslovakia, and Mexico. The disease has been found to have a wide distribution among animal species, but the muskrat has been associated with human infections only in Oregon, Idaho, Montana, New York State, and at Northway on the Upper Tanana River Drainage, Alaska.

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A PERFORMANCE TEST FOR RATING DISHWASHING DETERGENTS [1]

By Edward H. Mann, *Assistant Sanitarian (R)*, and C. C. Ruchhoft, *Principal Chemist, United States Public Health Service*

With the growth of urban populations and a large increase in the number of people traveling and eating out, restaurant sanitation has become an important factor in public health. The importance of multi-use eating utensils in the spread of disease has been demonstrated by extensive experiments conducted by the Army. Information demonstrating the need for better sanitizing practices in handling multi-use utensils is available in the files of many State health departments. The public, because of the increased practice of health departments in grading and placarding eating establishments, is becoming aware of the need for restaurant sanitation. Municipal and State health departments, in increasing numbers, are adopting new ordinances or revising old ones for the control and improvement of sanitation practices in public eating and drinking establishments. With each reduction of permissible utensil bacterial count and each refinement in examining technique, the question of efficient detergent operation in the cleansing of multi-use utensils assumes a greater importance. Unless a utensil is first completely cleaned, proper sterilization is improbable with hot water and steam and impossible with bactericidal chemicals. "Clean" is defined as being free from food soils, greasy films, and hard-water films which may harbor bacteria and protect them from the bactericidal agent which is used later.

Sanitarians in the field of food sanitation have for some time recognized a wide variation in proprietary compounds offered for washing dishes and other equipment in restaurants, bars, and dairies. As a result of the need for a better field criterion for judging detergent efficiency, this station has undertaken a very careful laboratory study of detergents. This report presents a laboratory washing-performance test that was developed in the course of this study. The present study was greatly aided by the previous work of Gilcreas and O'Brien (1), Tiedeman, and of Hucker (2) on detergents and detergent-performance tests. The soiling agent used in this study was proposed by Hucker (2). A technique for soiling, baking, examining, washing, rinsing, drying, and re-examining microscope slides to determine accurately the soil-removal efficiency of the various detergents was one of the objectives of the study. The description of the washing machine, photometer, and technique developed for such a test follows.

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[1] From the Division of Sanitary Engineering, Water and Sanitation Investigations, Cincinnati, Ohio.

DEVELOPMENT OF THE PHOTOMETER

Because the human eye is not sensitive enough to grade the amount of soil remaining on a utensil within fine limits, it was necessary to use some photoelectric means for this purpose. It was decided to use standard 1″ x 3″ microscope slides in the washing tests as they were quite uniform, flat, small, and not easily attacked by alkaline solution which might tend to etch and corrode cheaper grades of glass. The photometer (fig. 1) then was designed around the use of this type of slide.

A Weston Model 594 photoelectric cell was used in combination with a G-M Laboratories Galvanometer (catalog No. 2561-D). Various arrangements of lenses, light sources, and diffusing filters were tried before the best combination was found.

In the first models built, one slide was examined at a time, the arrangement being quite simple. A small 3.5-volt light bulb was used as a light source. This light was passed through a 1″ x 3″ microscope slide which had been frosted on both sides so as to diffuse the light over the 1″ x 3″ area. The light then passed through the slide used in the washing test and subsequently into the photoelectric cell. This arrangement proved unsatisfactory for two reasons: First, the light was not uniformly distributed before passing through the slide in question, and second, after passing through the soiled slide the light was broken up into light and dark areas because of uneven distribution of soil over the entire area of the slide.

To remedy the uneven distribution of light a lens was placed between the slide and the photoelectric cell to focus the image of the slide into the opening of the cell. This eliminated the difficulty arising from uneven light distribution but reduced the sensitivity because the entire area of the photoelectric cell was not covered by the light.

The lens was replaced by a second diffusing glass which was found to increase sensitivity, though only a part of the transmitted light was caught by the cell.

Reading but one slide at a time gave reductions in light transmission too small for meter deflections sufficient for very accurate calculations. This led to rebuilding the instrument on a larger scale and increasing the light source with the view of reading a number of slides at one time. Twelve slides were examined together, but this number was found to be too great to transmit sufficient light to deflect the meter when slides were soiled. By using a 40-watt, 110-volt light bulb in series with a 25-ohm rheostat, six slides were found to give approximately one unit deflection of the meter when soiled, whereas the meter with no slides in it read full-scale deflection. To eliminate further any uneven distribution of light passage through the instrument, a
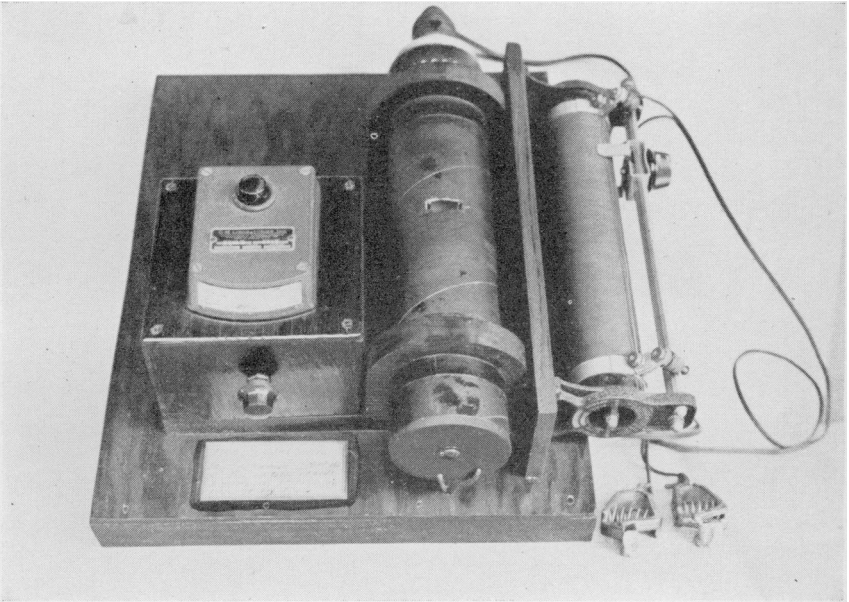
FIGURE 1.—The detergent efficiency photometer.

PLATE II
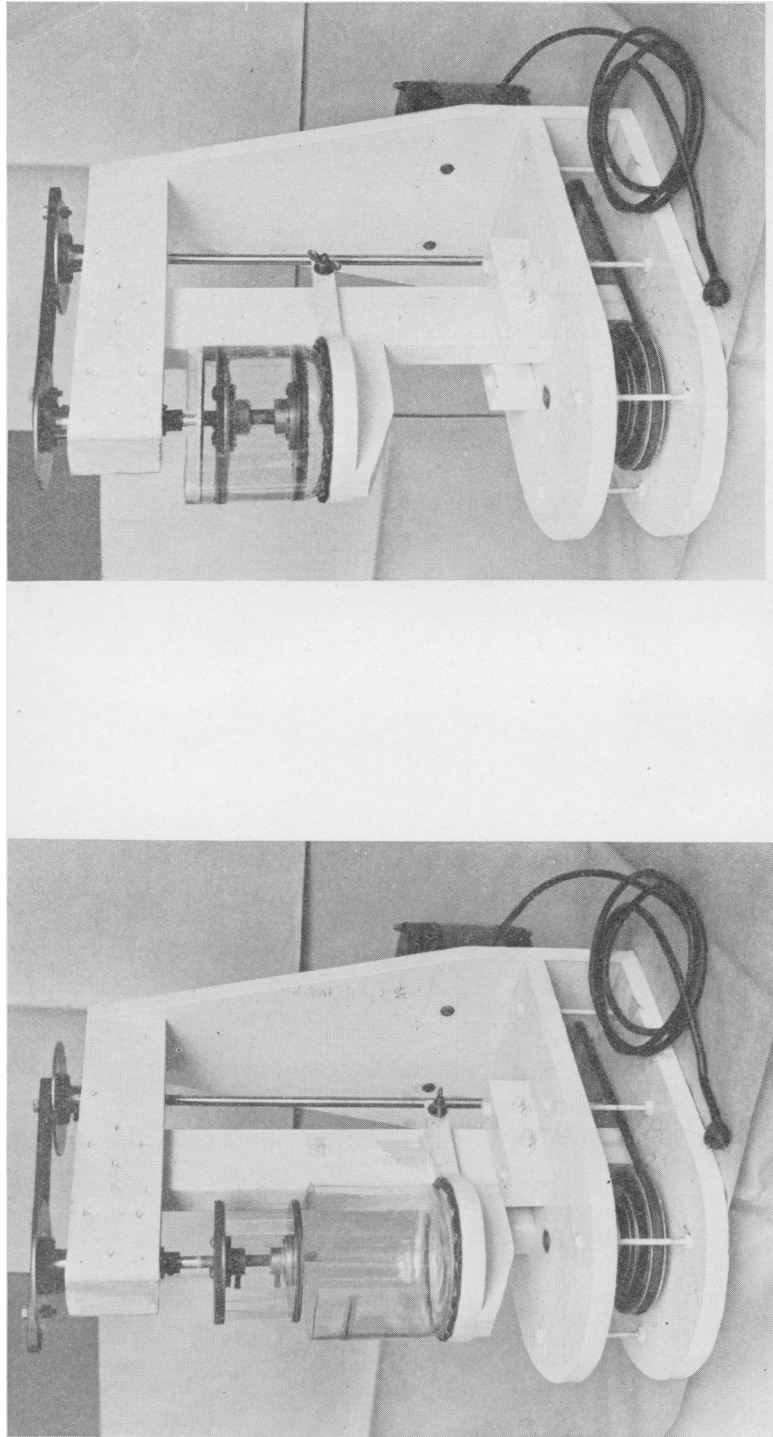
FIGURE 2.—Experimental dishwashing machine.

3" Diameter
cardboard
mailing tube

Focusing slide mounting
for light bulb

6 volt 32-32 auto
headlamp bulb, both
filaments used

Slot mounting to hold
6-1"x3" microscope slides

Bulls eye lens

Ground diffusing
glass

Weston Model 594
photo-electric cell

150 ohm variable
resistance

1 ohm variable
resistance

Ammeter

Galvanometer

6 volt Storage
Battery

6 volt Automobile
Generator

⅓ HP Motor

110 volt A.C.

U. S. Public Health Service
Water and Sanitation Investigations
Cincinnati, Ohio
DETERGENT EFFICIENCY PHOTOMETER
August 22, 1945
Not to Scale

FIGURE 1A.

convex lens was placed in front of the light source at such distance as to collimate the light into parallel rays. The dimensions given on the arrangement, as shown in figure 1A, were found to give the best results, though it must be kept in mind that because of variations in the focal length, the position of the convex lens may need to be altered when using a different lens in order to insure the emergence of paralleled light.

After use of the instrument, it was found that fluctuations in alternating current line voltage contributed a considerable variation in readings. To eliminate this, an automobile headlight bulb and heavy duty automobile battery were substituted for the 40-watt lamp. To obtain ample light, both filaments had to be used and this required current at the rate of 10 amperes from the storage battery. The high current requirement resulted in a regular drop in meter readings. Constant galvanometer readings were obtained by charging the battery with a 6-volt generator driven by a ⅛-horsepower, 110-volt motor during use of the instrument to compensate for the heavy drain of the battery.

DEVELOPMENT OF EXPERIMENTAL WASHING MACHINE

Numerous experiments were conducted during the development of the present washing machine with the purpose of building a machine which would remove as little soil as possible with distilled water and remove nearly all of the soil with the best detergents available.

Slides were soiled with the soiling agent and baked for 1 hour at approximately 100° C. These were then washed manually in numerous detergents until several samples were segregated which appeared to remove the soil with the greatest ease. These detergents were used in the development of the machine.

The first machine was built to hold twelve 1″ x 3″ standard microscope slides in a circular washing head 5 inches in diameter. The slides were placed in slots set at a 45° angle to a line tangent to the circumference of the head. The head was driven so as to reciprocate through an angular rotation of 120° each half cycle at a speed of 60 cycles per minute. This speed was insufficient to remove the soil by even the better detergents. The speed was increased, holding the rotation angle constant until 120 cycles per minute were reached. This speed failed to give satisfactory results, so the machine was again slowed down and the speed held constant while the arc of rotation was varied. This was increased up to as high as 500° each half cycle. Various combinations of speed and rotational arc were tried. It was found possible to increase the speed and rotational arc sufficiently to remove a considerable percentage of soil by hot water and mechanical action alone.

