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THE ELIMINATION OF CARBON MONOXIDE FROM BLOOD, BY TREATMENT WITH AIR, WITH OXYGEN, AND WITH A MIXTURE OF CARBON DIOXIDE AND OXYGEN.[1]

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INTRODUCTION.

The most important factor in the treatment of cases of carbon monoxide poisoning is the early elimination of the CO from the blood. This fact has been recognized by many investigators, and, as a result, the use of oxygen (O_2) was recommended several years ago. Apparently the importance of the early elimination of CO has not been sufficiently emphasized, as many cases do not receive the oxygen treatment, or its administration is delayed several hours after the removal of the victim from the impure air.

Recently, the addition of carbon dioxide (CO_2) to oxygen[2] has been recommended as being more efficacious than oxygen alone. In order to determine the relative values of the two treatments—that is, O_2 and a mixture of CO_2 in O_2, as shown by the speed of elimination of CO from the blood of man, the general effect on the victim, and the resulting condition—the following treatments were studied:

1. Subjects were allowed to breathe normal air.
2. Subjects were allowed to breathe pure oxygen (98.5 per cent O_2, 1.5 per cent N_2).
3. Subjects were allowed to breathe 8 to 10 per cent of carbon dioxide in pure oxygen.

These treatments were given after the subjects had been exposed to from 0.12 to 0.15 per cent of CO in air for a period of approximately one hour, which caused a blood saturation of 30 to 40 per cent, and symptoms ranging from severe basal headaches and dizziness to unconsciousness.

The writers are fully aware that the experiments are not sufficiently extensive to furnish all the data necessary for establishing a formula which would take care of all the factors affecting the elimination of CO from the blood, and likewise all those affecting recovery from CO poisoning; nevertheless, they have accomplished their purpose

[1] Investigations carried on in cooperation with the Bureau of Mines, Department of the Interior.

[2] The elimination of carbon monoxide from the blood after a dangerous degree of asphyxiation, and a therapy for accelerating the elimination. By Yandell Henderson and H. W. Haggard. Jour. Pharm. Exper. Therap., 1920, vol. 16, 1-11.

in establishing comparative measurements of the efficacy and merits of the three methods of treatment; and from the information obtained a suitable procedure may be selected.

CHEMICAL ACTION.

Chemically, the recognized primary action of carbon monoxide poisoning is the union of CO with the hemoglobin (Hb) of the blood, thus depriving the latter of its function of carrying oxygen from the lungs to the tissues. This union, from the standpoint of gas volumes, is similar to the normal one with oxygen, or, in either case, molecule for molecule—that is, the gas-volume capacity of the hemoglobin is the same whether it is in combination with O_2, CO, or a mixture of O_2 and CO. However, the union of Hb with CO is a great deal more stable than the union with O_2, which readily comes off at reduced pressures. Also the relative percentages or tensions of the two gases which will exert an equal combining force are greatly different, that of CO being but approximately 1/300 that of oxygen, or in a ratio of approximately 300:1, which is termed the relative affinity of hemoglobin for CO and O_2. Thus, the combining force of either of the two gases, when both are present, is the product of the affinity times the tension ($A \times T$), and the amount of hemoglobin that will combine with either gas (providing the tensions are high enough to saturate all of the hemoglobin completely) in a certain CO-air mixture will be in the ratio of these forces, or

$$\frac{COHb}{O_2Hb} = \frac{Aco \times Tco}{Ao_2 \times To_2} \qquad (1)$$

and since the percentage of $COHb = \frac{\text{Amount of } COHb \times 100}{\text{Total Hb or } (COHb + O_2Hb)}$, by substituting from equation (1), according to the rules of proportion,

$$\text{Percentage of } COHb = \frac{Aco \times Tco}{(Aco \times Tco) + (Ao_2 \times To_2)} \times 100 \qquad (2)$$

Thus, from equation (1) it can be readily understood why comparatively small quantities of CO in air may form a sufficient amount of COHb to cause oxygen deprivation, resulting in a deleterious effect. For example, 0.05 per cent (or 5 parts in 10,000, this being the usual way of expressing gas tensions), in pulmonary air which contains approximately 1,500 parts of oxygen would result in about 50 per cent of the hemoglobin combining with carbon monoxide.

$$\begin{aligned}\text{Percentage of } COHb &= \frac{Aco \times Tco}{(Aco \times Tco) + (Ao_2 \times To_2)} \times 100 \\ &= \frac{300 \times 5}{(300 \times 5) + (1{,}500 \times 1)} \times 100 \\ &= 50 \text{ per cent.}\end{aligned}$$

It is improbable in CO poisoning that there will be a complete equilibrium established, because, as the equilibrium point is approached, the tendency of the COHb to dissociate into CO and Hb also increases, thus slowing down the reaction, which at first proceeded rapidly. However, it has been found by Henderson and coworkers [3] that with low tension of CO, and with the subject at rest, about half the equilibrium is attained in the first hour and about three-fourths in the first two hours. Should the victims or subjects be exercising, a greater respiratory exchange would take place, and likewise an increase in the amount COHb formed in a given time. With higher amounts of CO in the air, the reaction is faster, and the time required to reach half the equilibrium point is greatly lessened, perhaps to about 15 minutes for concentrations of 50 parts of CO per 10,000 of air.