The combination giving the best results was determined to be a speed of 90 cycles per minute through an arc of rotation of 310° each half cycle.   At this speed it was found that the turbulence developed in the detergent solution was great enough to develop vacuum pockets between the slides when 12 were placed in the head at one time.   This was eliminated by reducing the number of slides washed at one time to six.

The machine was found later to give slightly better performance by reducing the angle at which the slides were set in the washing head from 45° to 20°.   A machine of the design shown in the photographs (fig. 2) and the accompanying diagram (fig. 2A) has a range of soil removal of from approximately 1 percent using distilled water at 140° F., up to approximately 98 percent using 0.3 percent castile soap made up in distilled water at 140° F., followed by a distilled-water rinse.

PREPARATION OF SLIDES FOR WASHING

A standard soiling agent prepared according to the following formula by Hucker (2) was used in this test.

Peanut butter_____ 10 gm.
Butter_____ 10 gm.
Lard_____ 10 gm.
Flour_____ 10 gm.
Dehydrated egg yolk_____ 10 gm.
Evaporated milk_____ 15 ml.
Distilled water_____ 50 ml.
Higgins India ink_____ 4 ml.
International printing ink RL3400, [2] diluted 1:1 with boiled linseed oil_ 10 drops

Copper trichlorphenate (Dowicide K611)_____ 1 gm.
N/1 NaOH_____ 3 ml.

The peanut butter, lard, and butter are melted together and mixed into a smooth paste.   The milk and flour are mixed together until homogeneous, and the egg yolk and water are mixed.   The egg-yolk mixture and the milk-and-flour mixture are then combined and beaten in a soda fountain malt-mixer for at least 2 minutes at high speed. The Higgins ink is then added and mixing is continued for another 2 minutes.   The printing ink-linseed oil mixture is then added to the melted-fat mixture drop by drop while being stirred rapidly.   The water mixture is then added to the fat mixture and beaten in the malted-milk mixer for 5 minutes.   The Dowicide K611 is mixed with the NaOH and added to the soil during final beating.

Prior to use, one part of distilled water is stirred into five parts of soil to obtain the proper consistency.   Several methods of applying the

[1] RL3400 may be secured from the International Printing Ink Division of the Interchemical Company, 432 W. 45th St., New York, N. Y.

soil to the slides were tried, including dipping, brushing on, and rolling on with a rubber roller similar to that used with fingerprinting inks. As the latter method gave the most uniform distribution and smoothest film, it was adopted for use in the test.

The soil is applied as follows: A quantity of soiling agent is poured out on a glass plate about 1 foot square. The rubber roller is brought into contact with the soil and rolled into a thin film over the plate. The roller is then rolled over one side of the slide to apply a thin film of soil. More soil is applied to the roller from the glass plate and the other side of the slide is soiled.

The soil must be baked on the slides; otherwise it is removed too easily. The temperature at which this baking is done is very critical from the standpoint of reproducibility. Numerous experiments were conducted to determine the optimum temperature with the following results:

Starting temperature of oven (baked 1 hr.)	Average soil removal (percent)	
	Distilled water [1]	Castile soap [2]
65° C.	73.4	100.0
70° C.	66.3	100.0
80° C.	50.7	100.0
95° C.	1.0	97.6
100° C.	0.0	59.3

[1] Slides washed in distilled water at 140° F.
[2] Slides washed in 0.3-percent castile soap in distilled water at 140° F.

The temperature of 95° C. was selected for two reasons: (1) It gave the broadest range of removal; and (2) the results were found to be more consistent over the entire detergent range at this temperature than at any other tried.

WASHING TECHNIQUE AND COMPUTATION

The slides to be used in the test first must be washed thoroughly with some good detergent or soap made up in distilled water and rinsed twice in distilled water. They must be dried by either wiping them on a clean dry towel, free from lint and dust, or by rinsing in alcohol and allowing them to dry by evaporation. Slides are afterwards preferably handled only with forceps. The cleaned and dried slides are then sorted into groups of six and placed in wooden slide boxes, the bottoms of which have been cut out, leaving only a ¼″ web strip on each side so as to support the ends of the slides. These boxes are used to support the slides in the oven during baking.

The motor-generator is then turned on and the photometer connected to the battery. Adjust the two variable resistances shown in

CRANK HEAD ASSEMBLY PLAN

5⅝" dia. Brass Disc.
⅛" thick
180 R.P.M.

⅜" cap screws

11½"

9 3/32"

5⅝" Brass Disc
⅛" thick

310°

90 cycle
310° reciprocation
each half cycle

2 3/32"

180 R.P.M.

2 3/16"

SLOTTED BRASS DISC ⅛" THICK

1½" x 1/16" slots

⅛" dia. holes on
1¾" dia. circle

½" d.

2¼" c.

2 3/16" c.

⅛" 2

20°

WASHING HEAD

ASSEMBLY

5⅝"

½"

¼" thick
rubber disc

2⅝"

Slotted Brass Discs

Shaft Collar "A"

⅛" Sheet Rubber

Solid Brass Disc

SIDE ELEVATION

¾"

3⅝"

9⅛"

1½"

9 3/32"

Crank Head Assembly

Shaft Collar "A"

Poured Babbitt Bearings

3½"

2"

Washing Head

¼" dia. steel drill rod

3½"

7" dia.

Rubber tubing tacked
to shelf

Adjustable Shelf

⅓ HP Motor
1750 R.P.M.

Bronze Sleeve Bearings

1½" V Pulley

6" V Pulley
5/16" round
leather belt

3¾" V Pulley

2'-1"

1½" V Pulley

12"

18¾"

Note: Pulley diameters measured to
bottom of groove
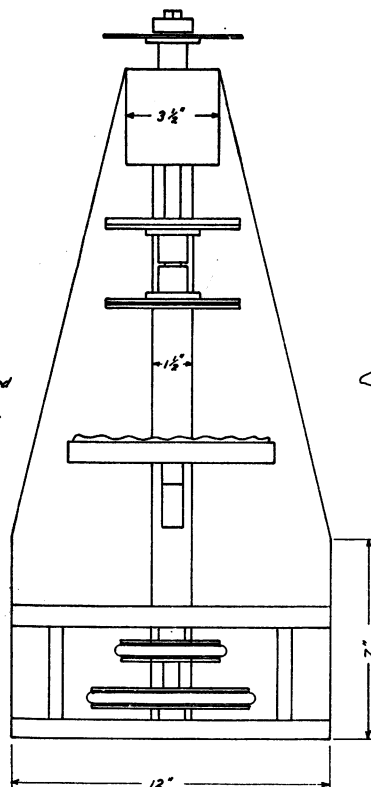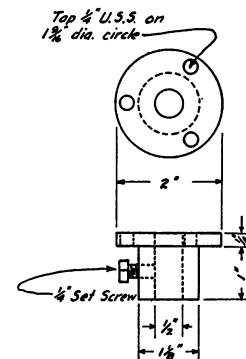
FRONT ELEVATION

3½"

1½"

7"

12"

SHAFT COLLAR "A"

Tap ¼" U.S.S. on
1¾" dia. circle

2"

¼" Set Screw

1"

½"

1⅝"

FIGURE 2A.

694661—46 (Face p. 882)

figure 1A, until the galvanometer reads full scale with no slides in the instrument. In the case of the galvanometer described above this will be a reading of 60. Allow 2 or 3 minutes for the battery and generator to come to a constant equilibrium and readjust the 1-ohm rheostat, if necessary, to re-establish the full-scale reading. The instrument reading, after this period of time, will usually remain constant. Place each set of six slides in the photometer and record the readings of the slides when clean.

Soil the slides as described above under "Preparation of Slides for Washing." Care must be taken to keep the sets isolated, for all slides are not the same. Place the soiled slides in an oven closely adjusted for 95° C. After the slides are put in the oven the thermometer will record a drop to as low as 85° to 87° C., but will climb back to 94° C. during the baking period of 1 hour. The slides are allowed to remain in the oven for 1 hour from the time they were placed there. As baking temperature must be carefully controlled for reproducible results, in some ovens it may be possible to bake only 1 box of 24 slides at a time due to variations in temperature throughout the oven. In such cases, each box will need to be placed in the same spot in the oven to insure uniformity.

At the end of 1 hour the slides are removed from the oven and allowed to cool for 30 to 40 minutes. When cool, readings are made as before in the photometer on the soiled sets of slides.

Each set of six slides is then placed in the washing head of the washing machine and the solution jar [3] raised around the head. A solution of the detergent to be tested, made up in the concentration recommended, which in most cases is 0.3 percent, is heated to 140° F. and the jar filled with it until the washing head is covered by about ½ inch of solution.

The machine is then turned on and timed with a stop watch, the washing process being carried on for exactly 3 minutes. During these 3 minutes the temperature of the washing solution will drop approximately 8° F. giving an average temperature of 135° F. to 136° F. during the washing.

At the end of the 3-minute washing period the machine is stopped and the jar of detergent removed. An empty jar is now placed around the washing head and the machine turned on and allowed to run for 10 complete cycles. This throws off the excess detergent prior to rinsing.

A third jar is now placed around the head and filled, as before, with boiling water. The machine is turned on and the slides are rinsed for 2 minutes. Boiling water was chosen for the rinse for three reasons: (1)

[3] Convenient washing and rinsing jars for this test were obtained by sawing off 4-liter pyrex serum bottles to give jars 5½ inches in depth.

It meets with the most drastic of sterilization requirements for eating utensils; (2) constant temperature is easily maintained; and (3) water hardness films are deposited more easily than at lower temperatures.

At the end of the 2-minute rinse, the rinsing jar is removed and the machine again turned on and allowed to run for 10 cycles to aid in drying the slides. The slides are now removed and placed in their appropriate place in the slide box to dry.

When dry a third reading is taken on the photometer and recorded as "reading after washing" (col. 7). From these three readings the percentage of soil removed may be calculated. The calculation is illustrated in table 1. It may be pointed out that the detergents used for this demonstration were chosen to show all degrees of washing performance.

TABLE 1.—*Typical examples of efficiency tests*

Detergent 0.3 percent solution	Set of 6 slides No.	Initial meter reading empty	Reading of 6 clean slides	Light absorbed by glass	Reading of 6 soiled slides	Light absorbed by dirt and glass	Light absorbed by dirt	Reading of 6 slides after washing	Light absorbed by residual dirt and glass	Light absorbed by residual dirt	Light absorbed by dirt moved	Soil removed (percent)
Column No.		1	2	3	4	5	6	7	8	9	10	11
Calculations				1−2		1−4	5−3		1−7	8−3	6−9	$\frac{10}{6}\times100$
A	1	60.0	41.0	19.0	1.0	59.0	40.0	2.5	57.5	38.5	1.5	3.8
B	2	60.0	40.5	19.5	2.5	57.5	38.0	9.0	51.0	31.5	6.5	17.1
C	3	60.0	43.0	17.0	9.0	51.0	34.0	16.0	44.0	27.0	7.0	20.6
D	4	60.0	41.5	18.5	14.0	46.0	27.5	22.0	38.0	19.5	8.0	29.1
E	5	60.0	40.5	19.5	.5	59.5	40.0	14.5	45.5	26.0	14.0	35.0
F	6	60.0	40.5	19.5	1.0	59.0	39.5	21.5	38.5	19.0	20.5	51.9
G	7	60.0	40.0	20.0	1.0	59.0	39.0	29.0	31.0	11.0	28.0	71.8
H	8	60.0	42.5	17.5	10.0	50.0	32.5	34.5	25.5	8.0	24.5	75.4
I	9	60.0	41.0	19.0	1.5	58.5	39.5	36.5	23.5	4.5	35.0	88.6
J	10	60.0	41.0	19.0	1.0	59.0	40.0	40.5	19.5	.5	39.5	98.7

REPRODUCIBILITY OF THE WASHING PERFORMANCE TEST

A number of series of washing performance tests were run, following the technique described, on six detergents showing removal efficiencies from about 2.6 to 100 percent. Tests on distilled water containing no detergent were included in this study. Each series of tests included from 6 to 10 determinations. Table 2 summarizes the data obtained in this study, including the mean and standard deviations and the coefficient of variation. Ten tests with distilled water showed a maximum of 3.8 percent and an average of only 1.02 percent removal. The standard deviation, however, was larger than the mean removal which resulted in a very high coefficient of variation of 122 percent. These 10 runs demonstrate that under the technique of the test it is

impossible to remove more than a very small fraction of the Hucker soil with hot distilled water alone. On the other hand, castile soap in distilled water, which was among the best of the detergents selected for the development of this test, showed from 93.1 to 100 percent removal in 10 tests. Here a very low standard deviation and the lowest coefficient of variation in performance of any of the detergents in this series was obtained. The data demonstrated that every test carried out by this technique using castile soap in distilled water showed a very excellent washing performance.