Thus the chemical reaction of CO poisoning is essentially that of reversible mass action, and can be made to proceed in either direction by changing the mass or tensions of the gases in the pulmonary air or blood; and it is on this basis that methods of treatment have been devised.

PHYSIOLOGICAL ACTION.

The principal physiological effects seem to be those of insufficient oxygen supply, or asphyxia.[4] In an exposure to carbon monoxide, the victim's blood is being continually and increasingly incapacitated for carrying oxygen from the lungs to the tissues, the degree of incapacitation depending upon the concentration of the gas and the duration of the period of inhalation, and the severeness of effects depending upon the extent of incapacitation and its duration. A more severe case of poisoning results from a long exposure to a low concentration than from a short exposure to a high concentration, even though the percentage of hemoglobin combining with the carbon monoxide is the same in each case. As the effects are almost in direct proportion to the length of time and the extent of oxygen deprivation, whether the time elapses during exposure or after exposure, it is plainly suggested, and has recently been emphasized by a number of investigators,[5] that the first and most important action in a method of treatment should be a speedy removal of the gas from the blood, so that it can resume its normal oxygen-carrying capacity and thus decrease the danger or extent of tissue degeneration and permanent damage.

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[3] Physiological effects of automobile exhaust gas and standards of ventilation for brief exposures. By Y. W. Henderson, H. W. Haggard, M. C. Teague, A. L. Prince, and Ruth M. Wunderlich. Jour. Ind. Hygiene, vol. 3, July, 1921, pp. 79-92.

[4] Studies of carbon monoxide asphyxia. By Howard W. Haggard. Amer. Jour. Phys., vol. 60, April 1922, pp. 244-249; vol. 56, July, 1921, pp. 390-403.

[5] The elimination of carbon monoxide from the blood after a dangerous degree of asphyxiation, and a therapy for accelerating the elimination. By Yandell Henderson and H. W. Haggard. Journ. Pharm. and Exp. Therap., vol. 16, 1920, p. 11.

PRINCIPLE OF TREATMENT FOR REMOVING CO FROM THE BLOOD.

The reversibility of the chemical reaction between CO and the blood is a fortunate one. When a victim of poisoning has been removed to fresh air, he immediately begins (in cases where respiration has not stopped, otherwise artificial respiration must be used) to eliminate that part of the CO which, through dissociation of the carbon monoxide-hemoglobin, is liberated in the lungs, and the portion of incapacitated hemoglobin begins to decrease. But this natural elimination, since it depends upon the mass of O_2 in normal air, is rather slow and does not attain the speed which is often necessary to save life or to reduce the after effects as much as possible; and it has become a practice to give inhalations of oxygen in an almost pure form, thereby increasing its tension both in the pulmonary air and in solution in the plasma, consequently hastening the elimination. This is further aided by increasing the respiratory exchange and, consequently, the removal from the lungs of the CO liberated through dissociation, so that its reunion with Hb is inhibited.

EXPERIMENTAL WORK.

Careful observations were made on the respiration, heart rate objective and subjective symptoms, and the percentage saturation of the blood. The experiments were planned to secure data as free as possible from individual differences. The same three men were used for three parallel experiments, and each individual took one of the three treatments, so that on completion of the series data were afforded relative to individual differences from which average results could be obtained.

The 1,000 cubic foot gas chamber [6] at the Pittsburgh Experiment Station of the Bureau of Mines was used for the experiments. The carbon monoxide for making the CO-air mixtures was generated by dropping formic acid into heated sulphuric acid, purified through a soda-lime canister, and then metered into the chamber. Samples of the resultant mixtures were taken in duplicate or triplicate at the beginning and end of each experiment. Analyses were made for CO by the liquid-air iodine-pentoxide method.[7] Blood samples were taken at various intervals and analyzed by two methods, namely, the spectrophotometric [8] and the tannic acid.[9]

[6] Described in detail in "Physiological effects of exposure to low concentrations of carbon monoxide." By R. R. Sayers, F. V. Meriwether, and W. P. Yant. Public Health Reports, vol. 37, No. 19, May 22, 1922, p. 1127. Reprint No. 748.