TABLE 2.—*Data showing the reproducibility of the washing-performance test*

Series No.	Detergent	Number of tests	Percentage of soil removed			Deviations		Coefficient of variation
			Minimum	Maximum	Mean	Mean	Standard	$\frac{S. D. \times 100}{M}$
1	None [1]	10	0.0	3.8	1.02	1.02	1.24	122.0
2	A	8	2.6	8.9	5.9	1.9	2.2	37.3
3	B	7	2.8	23.8	13.8	4.5	5.8	42.0
4	C	8	12.8	50.8	34.2	7.9	10.0	29.2
5	D	6	67.4	74.1	70.7	2.6	2.7	3.8
6	E	10	83.5	94.9	90.1	2.5	3.1	3.4
7	Castile soap	10	93.7	100.0	97.6	1.4	1.8	1.9

[1] In distilled water only.

Further examination of the data indicates that as the average percentage removal by a detergent increases, the coefficient of variation in performance is reduced. This relation between the washing performance of a detergent and the coefficient of variation is shown in figure 3. It may be seen from this figure that with poor detergents having washing performances by this technique under 15 percent, the coefficient of variation will probably exceed 40 percent. As the detergent washing performance increases from 15 percent to 30 percent removal, the coefficient of variation decreases below 40 percent. With removal efficiencies above 30 percent, the coefficient of variation is decreased regularly and seems to be of little importance in detergents with removal efficiencies of 70 percent or more.

Even with the poorer detergents which have larger coefficients of variation there is little possiblity of rating them incorrectly with this test. However, because of the possible variations with single tests, particularly with poor detergents, a minimum of four washing performance tests has been tentatively adopted as a satisfactory basis for studying and rating unknown detergents in our laboratory. The practice of making four washing performance tests by the technique described provides a practical and reliable method of grading detergents.
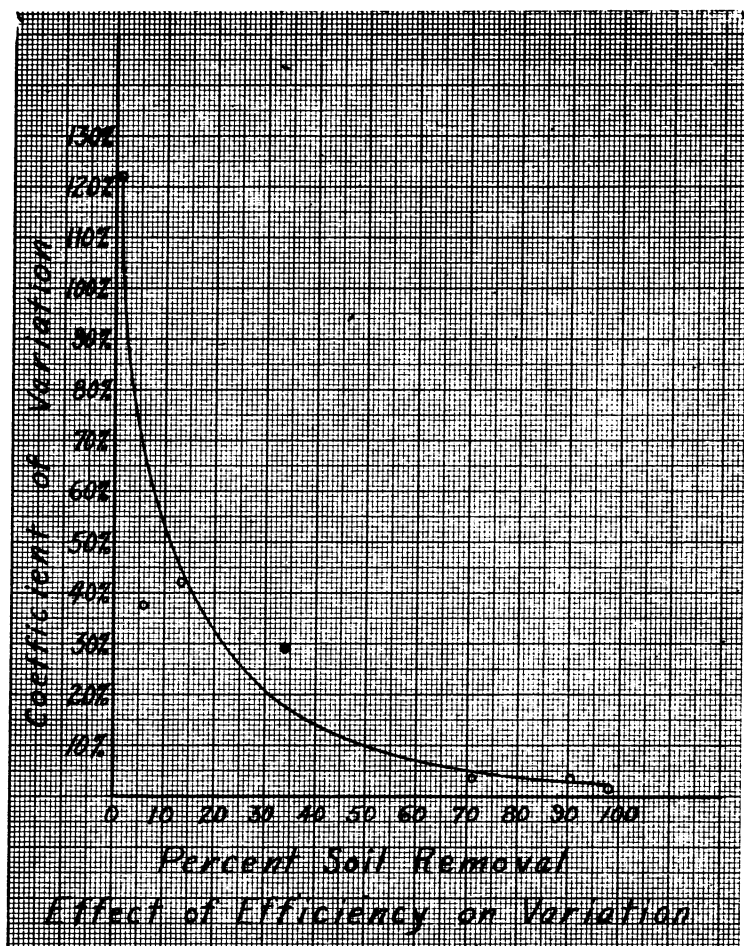
FIGURE 3.

DISCUSSION

It should be stated that the above range of soil removal and degree of reproducibility with detergents will be obtained only if the technique described is adhered to very carefully. .Changes in the washing machine, in the photometer, in the composition of the soil used, and in the technique of soiling, baking, washing, rinsing, and the examination of the slides, will all affect the results obtained. Such changes may prevent obtaining a soil removal range from 1.0 percent with plain distilled water to 98 percent with castile soap in distilled water. An increase in the time or temperature of baking will produce a remarkable reduction in the percentage of soil removed. It is therefore especially important to adhere strictly to the stated time and tempera-

ture in baking the soiled slides in preparation for the test. It was also learned that if the same slides are used repeatedly they become scratched. Soiled, scratched slides are harder to wash clean than unscratched slides. Our practice, therefore, is to discard immediately all slides which are noticeably scratched or etched and replace them with new ones for this test.

SUMMARY

A washing-performance test for studying detergents to be used in the cleansing of dishes and utensils in dairy and restaurant sanitization was developed. The standardized technique for soiling, baking, washing, rinsing, drying, and examining standard microscope slides for use in this test has been described in detail. A detailed description of the special washing machine and photometer developed for use in the test has been given. The photometer observations and calculations for determining the soil-removal efficiencies for a number of detergents have been presented. The equipment and technique have been developed so that the percentage of soil removed from the slides increases from about 5 percent for a very poor detergent to 98 percent for a very good one. A statistical study of the reproducibility of the soil-removal efficiencies obtained on a graded scale of detergents has shown that the test developed is a practical and reliable method of grading dishwashing detergents upon a performance basis.

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A SEROLOGICAL STUDY OF 37 CASES OF TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS) OCCURRING IN BURMA AND THE PHILIPPINE ISLANDS [1]

By IDA A. BENGTSON, *Senior Bacteriologist, United States Public Health Service*

Opportunity was afforded through the courtesy of the United States of America Typhus Commission and of members of the Medical Corps, United States Army, of making a serological study of cases of tsutsugamushi fever occurring in Burma and the Philippine Islands during the years 1944 and 1945. Three groups of specimens are considered in this report. The origin of these specimens was as follows:

Group 1.—Serums from 15 patients at the Twentieth General Hospital, Burma; received from Lt. Col. James S. Forrester, M. C., Chief of Laboratory Service. They were from an outbreak occurring in May 1944, and the clinical symptoms were of more than usual severity. There was one fatality in this group. In some of these cases no original ulcer could be found. Five or six specimens of serum were collected from each case at weekly intervals. A complete clinical history of all cases accompanied the specimens.

Group 2.—Serums from seven convalescent cases of tsutsugamushi disease; collected by Maj. Theodore E. Woodward at the Ninth General Hospital, Philippine Islands, February 2–7, 1945.

Group 3.—Serums from 15 cases of tsutsugamushi disease occurring in the Philippine Islands in April, May, and June 1945; collected by Maj. Theodore E. Woodward, M. C., United States of America Typhus Commission. Most of these cases were from the Island of Luzon. A clinical history of each case accompanied the specimens.

It has previously been shown (*1*) in a serological study of cases of accidental laboratory infections with known strains of virus and of serums from recovered guinea pigs inoculated with known strains, that higher complement-fixation titers were obtained when antiserums were tested against the infecting strain than when they were tested against other strains. Differences as great as one-thousand- or two-thousand-fold in titers against the homologous strain and a heterologous virus strain (Karp) were obtained in one case of laboratory infection with the Gilliam strain. In recovered guinea pigs there were also marked divergencies in the titers of immune serums from different strains, but the differences were of a lower order.

The testing of serums from a number of cases occurring in localities where the disease is prevalent afforded an opportunity to obtain further information concerning the serological relationship of strains. All serums were tested against antigens prepared from the Karp and Gilliam strains and many were also tested against antigens prepared from the Seerangayee strain. The Karp strain originated from a case in New Guinea and the Seerangayee strain from a case in the Federated Malay States. Both of these strains were furnished by Dr. R. Lewthwaite, of the Federated Malay States. The Gilliam strain was from a case infected in Assam. The tests were performed as previously described (*2*), employing two full units of complement and 37° C. water-bath fixation for 1 hour.

The antigens were prepared from infected yolk sacs as has been previously described (*1*). With increased growth in the yolk sac the aqueous portion of ether-treated material yielded antigens of satisfactory potency. Many of the specimens were tested several times, particularly the serial specimens of group 1. These had been pre-

served by the addition of 1.3 mg. sulfanilamide per cubic centimeter which apparently did not affect the results of the test and which afforded protection against contamination, since none of the serums became anticomplementary. The first tests in this group were made in July 1944 and the last tests in August 1945. While the results obtained were not entirely consistent, owing to the varying potency of the antigens and the difficulty of standardizing them (1), still, it was possible to obtain information as to the relationship of the strains used in the tests. The results shown are those in which the same antigens were used throughout the testing of each group of serums, with a few exceptions in group 1 where the sample of antigen had been exhausted.

The results of the tests are shown in tables 1 to 3. Also included are the results of the Weil-Felix test against OXK antigen. In group 1, the results of the Weil-Felix test were furnished by Col. Forrester; in groups 2 and 3 the tests were performed at the National Institute of Health.

In the last column in each table is indicated (by initials) the strain against which highest complement-fixing titers were obtained, K indicating the Karp strain, G the Gilliam strain, and S the Seerangayee strain (tables 1, 2, 3).

TABLE 1.—*Complement-fixation and Weil-Felix reactions of serums from cases of tsutsugamushi disease occurring in Burma* [1]

Case No.	Patient	Serial number of specimen	Approximate date of onset	Date of specimen	Days after onset	Agglutination titer OXK	Complement-fixing titer			Classification
							Karp	Gilliam	Seerangayee	
			1944	*1944*						
9796	Su 13885	I	May 30	June 12	13	1:25	1:2,048	1:128	--------	K(S)
9797		II		June 19	20	1:100	--------	--------	--------	
9798		III		June 26	27	1:50	--------	1:32	--------	
9977		IV		July 4	35	1:100	1:2,048	1:8	1:2,048	KS
9978		V		July 12	43	Neg.	1:1,024	1:8	1,024	KS
9979		VI		July 17	48	1:25	1:1,024	1:8	--------	K(S)
9799	Ta 13649	I	May 23	June 12	20	1:25	1:4	1:8,192	--------	G
9800		II		June 19	27	1:25	1:4	1:8,192	--------	G
9801		III		June 29	34	1:50	--------	--------	--------	
9802		IV				Neg.				
10010		V		July 12	50	1:25	Neg.	1:1,024	Neg.	G
10011		VI		July 17	55	Neg.	1:512	1:256	1:2,048	¹KGS
9802	Sh 13736	I	May 23	June 12	20	1:100	--------	--------	--------	
9803		II		June 19	27	1:50	--------	--------	--------	
9804		III		June 26	34	1:50	--------	--------	--------	
10003		(III)		June 28	36	--------	Neg.	1:32,768	1:32	G
9994		IV		July 4	42	--------				
9995		V		July 10	48	--------	1:8	1:8,192	1:32	G
9996		VI		July 17	55	--------	1:8	1:8,192	1:32	G
9805	Ra 14491	I	May 31-June 1	June 12	12	Neg.	1:256	1:32	1:2,048	S
9806		II		June 19	19	1:100	--------	--------	--------	
9807		III		June 29	26	1:50	1:64	1:8	--------	(S)
10009		(III)		July 1	31	--------				

See footnotes at end of table.

TABLE 1.—*Complement-fixation and Weil-Felix reactions of serums from cases of tsutsugamushi disease occurring in Burma*—Continued

Case No.	Patient	Serial number of specimen	Approximate date of onset	Date of specimen	Days after onset	Agglutination titer OXK	Complement-fixing titer			
							Karp	Gilliam	Seerangayee	Classification
			1944	*1944*						
10006		IV		July 4	34	1:50				
10007		V		July 12	42	Neg.	1:16	1:16	1:128	S
10008		VI		July 17	47	Neg.	1:16	1:16	1:128	S
9808	Va 13697	I	May 25	June 12	18	1:25				
9809		II		June 19	25	Neg.	1:16	1:8,192		G
9810		III		June 26	32	Neg.				
		IV				Neg.				
10004		V		July 10	46	1:4	1:4	1:1,024	1:32	G
10005		VI		July 17	53	1:50	1:4	1:512	1:16	G
9811	Sc 13641	I	May 21	June 12	22	1:400	1:1,024	1:128	1:4,096	KS
9812		II		June 19	29	1:200				
9813		III		June 26	36	1:50	1:512	1:128	1:2,048	KS
9980		IV		July 4	44	1:25	1:256	1:32	1:1,024	KS
9981		V		July 10	50	0	1:128	1:64	1:512	KS
9982		VI		July 17	57	0	1:8	1:1,024	1:32	² G
9814	Gr 14242	I	May 30	June 12	13	1:400	1:2,048	1:256	1:4,096	KS
9815		II		June 19	20	1:1,600	1:2,048	1:256		K(S)
9816		III		June 26	27	1:1,600				
9985		IV				1:400				
9986		V		July 10	41	1:100	1:1,024	1:256	1:4,096	KS
9987		VI		July 17	48	1:100	1:2,048	1:64	1:2,048	KS
9817	Ne 13692	I	May 21	June 12	22	1:200		1:4,096		G
9818		II		June 19	29	1:100	1:64	1:4,096	1:512	G
9819		III		June 26	36	1:50				
9988		IV		July 3	43	1:25				
9989		V		July 10	50	Neg.	1:16	1:1,024	1:64	G
9990		VI		July 17	57	Neg.	1:8	1:512	1:64	G
9820	Ba 13903	I	May 24	June 12	19	1:100				
9821		II		June 19	26	1:25	1:2,048	1:256	1:8,192	KS
9822		III		June 26	33	1:50				
9991		IV		July 4	41	1:100				
9992		V		July 10	47	1:25	1:256	1:64	1:1,024	KS
9993		VI		July 17	54	Neg.	1:64	Neg.	1:512	(K)S
9823	Rh 13779	I	May 19	June 12	24	1:800		1:65,536		G(S)
9824		II		June 19	31	1:800				
9825		III		June 26	38	1:400				
9997		IV		July 4	46	1:200		1:8,192		G(S)
9998		V		July 10	52	1:100	1:128	1:4,096	1:16,384	GS
9999		VI		July 17	59	Neg.	1:64	1:2,048	1:8,192	GS
9826	Ma 14016	I	May 14	June 12	29	1:800	1:32,768	1:32,768		KG(S)
9827		II		June 19	36	1:800				
9828		III		June 26	43	1:400	1:512	1:128	1:1,024	KGS
9983		IV		July 4	51	1:200	1:1,024	1:512	1:1,024	KGS
		V				1:100				
9984		VI		July 17	64	1:50	1:256	1:512	1:1,024	KGS
9829	La 13843	I		June 12		1:50	1:8,192	1:128	1:128	K
9830		II		June 19		1:100				
9831	Le 13673	I	May 23	June 12	20	1:800	1:8,192	1:1,024	1:65,536	KS
9832		II		June 19	27	1:400	1:16,384			K(S)
9833		III		June 26	34	1:200	1:8,192	1:1,024		K(S)
10000		IV		July 5	43	1:50				
10001		V		July 12	50	1:50	1:1,024	1:256	1:8,192	KS
10002		VI		July 17	57	1:25	1:512	1:128	1:4,096	KS
9834	Ca 14474	I	May 26	June 12	17	1:100	1:64	1:131,072		G
9835	We 14164	I	May 24	June 12	19	Neg.	1:4			
9836		II		June 19	26	1:50				
9837		III				Neg.				

¹ Collected at the Twentieth General Hospital, June, July 1944 (group 1).
² See text p. 889.

TABLE 2.—*Complement-fixation and Weil-Felix reactions of serums from cases of tsutsugamushi disease occurring in the Philippine Islands* [1]

Case No.	Pa-tient	Locality where infec-tion occurred	Date of specimen	Aggluti-nation titer OXK	Complement-fixing titer			
					Karp	Gilliam	Seeran-gayee	Classifi-tion
10838	Mi	Mindoro_____	32d day_____	1:320	1:8,192	1:8,192	1:4,096	KGS
10839	Ba	Sansapor_____	6th month_____	Negative	1:16	Negative	1:8	K(G)S
10840	Bu	Wadki Island_____	2d month_____	1:20	1:16	1:64	1:64	KGS
10841	Al	Sansapor_____	4th month_____	1:20	1:4	1:2,048	1:4	G
10842	Sa	Sansapor_____	6th month_____	1:20	1:4	1:512	Negative	G
10843	Lo	Mindoro_____	33d day_____	1:640	1:8,192	1:16,384	1:64	KG
10844	Sn	Biak_____	76th day_____	Negative	1:16	1:32	1:16	KGS

[1] Collected at the Ninth General Hospital Feb. 2–7, 1945 (group 2).

TABLE 3.—*Complement-fixation and Weil-Felix reactions of serums from cases of tsutsugamushi disease occurring in the Philippine Islands* [1]

Case No.	Patient	Date of onset	Date of speci-men	Days after onset	Aggluti-nation titer OXK	Complement-fixing titer			
						Karp	Gilliam	Seeran-gayee	Classifi-cation
		1945	*1945*						
11272_____	Os	Apr. 18	May 12	24	1:640	1:8,192	1:8,192	1:4,096	KGS
11273_____	Hc	Apr. 18	May 23	35	1:320	1:64	1:32,768	1:64	G
11274_____	Jp	Apr. 30	May 29	29	1:1,280	1:2,048	1:2,048	1:4,096	KGS
11275_____	Wm	May 6	May 30	24	1:80	1:2,048	1:128	1:512	KS
11276_____	Tb	May 7	May 30	23	1:40	1:128	1:128	1:64	KGS
11277_____	Es	May 6	May 29	23	1:160	1:128	1:2,048	1:64	G
11278_____	Mz	May 25	June 12	18	1:640	1:2,048	1:128	1:2,048	KS
11279_____	Hm	May 17	June 12	26	1:320	1:4,096	1:16,384	1:4,096	KGS
11280_____	Lm	May 27	June 12	16	1:1,280	1:16,384	1:32,768	1:32,768	KGS
11281_____	Gb	May 16	June 13	28	1:40	1:256	1:256	1:512	KGS
11282_____	Lc	May 13	June 12	30	Negative	1:4	1:8,192	1:32	G
11283_____	Lg	May 15	June 12	28	1:320	1:32,768	1:8,192	1:4,096	KGS
11284_____	Cb	May 16	June 12	27	1:640	1:512	1:128	1:512	KGS
11285_____	Db	May 14	June 12	29	1:320	1:32,768	1:4,096	1:1,024	K
11286_____	Em	June 1	June 12	11	1:20	1:256	1:8	1:4	K
11287_____	Hm	May 9	June 12	34	1:320	1:32,768	1:8,192	1:256	KG

[1] Collected June 1945 (group 3).

DISCUSSION

The results of the tests as shown in tables 1, 2, and 3 do not indicate any clear differentiation of serological types as may have been suggested by tests of serums from laboratory cases infected with known strains (*1*). Though a certain number show a decidedly higher complement-fixing titer against either the Karp, Gilliam, or Seeran-gayee strain, a considerable number of cases have the same or approximately the same titer against all three of the strains used in the preparation of antigens. It therefore appears that the strains may be more closely related than was suggested by the results of tests on serums from cases infected with known strains.

Among the 23 Philippine cases (tables 2 and 3), 12 show the inclusive type of reaction; the antigens from the Karp, Gilliam, and Seeran-

gayee strains reacting to approximately the same titer (KGS). In five cases the Gilliam strain reacted to a decidedly higher titer than the Karp and Seerangayee strains (cases No. 11273, 11277, 11282, 10841, 10842) (G). In two cases (cases No. 11285 and 11286) a considerably higher titer was obtained against the Karp strain than against the Gilliam and Seerangayee strains (K). In two cases (No. 11275 and 11278), titers against the Karp and Seerangayee strain were higher than against the Gilliam strain (KS) and in two others (No. 11287 and 10843) higher titers were obtained against the Karp and Gilliam strains than against the Seerangayee strain (KG).

Among the Burma cases the KGS combination occurs consistently in only one instance (Ma 14016). There are five G cases (Ta 13649, Sh 13736, Va 13697, Ne 13692, Ca 14474); one K case (La 13843); one S case (Ra 14491); five KS cases (Su 13885, Sc 13641, Gr 14242, Ba 13903, Le 13673); one GS case (Rh 13779).

The results are summarized in table 4.

TABLE 4.—*Summary of results of complement-fixation tests*

Predominant antigenic factors	Philippine cases	Burma cases	Total
K	2	1	3
G	5	5	10
S		1	1
KGS	12	1	13
KS	2	5	7
KG	2		2
GS		1	1

The results tabulated in table 4 may be represented graphically as shown in figure 1. All possible combinations of KG and S are represented in the results of the tests on the serums from the 37 cases. It thus appears that the Karp, Gilliam, and Seerangayee strains do not represent separate complement-fixing types but that they are more closely related. Antibodies present in the different serums may contain either one, two, or three major components which react with the corresponding antigens. The great majority of the cases studied had antibodies responding to K or G antigens separately or to each of these together with other antigens. Only one case (Ra 14491) was found to be an S case and the serum from this case had a titer of 1:2,048 with S antigen and 1:256 with K antigen on the twelfth day. Later in the course of the illness these titers were reduced to 1:128 against the S antigen and 1:16 against the K antigen. Thus by the use of the K and G antigens, antibodies would have been detected in all the serums tested. The Gilliam strain appears to be particularly distinctive (1), and the Karp strain differs in several respects from the Gilliam strain. It therefore seems

indicated that these two strains should be employed as antigens unless further tests may show that other strains should also be included, or unless a strain is found which is of an inclusive nature, so that it will elicit a response from all antibody combinations.

The results obtained with the serial specimens from the Burma cases were fairly consistent throughout, falling in orderly fashion
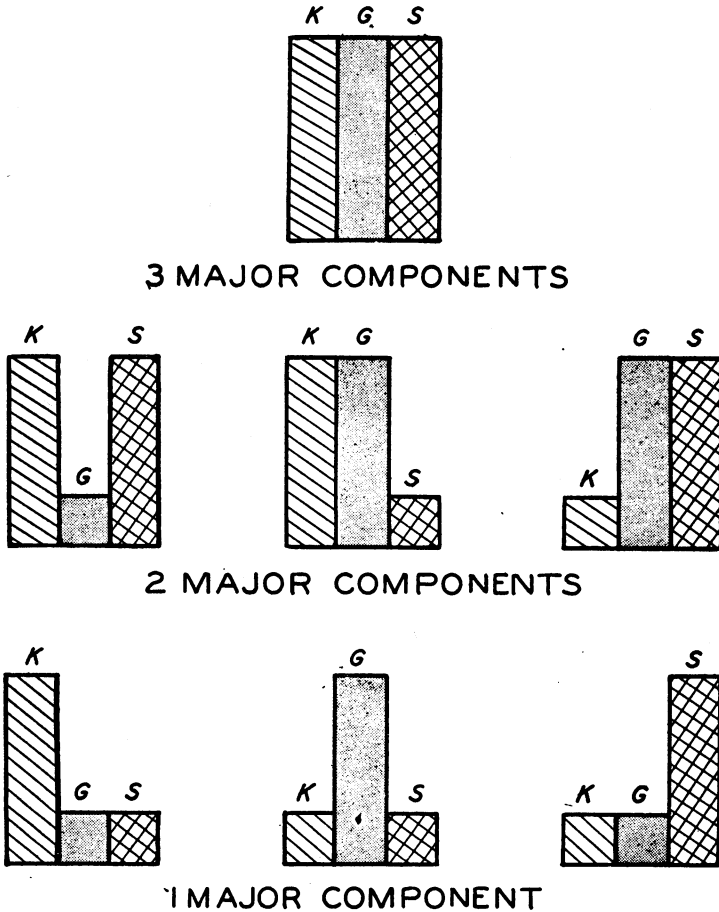
FIGURE 1.—Karp, Gilliam, and Seerangayee antibody patterns.

following initial high titers, with the exception of a few discrepancies which will be considered later. High titers were present in 12, 13, 17, 20, 22, 24, and 27 days (1:2,048, 1:4,096, 1:65,536, 1:131,072). High titers also persisted through 36, 41, 50, 52, 57, and 59 days (1:2,048, 1:4,096, 1:8,192 1:16,384). The highest titer recorded was that of case Ca 14474 which was fatal. The serum collected on the seventeenth day had a titer of 1:131,072.