[7] The determination of carbon monoxide in air contaminated with motor exhaust gas. By M. C. Teague. Jour. Ind. and Eng. Chem., vol. 12, Oct. 1920, pp. 964-968.

[8] Work cited in footnote 6.

[9] The tannic acid method for quantitative determination of carbon monoxide in blood. By R. R. Sayers and W. P. Yant. Bureau of Mines Report of Investigations, Serial No. 2356, May, 1922. Public Health Reports, vol. 37, No. 40, Oct. 6, 1922, pp. 2433-2439. Reprint No. 790.

As further data, analyses of the carbon-monoxide alveolar air were made, the samples being obtained by rebreathing 8 times into a 1,500-c.c. rubberized bag. The gas thus obtained should be in equilibrium with the blood, and a determination of the CO content should show any change taking place in the amount of carbon monoxide-hemoglobin. Analyses were made for CO by the liquid-air iodine-pentoxide method previously referred to.

The apparatus used for supplying the O_2 and CO_2–O_2 mixtures to the subject is shown in Figure 1. It is the regulation inhaler used for such work, and consists of a storage tank containing the oxygen at high pressure, a pressure-reducing valve, a gauge to indicate the quantity in the tank, a rubberized supply reservoir, a Tissot type Army gas mask facepiece, and a connecting hose so fitted with mica valves that the gas to be breathed is drawn into the lungs from the air reservoir bag and then exhaled to the exterior, thus giving a continuous and fresh supply. While in use, the valves are adjusted to let the gas escape through the reducing valve into the reservoir at a rate approximately equal to the breathing of the subject. In all experiments, analyses were made on the contents of the storage tanks, and the volume used was calculated from the indicated pressures. Tables I, II, and III include these data.

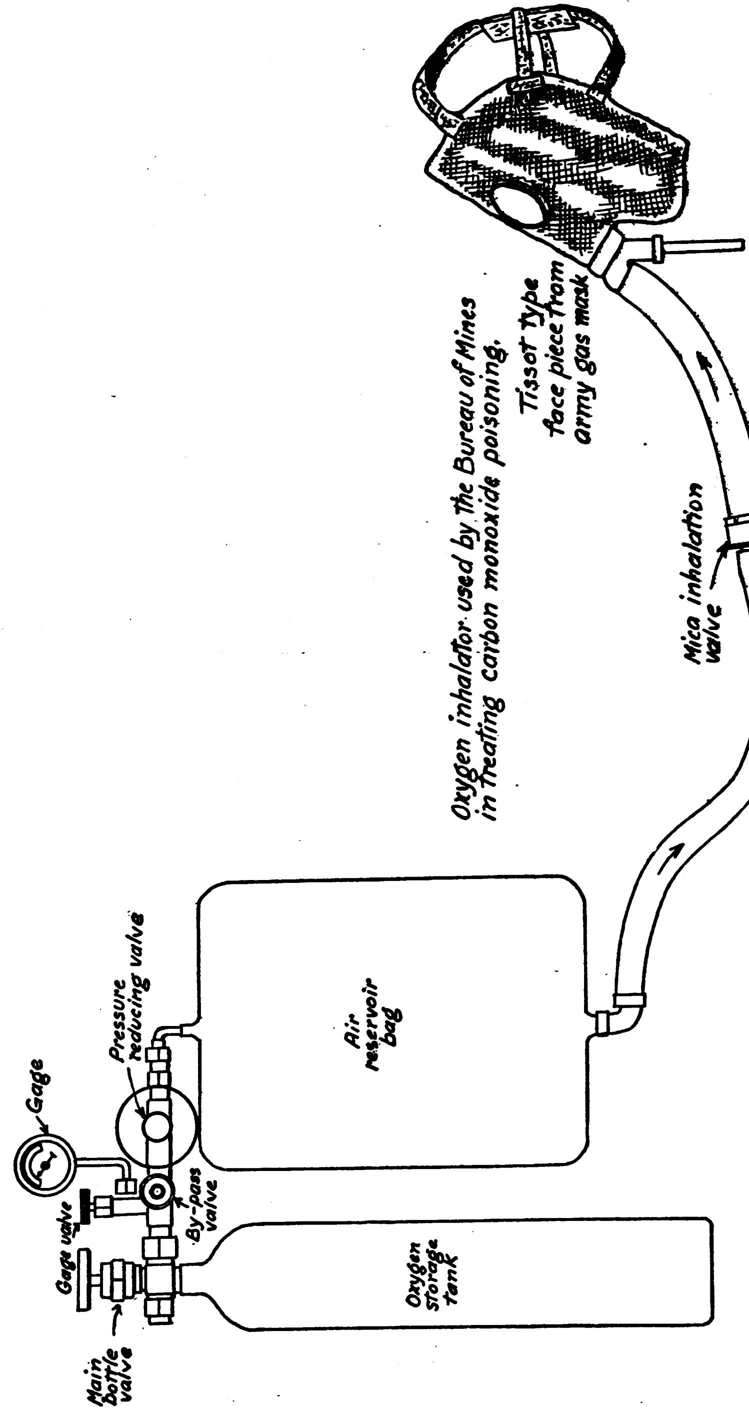
Oxygen inhalator used by the Bureau of Mines
in treating carbon monoxide poisoning.
Tissot type
face piece from
army gas mask
Mica inhalation
valve
Air
reservoir
bag
Oxygen
storage
tank
Gage
Pressure
reducing valve
By-pass
valve
Gage valve
Main
bottle
valve