Two discrepancies (table 1) in the results are noticeable, that of 10011 (specimen 6 of Ta 13649) and 9982 (specimen 6 of Sc 13641). In the first instance unexpectedly high titers were obtained against Karp and Seerangayee antigens whereas negative results against these two antigens had been obtained with a specimen collected 5 days earlier. Likewise specimen 9982 had an unexpectedly high titer against the Gilliam antigen, while the specimen collected 7 days earlier had had a low titer against this strain. Tests were repeated several times with the same and with different antigens, with approximately the same results. No clue to indicate that the specimens had been mislabeled could be found. It is to be noted in both of these cases that titers of the preceding specimens against the other antigens approximated those of the later specimens against the antigens under discussion. If these results are valid it would appear that the relationship between the three strains is still closer than has been suspected.

In general, considerably higher titers were obtained in the complement-fixation test than in the agglutination test using OXK as antigen. Highest titers obtained in the Weil-Felix agglutination test were 1:1,600 (twentieth, twenty-seventh days); 1:1,280 (sixteenth, twenty-ninth days). Significant complement-fixation reactions persisted after the agglutination titer had fallen to negative or 1:20 and 1:40.

SUMMARY

A serological study by complement fixation, of 37 cases of tsutsugamushi disease occurring in Burma and the Philippine Islands revealed a variety of antigenic responses to the three strains of tsutsugamushi used in the tests, namely the Gilliam, Karp, and Seerrangayee strains. Certain cases were predominantly of the Gilliam type, others of the Karp or Seerangayee type, and the serums of a number of cases responded equally well to all three types. Cross fixation occurred in practically all cases. The results obtained as a whole do not indicate any clear differentiation of serological types. The Karp and Gilliam strains appear sufficiently distinctive, however, to warrant the use of these two in the testing of serums from cases of suspected tsutsugamushi illness. Weil-Felix titers with OXK antigen were much lower than complement-fixing titers with rickettsial antigens and persisted for a shorter time.

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COMPLEMENT FIXATION IN TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS) [1]

By Ida A. Bengtson, *Senior Bacteriologist, United States Public Health Service*

A complement-fixation test has been developed against a strain of rickettsiae designated as tsutsugamushi Karp along the lines previously described for endemic and epidemic typhus and Q fever (1). Various frozen specimens of chick embryo material inoculated with this strain were received from Dr. Lewthwaite (4) of the Federated Malay States through the courtesy of the National Naval Medical Research Institute.

The propagation of the strain was continued in yolk sacs of fertile eggs by the Cox method (2), by inoculation of mice and guinea pigs, and into the anterior chamber of rabbits' eyes. The growth in the yolk sac was poor at first but as serial passages were continued it improved. Intraperitoneal inoculations in guinea pigs elicited a febrile illness characterized by approximately a 5-day incubation period and fever (above 39.6° C.) for about 4 days. The strain is fatal for white mice when inoculated intraperitoneally with either yolk-sac virus or guinea pig passage material (liver or liver-and-spleen suspension). When yolk-sac passage virus is inoculated into the anterior chamber of the rabbit's eye an iritis and then an ophthalmitis is evoked, similar to that described for tsutsugamushi disease (3). Rickettsia-like bodies can be easily demonstrated in the infected yolk sacs, and in various infected animal tissues.

Antigens were prepared from the yolk sacs of infected eggs. Serums from a number of recovered inoculated guinea pigs furnished by Dr. Norman H. Topping [2] and six human convalescent serums were available for testing. Two units of hemolysin, two full units of complement, and four units of antigen (four times the highest dilution showing complete or almost complete fixation with control guinea pig serum) were employed in the tests.

The serums of two guinea pigs inoculated September 25, 1943, and October 2, 1943, respectively, and bled October 15, 1943, were arbitrarily diluted 1:8 and tested October 20, 1943, against two experimental antigens K1 and K2. Of these, K2 was shown to be the better, complete fixation being obtained in dilution 1:2 and partial fixation in dilution 1:4. K1 showed complete fixation when used undiluted. Antigen K2 was therefore employed in testing the serums of four inoculated guinea pigs October 23. Of these, one guinea pig serum, No. 16, showed complete fixation in dilution 1:64 and partial

fixation in 1:128. The other three showed slightly less than complete fixation in dilution 1:4. The highest titered serum was used thereafter in the titration of other antigens until the supply was exhausted.

Antigens K1 and K2 had been prepared according to the methods previously employed in the preparation of epidemic typhus antigen. Ten-percent suspensions of infected yolk sac in normal saline containing 1:10,000 merthiolate were prepared in a Waring Blendor. The suspensions were left standing overnight and were then treated with an equal volume of ethyl ether in a separatory funnel. Usually rather good separation was obtained though the aqueous layer was often more or less turbid. A layer, to be referred to as "emulsion," between the lower aqueous layer and the ether layer contained most of the tissue.

Though the aqueous phase was used as antigen in the earlier part of the work, it was found that much of the antigenic-substance was contained in the emulsion and that it was more concentrated here, as a rule, than in the aqueous portion. Since the emulsion was not anticomplementary, it was found feasible to use this material as antigen in spite of its turbidity. On standing, a fluid layer usually separated in the lower part of the container, or, if the original material contained more fluid than emulsion, a precipitate formed. However, by mixing, a uniform suspension could easily be obtained. If the emulsion was too concentrated a small amount of the aqueous portion was added. This antigen, though admittedly crude, gave satisfactory results in all tests.[3]

The presence of a considerable portion of the antigenic substance in the emulsion layer might be related to the fact that most of the rickettsiae appear to be embedded in certain yolk-sac material from which they are not easily separated. Lewthwaite (4) describes this material as "circumscribed islets of lipoid tissue having the histological appearance of fatty or areolar tissue." The rickettsiae are not often seen in formed cells and are not numerous as free organisms.

A titration of antigen K19 illustrates the distribution of the antigenic substance in the different portions. The aqueous portion of this material showed rather numerous rickettsiae (++). The emulsion, also showing a fair number of rickettsiae, appeared to be in two layers which were taken off separately. The volume of each portion and results of the complement-fixation test are shown in table 1.

In the case of certain other experimental antigens no rickettsiae were visible in the aqueous portion and complement fixation was nega-

tive. The reaction of the aqueous portions which were tested varied from pH 7.2 to pH 7.8, no adjustment of the reaction having been made prior to ether treatment. The supernatant fluids of the aqueous portions of two different positive experimental antigens gave negative complement fixation after spinning for 1 hour in the angle centrifuge at 4,000 revolutions per minute.

TABLE 1.—*Distribution of antigenic substance in antigen K19*

	1:1	Dilutions of antigen					
		1:2	1:4	1:8	1:16	1:32	1:64
K19 aqueous (85 cc.)	4	4—	1	0	0	0	
K19 emulsion 1 (1 cc.)			4	4	4	2	1
K19 emulsion 2 (9½ cc.)		4	4—	3	Trace	0	0

Attempts were made to free the rickettsiae from the tissue in the emulsion by various means—freezing and thawing, shaking with glass beads, grinding with alundum, and digestion with trypsin. Using the first three methods the emulsion was freed of most of the fluid by centrifugation. After subjection to the various processes, the emulsion was shaken up with a volume of saline equivalent to the amount of fluid that had been removed. The suspension was centrifuged at low speed and the supernatant fluid titrated. No increase in titer was obtained. The precipitate also was titrated, with results similar to those obtained with the untreated emulsion. It was therefore evident that the antigenic substance was still contained in the tissue. Trypsin destroyed the antigenicity, though further investigation of this and other methods is indicated.

As indicated by microscopic examination, rickettsiae were fairly numerous in some of the yolk sacs used for antigen material, but in spite of this no high-titered antigens were obtained as in epidemic and endemic typhus. Titers reached 1:4 or 1:8 in contrast to 1:16 up to 1:64, or occasionally higher with the latter.

Eight other guinea pig serums in addition to those shown, and six human serums have been tested. The guinea pig serums were obtained from animals which had been inoculated with liver-and-spleen emulsions from infected guinea pigs and had had a typical febrile period. Three of the human serums were obtained from recovered cases in New Guinea through the United States of America Typhus Commission and three were procured from the India-Burma border through Lt. Col. M. H. P. Sayers, Assistant Director of Pathology, Fourteenth Army.

Tables 2 and 3 show the results of tests on a number of guinea pig serums. Table 4 shows results with human serums.

TABLE 2.—*Titration of 8 guinea pig serums against Karp antigen K2 (undiluted)*

Guinea pig No.	Days after fever	Dilutions of serums							
		1:4	1:8	1:16	1:32	1:64	1:128	1:256	1:512
13	31	4	4	4	4	4—	1	0	0
18	34	4	4	4	4	4	4—	4—	1
19	30	4	4	4	4	4	4	4—	0
22	34	4	4	4	4	4	3	3	1
35	26	4	4	4	4	4	4—	4—	4—
38	20	4	4	4	4—	3	1	1	1
41	22	4	4	4	4—	1	0	0	0
51	9	2	1	1	1	0	0	0	0

The serum from guinea pig 16 obtained 13 days after defervescence was tested against hetrologous antigens as well as the homologous with results shown in table 3.

TABLE 3.—*Titration of guinea pig serum showing results against heterologous antigens*

Antigen	Dilutions of serums							
	1:4	1:8	1:16	1:32	1:64	1:128	1:256	1:512
Karp	4	4	4	4	4	3	0	
Endemic typhus	0	0	0	0				
Epidemic typhus	0	0	0	0				
Rocky Mountain spotted fever	0	0	0	0				
Q fever	0	0	0	0				

Tests for specificity were done with the following heterologous serums:

Epidemic typhus—complement-fixation titer against homologous antigen 1:256
Endemic typhus—complement-fixation titer against homologous antigen_ 1:512
Rocky Mountain spotted fever—complement-fixation titer against homologous antigen_____ 1:1, 024
Undulant fever—agglutination titer against homologous antigen_____ 1:160
Undulant fever—agglutination titer against homologous antigen_____ 1:1, 280
Tularemia—agglutination titer against homologous antigen_____ 1:5, 120
Tularemia—agglutination titer against homologous antigen_____ 1:1, 280
Typhoid fever—agglutination titer against homologous antigen_____ 1:320
Syphilis—complement-fixation titer (Albany method)_____ 11. 4

No fixation was obtained in any dilution of these serums from 1:4 to 1:32 against the Karp antigen.

TABLE 4.—*Titration of human convalescent serums*

Antigen	Case No.	Day of illness	1:4	1:8	1:16	1:32	1:64	1:128	1:256	1:512	1:1,024	1:2,048	1:4,096	Well-Felix OXK
Cases from New Guinea														
Karp K9A	2	28th	4-	2	1	1-	0	0	0	0				1:1,280
Endemic typhus			0	0	0	0	0	0	0	0				
Karp K9A	13	29th	4	4	4	4	4	4	4	4-	0			1:5,120
Endemic typhus			0	0	0	0	0	0	0	0				
Karp K9A	15	29th	4	4	4	4	2	1	0	0				1:2,560
Endemic typhus			0	0	0	0	0	0	0	0				
Cases from India-Burma border														
Karp K9A	5624329	3d–4th week	4	4	4	4	4	4	4	4-				1:2,560
Epidemic typhus			0	0	0	0								
Endemic typhus			0	0	0	0								
Rocky Mountain spotted fever			0	0	0	0								
Q fever			0											
Karp K9A	10870302	3d–4th week	4	4	4	4	4	4	4	4	4	4	1	1:20,480
Epidemic typhus			0	0	0	0								
Endemic typhus			0	0	0	0								
Rocky Mountain Spotted fever			0	0	0	0								
Q fever			0											
Karp K9A	2067330	3d–4th week	4	4	4	4	4	4	4-	3				1:20,480
Epidemic typhus			0	0	0	0								
Endemic typhus			0	0	0	0								
Rocky Mountain spotted fever			0	0	0	0								
Q fever			0											

SUMMARY

A complement-fixation test for the Karp strain of tsutsugamushi (scrub typhus) has been developed. It appears to be specific as far as tested for the disease, and shows good agreement with the results of the Weil-Felix tests with OXK antigen.

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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

April 21–May 18, 1946

The accompanying table summarizes the prevalence of nine important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State for each week are published in the Public Health Reports under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4 weeks ended May 18, 1946, the number reported for the corresponding period in 1945, and the median number for the years 1941–45.

DISEASES ABOVE MEDIAN PREVALENCE

Diphtheria.—For the 4 weeks ended May 18 there were 1,068 cases of diphtheria reported, as compared with 816 for the corresponding period in 1945 and a 1941–45 median of 780 cases. The incidence was above the preceding 5-year median expectancy in all sections of the country except the East South Central; there the number of cases was slightly below the normal seasonal incidence. For the country as a whole the current incidence was the highest since 1939 when approximately 1,200 cases were reported for the corresponding 4 weeks.

Measles.—The number of reported cases of measles dropped from 153,000 for the preceding 4 weeks to 147,500 during the current 4 weeks. The number was almost 8 times that reported for the corresponding 4 weeks in 1945 and 1.4 times the 1941–45 median. All sections of the country except the East North Central reported an increase over the preceding 5-year median, the numbers of cases ranging from 1.2 times the median in the East South Central section to 3.3 times the median in the Middle Atlantic section. With the exception of the year 1941 when 172,000 cases were reported the current incidence is the highest for this period in the 18 years for which these data are available.

Poliomyelitis.—For the 4 weeks ended May 18 there were 210 cases of this disease reported. The number represents an increase of more than 50 percent over the 1945 figure for this period and is more than twice the 1941–45 median for the corresponding weeks. Of the total number of cases reported, 53 occurred in Florida, 32 in Texas, 24 in California, 15 in New York, and 8 in Louisiana; the remaining cases were widely scattered, and no more than 6 cases were reported from any State. Since the beginning of the year there have been 116 cases of poliomyelitis in the State of Florida, 21 cases and 1 death occurring in Miami, 12 cases and 2 deaths in Tampa, 24 cases in Palm Beach County, with the remaining cases scattered over the State.

DISEASES BELOW MEDIAN PREVALENCE

Influenza.—The number of cases of influenza was relatively low, the current incidence (3,873 cases) being about 70 percent of the 1941–45 median, which is represented by the 1945 figure (5,272 cases). The disease was about normal in the New England and Middle Atlantic sections, but in all other parts of the country the numbers of cases were considerably below the normal seasonal expectancy.

Meningococcus meningitis.—The number of cases (428) of this disease reported for the 4 weeks ended May 18 was the lowest for this period since 1942 when there were 389 cases. The number for the current period was about 60 percent of the 1941–45 median. The comparison with the 1941–45 median is favorable because it included 3 years in which this disease was unusually prevalent, but the incidence is still considerably above the average of normal years (approximately 200 cases).

Scarlet fever.—The scarlet fever incidence was also relatively low, the number of cases (13,617) being about 70 percent of the 1945 figure for this period and 85 percent of the median for the corresponding periods in 1941–45. The South Atlantic and Pacific sections reported a few more cases than might normally be expected, but in all other sections the numbers of cases were comparatively low.

Number of reported cases of 9 communicable diseases in the United States during the 4-week period April 21–May 18, 1946, the number for the corresponding period in 1945, and the median number of cases reported for the corresponding period, 1941–45

Division	Current period	1945	5-year median	Current period	1945	5-year median	Current period	1945	5-year median
	Diphtheria			Influenza [1]			Measles [2]		
United States	1,068	816	780	3,873	5,272	5,272	147,499	19,081	104,755
New England	48	26	26	9	116	14	13,252	1,369	8,089
Middle Atlantic	173	85	112	44	25	41	49,905	2,229	14,927
East North Central	148	73	117	170	200	301	28,564	2,593	19,422
West North Central	137	93	73	23	59	102	5,337	869	7,512
South Atlantic	187	153	142	1,232	1,207	1,577	12,944	968	7,852
East South Central	52	71	64	117	121	374	2,796	449	2,269
West South Central	148	155	124	1,983	2,868	2,245	9,766	2,267	6,894
Mountain	68	51	51	212	611	476	8,030	1,522	4,324
Pacific	107	109	87	83	65	290	16,905	6,815	7,313
	Meningococcus meningitis			Poliomyelitis			Scarlet fever		
United States	428	712	712	210	136	103	13,617	19,001	15,612
New England	25	34	48	5	5	3	1,271	2,023	2,023
Middle Atlantic	104	156	156	18	21	8	4,577	5,262	4,590
East North Central	87	133	133	8	12	8	3,681	4,756	4,189
West North Central	34	49	49	13	6	4	914	1,621	1,153
South Atlantic	54	93	93	66	24	16	1,276	1,692	1,104
East South Central	35	71	71	5	12	12	263	507	507
West South Central	38	68	68	42	45	19	277	480	337
Mountain	4	15	15	24	1	3	470	765	765
Pacific	47	93	93	29	10	12	888	1,895	806
	Smallpox			Typhoid fever, para-typhoid fever			Whooping cough [3]		
United States	41	38	75	249	281	345	8,037	10,548	15,291
New England	0	0	0	7	9	20	965	1,144	1,144
Middle Atlantic	0	0	0	31	38	44	1,703	2,193	2,640
East North Central	16	18	18	26	41	39	1,683	1,316	3,367
West North Central	6	2	9	16	9	14	324	304	475
South Atlantic	2	1	4	48	51	71	1,143	1,629	1,629
East South Central	0	7	9	16	39	40	216	331	641
West South Central	3	5	9	64	68	68	911	1,341	1,341
Mountain	3	4	4	17	16	16	479	523	536
Pacific	11	1	3	24	10	19	613	1,767	1,767

[1] Mississippi and New York excluded; New York City included.
[2] Mississippi excluded.

Smallpox.—Of the total of 41 cases of smallpox reported for the current 4 weeks, 11 occurred in Indiana, 11 in the State of Washington and 5 in Iowa; the remaining cases were widely scattered over the country, no State reporting more than 2 cases. In Washington where an outbreak of this disease occurred during the last week of March, the cases dropped from 19 for that week to 2 for the week ended May 18. In all sections except the Pacific the current incidence either approximated the median or fell below it.

Typhoid and paratyphoid fever.—For the 4 weeks ended May 18 there were 249 cases of these diseases reported, as compared with 281 in 1945 and a 1941–45 median of 345 cases. For the country as a whole the current incidence was the lowest for this period in the 18 years for which these data are available. The situation was

favorable in all sections of the country; 4 sections reported about the normal number of cases and in the other 5 the incidence was relatively low.

Whooping cough.—The number of cases of whooping cough (8,037) was about 75 percent of the number reported for the corresponding weeks in 1945 and 50 percent of the 1941–45 median expectancy. Each section of the country except the East and West North Central reported fewer cases than in 1945, while in all sections the current incidence was lower than the preceding 5-year median figures.

MORTALITY, ALL CAUSES

For the 4 weeks ended May 18 there were 36,467 deaths from all causes reported to the Bureau of the Census by 93 large cities. The average number of deaths for the same weeks in the years 1943–45 was 37,000. The number of deaths was lower in each of the weeks in the current period than the average for the corresponding week in the 3 preceding years.

DEATHS DURING WEEK ENDED MAY 18, 1946

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

	Week ended May 18, 1946	Correspond-ing week, 1945
Data for 93 large cities of the United States:		
Total deaths	8, 901	9, 202
Average for 3 prior years	9, 047	
Total deaths, first 20 weeks of year	196, 267	190, 001
Deaths under 1 year of age	611	539
Average for 3 prior years	589	
Deaths under 1 year of age, first 20 weeks of year	12, 216	12, 470
Data from industrial insurance companies:		
Policies in force	67, 170, 616	67, 314, 940
Number of death claims	11, 946	13, 633
Death claims per 1,000 policies in force, annual rate	9. 3	10. 6
Death claims per 1,000 policies, first 20 weeks of year, annual rate	10. 7	11. 0

PREVALENCE OF DISEASE

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

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED MAY 25, 1946

Summary

Of the total of 10 cases of smallpox reported during the week, 5 occurred in Indiana, 3 in Colorado, and 1 each in Minnesota and Kansas. The total for the year to date is 214 as compared with 211 for the corresponding period last year, and a 5-year (1941–45) median of 471 (see p. 911).

Of the total of 77 cases of poliomyelitis reported, as compared with 84 last week and 28 for the 5-year median, only 4 States reported more than 2 cases each—Texas 23 (last week 10), Florida 22 (last week 18), California 9 (last week 11), and Louisiana 5 (last week 5). The total to date is 890, as compared with 740 for the period last year and a 5-year median of 499. The total since March 16 (the approximate date of lowest 3-week moving average in each of the years 1944, 1945, and 1946) is 424, as compared with 343 for the same period last year.

The incidence of measles declined during the week in all of the 9 geographic areas except the New England area (increased from 3,257 to 3,890) and the East South Central area (increased from 416 to 559 cases). The current total is 29,444, as compared with 32,317 last week and a 5-year median of 19,116. The total for the year to date is 516,099, as compared with a 5-year median of 422,983.

Of the current total of 277 cases of diphtheria, as compared with 259 last week and a 5-year median of 189, 35 occurred in Texas, 23 in New York, 18 in Maryland, and 16 each in Pennsylvania and California. The cumulative total, 7,206, is 27 per cent above the average for the corresponding periods of the past 6 years. The largest number reported for a corresponding period in those years was 6,849, in 1940.

During the current week, 8,875 deaths were recorded in 93 large cities of the United States, as compared with 8,901 last week, 9,033 and 8,638 for the corresponding weeks, respectively, of 1945 and 1944, and a 3-year (1943–45) average of 8,945. The cumulative number is 205,142, as compared with 199,034 for the corresponding period last year.

(904)

Telegraphic morbidity reports from State health officers for the week ended May 25, 1946, and comparison with corresponding week of 1945 and 5-year median

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

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median 1941-45	Week ended—		Median 1941-45	Week ended—		Median 1941-45	Week ended—		Median 1941-45
	May 25, 1946	May 26, 1945		May 25, 1946	May 26, 1945		May 25, 1946	May 26, 1945		May 25, 1946	May 26, 1945	
NEW ENGLAND												
Maine	3	0	0				354		81	0	0	1
New Hampshire	0	0	0				37	7	27	0	0	0
Vermont	0	0	0				62	17	88	0	0	0
Massachusetts	4	4	3				2,738	286	968	2	6	6
Rhode Island	0	0	0		26		106	4	9	0	0	0
Connecticut	1	0	0		2	2	593	117	400	2	1	4
MIDDLE ATLANTIC												
New York	23	10	13	¹2	¹3	¹3	3,323	143	776	12	26	26
New Jersey	6	1	2	5	5	2	3,455	64	925	6	11	11
Pennsylvania	16	12	9	2		1	3,184	504	1,143	9	9	9
EAST NORTH CENTRAL												
Ohio	12	5	7	3	7	12	745	94	412	8	6	6
Indiana	14	7	5		6	6	373	49	162	3	4	2
Illinois	4	7	13	1	1	9	625	299	419	9	23	16
Michigan ²	5	6	4	1	1	2	926	288	886	4	4	4
Wisconsin	4	3	2	16	24	24	2,471	73	1,644	3	2	1
WEST NORTH CENTRAL												
Minnesota	8	4	3		2		71	15	476	3	2	2
Iowa	1	2	2				231	76	226	0	3	2
Missouri	3	1	2	5		1	113	40	189	3	17	12
North Dakota	1	1	0	5	7	4	13	1	15	0	0	0
South Dakota	1	1	0				44	6	37	1	0	0
Nebraska	1	5	0		2	1	489	19	37	0	0	0
Kansas	8	6	4			1	201	49	287	1	1	0
SOUTH ATLANTIC												
Delaware	1	1	0				24	1	20	0	0	0
Maryland ²	18	9	6	3	1	1	819	38	290	3	4	5
District of Columbia	0	0	0				219	6	92	1	1	1
Virginia	5	4	4	133	51	59	687	30	186	4	10	9
West Virginia	5	3	3		10	4	83	3	51	0	1	0
North Carolina	6	2	3			3	293	31	557	2	4	4
South Carolina	9	8	5	138	155	155	456	26	141	0	2	2
Georgia	5	1	2	5	6	8	172	13	142	0	1	1
Florida	6	2	1	3		1	202	14	168	2	3	3
EAST SOUTH CENTRAL												
Kentucky	6	3	2		1	1	111	42	74	6	1	1
Tennessee	1	2	2	5	12	10	148	69	69	3	3	3
Alabama	6	3	5	37	9	17	300	7	105	10	2	2
Mississippi ²	7	3	3							1	2	3
WEST SOUTH CENTRAL												
Arkansas	3	0	3	8	16	16	175	38	75	6	6	1
Louisiana	0	5	1	1	5	2	64	249	34	0	2	2
Oklahoma	3	2	3	19	26	19	158	26	98	0	0	0
Texas	35	30	17	261	558	398	1,194	457	641	5	9	9
MOUNTAIN												
Montana	0	0	0		4	2	93	16	74	0	0	0
Idaho	0	0	0	11			70	21	21	0	2	0
Wyoming	0	1	0				56	4	82	0	0	0
Colorado	7	9	7	8	17	20	730	26	203	1	0	1
New Mexico	4	0	1	2		1	65	9	23	0	0	0
Arizona	15	4	3	24	33	40	161	10	67	0	0	0
Utah ²	0	0	0		4	5	303	226	134	0	0	0
Nevada	0	0	0				1	5	5	0	0	0
PACIFIC												
Washington	3	6	4			1	278	285	285	2	1	1
Oregon	1	3	2	3	5	5	321	69	135	1	0	0
California	16	13	13	18	10	33	2,107	1,463	1,463	8	13	13
Total	277	189	189	719	1,009	1,009	29,444	5,335	19,116	121	182	182
21 weeks	7,206	5,726	5,590	185,224	62,533	75,305	516,099	69,505	422,983	3,500	4,704	4,704

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

*Telegraphic morbidity reports from State health officers for the week ended May 25, 1946, and comparison with corresponding week of 1944 and 5-year median—*Con.

Division and State	Poliomyelitis Week ended— May 25, 1946	May 26, 1945	Median 1941–45	Scarlet fever Week ended— May 25, 1946	May 26, 1945	Median 1941–45	Smallpox Week ended— May 25, 1946	May 26, 1945	Median 1941–45	Typhoid and paratyphoid fever [3] Week ended— May 25, 1946	May 26, 1945	Median 1941–45
NEW ENGLAND												
Maine	0	0	0	23	40	17	0	0	0	1	0	0
New Hampshire	0	0	0	24	26	7	0	0	0	0	0	0
Vermont	0	0	0	7	10	6	0	0	0	0	0	0
Massachusetts	0	0	0	174	317	286	0	0	0	2	1	2
Rhode Island	0	1	0	12	20	7	0	0	0	2	0	0
Connecticut	0	0	0	37	57	58	0	0	0	2	0	1
MIDDLE ATLANTIC												
New York	0	4	3	494	739	448	0	0	0	2	2	4
New Jersey	1	0	0	142	116	116	0	0	0	0	1	1
Pennsylvania	1	1	1	395	567	384	0	0	0	2	8	8
EAST NORTH CENTRAL												
Ohio	1	3	1	301	332	260	0	1	1	1	1	2
Indiana	0	3	0	52	90	39	5	0	0	1	2	2
Illinois	2	0	0	179	312	269	0	1	1	1	2	3
Michigan [2]	0	0	0	135	325	267	0	0	0	0	0	0
Wisconsin	0	0	0	98	244	244	0	0	1	0	0	0
WEST NORTH CENTRAL												
Minnesota	2	0	0	56	87	52	1	0	0	0	0	0
Iowa	1	0	0	63	34	34	0	0	0	2	0	1
Missouri	0	1	0	23	49	58	0	0	0	3	0	1
North Dakota	0	0	0	3	31	5	0	0	0	0	1	0
South Dakota	0	0	0	9	8	8	0	0	0	0	0	0
Nebraska	0	0	0	24	59	11	0	1	0	0	0	0
Kansas	1	0	1	45	59	51	1	0	0	1	1	1
SOUTH ATLANTIC												
Delaware	0	0	0	2	4	8	0	0	0	0	0	0
Maryland [2]	0	0	0	98	132	53	0	0	0	0	0	3
District of Columbia	0	0	0	12	30	14	0	0	0	0	0	0
Virginia	0	1	1	37	62	25	0	0	0	1	3	3
West Virginia	0	0	0	17	32	32	0	0	0	0	1	1
North Carolina	1	0	0	28	72	15	0	0	0	0	2	2
South Carolina	1	3	0	9	14	5	0	0	0	2	3	3
Georgia	1	1	0	1	23	13	0	0	0	4	2	6
Florida	22	0	1	1	6	3	0	0	0	2	3	3
EAST SOUTH CENTRAL												
Kentucky	0	0	0	33	42	42	0	0	0	0	1	2
Tennessee	0	0	0	25	29	26	0	0	0	3	5	4
Alabama	0	1	1	7	12	9	0	0	0	6	3	3
Mississippi [2]	0	0	0	9	7	2	0	0	1	3	2	2
WEST SOUTH CENTRAL												
Arkansas	1	1	0	1	8	4	0	0	1	3	3	3
Louisiana	5	0	1	0	12	4	0	0	0	3	1	4
Oklahoma	1	0	0	1	20	13	0	1	0	0	1	3
Texas	23	17	4	33	69	43	0	0	0	7	3	7
MOUNTAIN												
Montana	0	0	0	3	16	10	0	0	0	0	1	0
Idaho	0	0	0	7	18	18	0	0	0	1	1	0
Wyoming	0	0	0	2	3	6	0	0	0	0	0	0
Colorado	2	0	0	34	39	29	3	0	0	0	1	0
New Mexico	0	0	0	7	11	7	0	0	0	2	0	0
Arizona	0	0	0	8	48	15	0	0	0	1	1	0
Utah [2]	0	0	0	19	18	20	0	3	0	1	1	0
Nevada	0	0	0	0	1	0	0	0	0	0	0	0
PACIFIC												
Washington	2	3	0	18	51	43	0	0	0	1	1	0
Oregon	0	0	0	39	26	16	0	0	0	3	0	1
California	9	4	4	145	352	129	0	0	0	5	0	4
Total	77	44	28	2,892	4,679	3,218	10	7	19	65	58	100
21 weeks	890	740	499	72,816	112,829	82,498	214	211	471	1,094	1,242	1,603

[2] Period ended earlier than Saturday.
[3] Including paratyphoid fever reported separately, as follows: Massachusetts 1; Connecticut 1; Georgia 2; Florida 1; Louisiana 2; Texas 1; Arizona 1; Oregon 3; California 1.

Telegraphic morbidity reports from State health officers for the week ended May 25, 1946, and comparison with corresponding week of 1945 and 5-year median—Con.

Division and State	Whooping cough			Week ended May 25, 1946							
	Week ended—		Median 1941-45	Dysentery			Encephalitis, infectious	Rocky Mt. spotted fever	Tularemia	Typhus fever, endemic	Undulant fever
	May 25, 1946	May 26, 1945		Amebic	Bacillary	Unspecified					
NEW ENGLAND											
Maine	3	25	24								1
New Hampshire	3	4	4								
Vermont	14	24	13								1
Massachusetts	108	156	156								1
Rhode Island	25	20	20								1
Connecticut	53	77	66								1
MIDDLE ATLANTIC											
New York	172	210	254	7	6		2				8
New Jersey	112	117	172								1
Pennsylvania	100	221	221		2		1	1			1
EAST NORTH CENTRAL											
Ohio	57	132	132	1							1
Indiana	15	30	32								
Illinois	94	39	106	9			1		1		13
Michigan ³	108	44	279		1						1
Wisconsin	98	31	111						2		2
WEST NORTH CENTRAL											
Minnesota	5	14	20	5							3
Iowa	27	3	18								4
Missouri	18	16	16			2					
North Dakota	2	4	4								
South Dakota			5								1
Nebraska	5	4	4						1		1
Kansas	15	19	43				1				9
SOUTH ATLANTIC											
Delaware		1	1								
Maryland ³	17	55	55			1	1	1			
District of Columbia	8	14	14								
Virginia	80	54	89			50		1			1
West Virginia	18	10	21						1		
North Carolina	72	182	182	3	1					1	
South Carolina	64	60	89	1	30				1	2	
Georgia	13	12	35	1	7					12	3
Florida	21	7	24	1						4	
EAST SOUTH CENTRAL											
Kentucky	24	58	58		29						
Tennessee	61	27	65		2			1		1	1
Alabama	12	35	35	2			1			4	
Mississippi ³									1	1	1
WEST SOUTH CENTRAL											
Arkansas	18	2	32	1	2				11		3
Louisiana	9	7	5							2	
Oklahoma	24	10	13							2	2
Texas	170	250	263	23	377	48	2		2	25	11
MOUNTAIN											
Montana	6	2	4								
Idaho	21	2	3			1					
Wyoming		3	3								
Colorado	23	35	21				1	3			
New Mexico	18	3	10								2
Arizona	41	15	18			45	1				2
Utah ³	8	43	43								2
Nevada											
PACIFIC											
Washington	35	24	37								
Oregon	46	27	27			1					
California	71	412	378	4	3				2		7
Total	1,914	2,540	3,752	58	460	148	11	9	20	54	85
Same week, 1945	2,540			33	375	117	7	8	14	81	107
Average, 1943–45	2,781			29	392	96	9	[4] 16	15	[4] 48	
21 weeks: 1946	38,940			821	6,714	2,255	177	65	376	963	1,769
1945	52,392			628	8,879	2,404	140	50	331	1,063	1,867
Average, 1943–45	58,429		[4] 80,538	596	6,222	1,665	198	[4] 68	305	[4] 942	

³ Period ended earlier than Saturday.   [4] 5-year median, 1941–45.

Anthrax: New Jersey 1 case.   Leprosy: Florida 3 cases.

WEEKLY REPORTS FROM CITIES

City reports for week ended May 18, 1946

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

	Diphtheria cases	Encephalitis, infectious, cases	Influenza Cases	Influenza Deaths	Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
NEW ENGLAND												
Maine:												
Portland	0	0	-----	0	-----	0	2	0	2	0	0	9
New Hampshire:												
Concord	0	0	-----	0	-----	0	1	0	0	0	0	-----
Vermont:												
Barre	0	0	-----	0	-----	0	1	0	0	0	0	-----
Massachusetts:												
Boston	2	0	-----	0	337	0	9	0	59	0	0	8
Fall River	0	0	-----	0	64	0	0	0	5	0	0	1
Springfield	0	0	-----	0	96	0	1	0	8	0	0	1
Worcester	0	0	-----	0	405	0	11	0	9	0	0	34
Rhode Island:												
Providence	0	0	-----	0	57	0	1	0	4	0	0	21
Connecticut:												
Bridgeport	0	0	-----	0	2	0	0	0	1	0	0	-----
Hartford	0	0	-----	0	11	0	3	0	5	0	0	8
New Haven	0	0	-----	0	94	0	2	0	3	0	0	2
MIDDLE ATLANTIC												
New York:												
Buffalo	8	0	-----	0	91	0	4	0	17	0	0	8
New York	11	0	6	1	950	3	38	1	309	0	1	42
Rochester	0	0	-----	0	192	1	3	0	26	0	0	2
Syracuse	0	0	-----	0	11	0	1	0	14	0	0	-----
New Jersey:												
Camden	2	0	1	1	25	0	1	0	0	0	0	2
Newark	0	0	-----	1	359	0	2	0	12	0	0	30
Trenton	0	0	-----	0	80	0	1	0	4	0	0	3
Pennsylvania:												
Philadelphia	3	0	1	0	389	1	19	0	64	0	3	25
Pittsburgh	1	0	-----	0	23	3	7	0	28	0	0	1
Reading	2	0	-----	0	13	0	3	0	11	0	0	12
EAST NORTH CENTRAL												
Ohio:												
Cincinnati	0	0	-----	0	32	5	7	0	11	0	0	8
Cleveland	1	0	6	0	145	2	11	0	49	0	2	12
Columbus	2	0	-----	0	6	0	3	1	16	0	0	2
Indiana:												
Fort Wayne	0	0	-----	0	5	0	3	0	3	0	0	2
Indianapolis	0	0	-----	0	119	0	2	0	18	0	0	10
South Bend	0	0	-----	0	3	0	0	0	3	0	0	-----
Terre Haute	0	0	-----	0	13	0	2	0	0	0	0	-----
Illinois:												
Chicago	1	0	-----	1	218	5	25	0	87	0	1	48
Springfield	0	0	-----	0	11	0	2	0	1	0	0	-----
Michigan:												
Detroit	1	2	1	1	191	3	4	0	60	0	0	34
Flint	0	0	-----	0	9	1	3	0	4	0	0	1
Grand Rapids	0	0	-----	0	147	0	0	0	13	0	1	4
Wisconsin:												
Kenosha	0	0	-----	0	126	0	0	0	3	0	0	-----
Milwaukee	1	0	-----	0	978	0	9	0	24	0	0	42
Racine	0	0	-----	0	94	0	0	0	6	0	0	-----
Superior	0	0	-----	0	3	0	0	0	0	0	0	-----
WEST NORTH CENTRAL												
Minnesota:												
Duluth	0	0	-----	0	9	0	1	0	2	0	0	-----
Minneapolis	2	0	-----	0	21	1	2	1	12	0	0	2
St. Paul	0	0	-----	0	9	1	5	0	10	0	0	8
Missouri:												
Kansas City	1	0	-----	0	6	0	5	0	8	0	1	5
St. Joseph	0	0	-----	0	3	0	0	0	1	0	0	-----
St. Louis	0	0	2	0	119	4	9	0	13	0	1	6

City reports for week ended May 18, 1946—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza Cases	Influenza Deaths	Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
WEST NORTH CENTRAL— continued												
Nebraska:												
Omaha	3	0	------	0	42	1	0	0	5	0	0	1
Kansas:												
Topeka	0	0	------	0	3	0	2	0	8	0	0	3
Wichita	0	0	------	0	105	0	2	1	7	0	0	1
SOUTH ATLANTIC												
Delaware:												
Wilmington	1	0	------	0	12	0	1	0	1	0	1	------
Maryland:												
Baltimore	12	1	------	0	439	0	4	0	26	0	0	6
Cumberland	0	0	------	0	------	0	0	0	1	0	0	------
Frederick	0	0	------	0	------	0	0	0	0	0	0	------
District of Columbia:												
Washington	0	0	------	0	332	1	6	0	14	0	0	13
Virginia:												
Lynchburg	0	0	------	0	29	0	0	0	0	0	0	------
Richmond	0	0	------	0	95	0	1	0	5	0	0	4
Roanoke	0	0	------	0	8	0	0	0	3	0	0	------
West Virginia:												
Charleston	0	0	------	0	2	0	0	0	3	0	0	------
Wheeling	0	0	------	0	1	0	1	0	0	0	0	27
North Carolina:												
Raleigh	0	0	------	0	25	0	4	0	0	0	0	------
Wilmington	1	0	------	0	28	0	0	0	0	0	0	------
Winston-Salem	0	0	------	0	34	0	0	0	4	0	0	11
South Carolina:												
Charleston	0	0	2	0	6	0	1	0	1	0	0	------
Georgia:												
Atlanta	0	0	------	0	------	0	1	0	0	0	0	------
Brunswick	0	0	------	0	------	0	0	0	0	0	0	------
Savannah	0	0	------	0	6	0	1	0	0	0	0	------
Florida:												
Tampa	1	0	1	1	56	0	0	2	1	0	0	2
EAST SOUTH CENTRAL												
Tennessee:												
Memphis	0	0	------	1	43	0	6	0	2	0	1	10
Nashville	0	0	------	0	5	0	2	0	3	0	0	1
Alabama:												
Birmingham	1	0	------	0	28	0	1	0	2	0	0	1
Mobile	0	0	------	0	1	0	1	0	1	0	0	------
WEST SOUTH CENTRAL												
Arkansas:												
Little Rock	0	0	------	0	23	1	1	0	1	0	0	------
Louisiana:												
New Orleans	7	0	6	0	42	0	3	5	6	0	1	1
Shreveport	0	0	------	0	------	0	5	0	0	0	1	------
Texas:												
Dallas	1	0	------	0	59	0	1	0	1	0	0	1
Galveston	0	0	------	0	1	0	1	0	1	0	0	------
Houston	0	0	1	0	11	0	3	0	2	0	1	2
San Antonio	2	1	------	0	21	1	5	6	1	0	0	2
MOUNTAIN												
Montana:												
Billings	0	0	------	0	4	0	0	0	0	0	0	------
Great Falls	0	0	------	0	4	0	0	0	0	0	0	------
Helena	0	0	------	0	9	0	0	0	0	0	0	------
Missoula	0	0	------	0	18	0	0	0	4	0	0	------
Idaho:												
Boise	0	0	------	0	------	0	0	0	1	0	0	------
Colorado:												
Denver	0	0	------	0	478	0	9	4	17	0	0	14
Pueblo	0	0	------	0	44	0	0	0	1	0	0	4
Utah:												
Salt Lake City	0	0	------	0	90	0	1	0	5	0	0	5

City reports for week ended May 18, 1946—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
PACIFIC												
Washington:												
Seattle	1	0	------	0	51	0	1	0	10	1	0	8
Spokane	0	0	------	0	24	0	0	0	1	0	0	9
Tacoma	2	0	------	0	12	1	0	0	2	0	1	7
California:												
Los Angeles	0	0	6	1	285	1	1	1	32	0	1	9
Sacramento	0	0	------	0	108	0	0	0	0	0	0	3
San Francisco	1	0	2	1	111	0	5	0	17	0	0	------
Total	71	4	35	9	8,161	36	273	22	1,114	1	16	538
Corresponding week, 1945	56	------	29	14	1,593	------	296	------	1,525	0	14	575
Average, 1941–45	57	------	48	¹17	²5,605	------	¹376	------	1,402	1	16	970

¹ 3-year average, 1943–45.
² 5-year median, 1941–45.

Anthrax.—Cases: Camden 1.
Dysentery, amebic.—Cases: New York, 1; Indianapolis, 1; Chicago, 1; Memphis, 1; Los Angeles, 1.
Dysentery, bacillary.—Cases: New Haven, 1; New York, 6; Detroit, 1; Baltimore, 2; Charleston, S. C., 4.
Dysentery, unspecified.—Cases: Baltimore, 1; San Antonio, 29.
Rocky Mountain spotted fever.—Cases: Richmond, 1; Spokane, 1.
Tularemia.—Cases: Birmingham, 1; New Orleans, 1.
Typhus fever, endemic.—Cases: New York, 1; Winston-Salem, 1; Savannah, 1; New Orleans, 1; San Antonio, 1.

Rates (annual basis) per 100,000 population, by geographic groups, for the 89 cities in the preceding table (estimated population, 1943, 34,366,400)

	Diphtheria case rates	Encephalitis, infectious, case rates	Influenza		Measles case rates	Meningitis, meningococcus, rates	Pneumonia death rates	Poliomyelitis case rates	Scarlet fever case rates	Smallpox case rates	Typhoid and paratyphoid fever case rates	Whooping cough case rates
			Case rates	Death rates								
New England	5.2	0.0	0.0	0.0	2,786	0.0	81.0	0.0	251	0.0	0.0	220
Middle Atlantic	12.5	0.0	3.7	1.4	987	3.7	36.6	0.5	224	0.0	1.9	58
East North Central	3.6	1.2	4.3	1.2	1,277	9.7	43.2	0.6	181	0.0	2.4	99
West North Central	12.1	0.0	4.0	0.0	638	14.1	52.3	4.0	133	0.0	4.0	52
South Atlantic	24.5	1.6	4.9	1.6	1,754	1.6	32.7	3.3	96	0.0	1.6	103
East South Central	5.9	0.0	0.0	5.9	454	0.0	59.0	0.0	47	0.0	5.9	71
West South Central	28.7	2.9	20.1	0.0	451	5.7	54.5	31.6	34	0.0	5.7	17
Mountain	0.0	0.0	0.0	0.0	5,139	0.0	79.4	31.8	222	0.0	0.0	183
Pacific	6.3	0.0	12.7	3.2	935	3.2	11.1	1.6	98	1.6	3.2	57
Total	10.8	0.6	5.3	1.4	1,242	5.5	41.5	3.3	169	0.2	2.4	82

PLAGUE INFECTION IN TEXAS

Under date of May 20, 1946, Dr. N. E. Wayson, of the Office of Plague Suppressive Measures in San Francisco, Calif., reported that plague infection had been found in 8 different pools of fleas taken from ground squirrels, prairie dogs, grasshopper mice, and kangaroo rats in Cochran County, Tex. The specimens were collected during the period April 27–30, 1946.

This is the first report of sylvatic plague in the State, although repeated surveys had previously been made in this district. The specimens were collected over an area of approximately 300 square miles.

In 1920, human cases of plague occurred in Beaumont (14 cases, 6 deaths), Galveston (18 cases, 12 deaths), and Port Arthur (1 fatal case). Intensive plague-suppressive measures soon brought the infection under control, although infected rodents were found in Galveston until 1922. These were the only previously reported instances of plague infection in the State.

Dr. Wayson reports positive findings in pools of fleas as follows: 31 fleas from 26 prairie dogs (*Cynomys* sp.), 12 fleas from 8 ground squirrels (*C. tridecemlineatus*), and 15 fleas from 14 grasshopper mice (*Onychomys* sp.), collected April 27, 5 miles west of a point 5 miles south of Morton on State highway 214; 50 fleas from 31 prairie dogs (*Cynomys* sp.), collected April 27, 5 miles further west; 85 fleas from 30 prairie dogs (*Cynomys* sp.), collected April 30, 10 miles east of a point 10 miles south of Morton on State highway 214; 15 fleas from 17 grasshopper mice (*Onychomys* sp.), 25 fleas from 11 prairie dogs (*Cynomys* sp.), and 6 fleas from 31 kangaroo rats (*Dipodomys* sp.) collected April 30, 5 miles east of a point 20 miles south of Morton on State highway 215.

SMALLPOX IN THE UNITED STATES

No case of smallpox was reported in either California or Washington State during the week. Date of onset of last reported local case in San Francisco was March 27; onset of last case in Seattle was May 9.

During the current week a total of 10 cases of smallpox was reported in the United States, of which 5 occurred in Indiana, where 7 cases were reported during the preceding week (6 in one household; cases of local origin), and 3 in Colorado (2 in the preceding week).

In spite of the outbreak of the disease on the West Coast (61 cases in Washington State, 13 in California), introduced from the Orient, only three more cases have been reported in the United States to date this year than for the same period last year (214 to date this year, 211 for the same period last year).

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended April 27, 1946.— During the week ended April 27, 1946, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox		12		105	207	7	21	28	130	510
Diphtheria		7	1	15	3	3			2	31
German measles				35	33		3	3	5	79
Influenza		3			6				4	13
Measles		70	4	628	1,396	18	3	64	17	2,200
Meningitis, meningococcus		1		2	2				2	7
Mumps			1	40	308	71	22	64	196	702
Poliomyelitis					1					1
Scarlet fever		7	2	58	70	8		8	27	180
Tuberculosis (all forms)		1	4	105	60	12	16	29	53	280
Typhoid and paratyphoid fever				11	1		1		6	19
Undulant fever					3				1	4
Venereal diseases:										
Gonorrhea	3	14	12	94	146	65	75	29	75	513
Syphilis	2	16	6	120	76	12	7	5	21	265
Other forms									1	1
Whooping cough		1		47	36	1		15		100

NEW ZEALAND

*Notifiable diseases—4 weeks ended February 23, 1946.—*During the 4 weeks ended February 23, 1946, certain notifiable diseases were reported in New Zealand as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal meningitis	11	1	Lead poisoning	2	
Diphtheria	76	1	Malaria	7	
Dysentery:			Poliomyelitis	28	
Amebic	6		Puerperal fever	7	1
Bacillary	7		Scarlet fever	90	
Erysipelas	12		Tuberculosis (all forms)	178	57
Food poisoning	117		Typhoid fever	9	1
Influenza	1		Undulant fever	3	

SWEDEN

*Vital statistics—1945.—*The following are preliminary data for Sweden for 1945: Deaths, all ages, 71,194; infant deaths, 3,963; live births, 133,793. (Population, December 31, 1944—6,597,348; December 31, 1945—6,673,956.)

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

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

A table showing the accumulated figures for these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

Cholera

China—Cholera has been reported in China as follows: Hupeh Province, April 1–30, 1946, 111 cases with 25 deaths; Kwangtung Province, April 1–30, 1946, 66 cases with 8 deaths, including 24 cases with 2 deaths in Canton for the period April 21–30, 1946. For the period May 1–10, 1946, 7 cases of cholera were reported in Canton. The following cases in Kiangsi Province were reported to have been imported with Japanese repatriates from Hankow: During the period April 1–10, 1946, 32 cases in Shanghai, and during the period May 1–20, 1946, 47 cases with 3 deaths, in Nanking.

Plague

China—Fukien and Kiangsi Provinces.—For the period April 1–30, 1946, a total of 336 cases of plague, with 131 deaths, was reported in Fukien Province, including 287 cases and 103 deaths in Foochow. During the period February 21–April 20, 1946, 66 cases with 35 deaths were reported in Kiangsi Province.

Smallpox

China—Shanghai.—For the period April 21–30, 1946, 27 cases of smallpox, with 5 deaths, were reported in Shanghai, for the period May 1–10, 52 cases, with 8 deaths, and for the period May 11–20, 32 cases, with 10 deaths.

India—Bombay.—During the week ended April 27, 1946, 125 cases of smallpox, with 25 deaths were reported in the city of Bombay, and 520 cases, with 124 deaths, were reported in the area outside the city of Bombay, within a radius of 400 miles of the city.

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