Fig. 1.

TABLE I.—*Rate of elimination of carbon monoxide as obtained by different methods of treatment.*

[Test No. 1, Feb. 16, 1922. Began 9.45 a. m. Conditions of test: Concentration of CO, parts per 10,000—start, 11.6; end, 10.5; average, 11.0; duration of exposure, 65 minutes; duration of treatment, 45 minutes.]

Carbon monoxide saturation of blood, per cent.			Carbon monoxide in alveolar air.		Respiration.		Pulse.		Personal data.		Remarks.
Time (min.).	Spectrophotometer.	Tannic acid.	Time (min.).	Parts per 10,000.	Time (min.).	Rate per minute.	Time (min.).	Rate per minute.	Time (min.).	Symptoms.	
SUBJECT, SAYERS. TREATMENT, NORMAL AIR.											
0	...	0.0	...	...	...	...	0	88	0	Very slight headache at time of entrance..	
					5	20	5	84	5	Headache disappeared on sitting down....	
28	14.0	12.0	35	1.2	...	...	30	78	30	No symptoms. Headache appears on moving but disappears when sitting.	
					40	21	...	...	40	Headache when sitting...	
52	...	26.5	60	1.7	...	...	55	96	55	Headache more persistent...	
OUT OF CHAMBER AT END OF 65 MINUTES.											
70	34.0	35.0	...	...	...	...	...	...	75	Dizzy and full frontal headache...	
90	28.0	30.0	...	...	...	...	100	88	100	Headache more severe...	
105	24.0	25.0	105	1.2	115	20	115	88	115	Headache frontal and through temples....	
165	18.0	20.0	165	1.1	...	...	...	...	...	...	Headache persisted the remainder of the day.
225	19.0	15.0	225	.7	...	...	...	...	...	...	
283	14.0	11.0	283	.7	...	...	...	...	...	...	
375	11.0	10.0	375	.4	...	...	...	...	...	...	

TABLE I.—*Rate of elimination of carbon monoxide as obtained by different methods of treatment*—Continued.

Carbon monoxide saturation of blood, per cent.			Carbon monoxide in alveolar air.		Respiration.		Pulse.		Personal data.		Remarks.
Time (min.).	Spectrophotometer.	Tannic acid.	Time (min.).	Parts per 10,000.	Time (min.).	Rate per minute.	Time (min.).	Rate per minute.	Time (min.).	Symptoms.	
SUBJECT, McCONNELL, TREATMENTS CO_2-O_2 MIXTURE. VOLUME USED, 596 LITERS.[1]											
0	....	0.0	....	....	0	16	0	90	0	Good condition at time of entrance	
					5	14	5	88			
27	....	12.0	38	1.3	30	14	30	85	30	No symptoms	A urine test for albumin made 165 minutes after test was negative.
50	23.0	28.0	60	3.4	55	16	55	86	60	Slight tightness across forehead	
OUT OF CHAMBER AT END OF 65 MINUTES AND STARTED INHALATION OF CO_2-O_2 MIXTURE.											
68	33.0	32.0	....	....	80	24	....	....	....	....	The respiration during the treatment period was very little, if any, deeper than normal.
89	18.0	20.0	....	....	95	23	....	....	....	....	
111	14.0	11.0	108	0.6	115	24	115	87	115	Frontal headache	
STOPPED INHALATION AT END OF 110 MINUTES.											
165	13.0	6.0	165	0.5	....	....	....	....	....	....	
225	7.0	4.0	225	.4	....	....	....	....	....	....	
285	8.0	2.0	285	.4	....	....	....	....	....	....	

SUBJECT, MASSEY. TREATMENT, OXYGEN. VOLUME USED, 490 LITERS.[2]

0	....	0.0	....	....	....	....	....	....	0	Had bad headache night before: 6 grains acetanalid taken, but in good condition before test.
33	20.0	20.0	30	1.1	....	....	....	....	40	Slight fullness in head........
57	28.0	30.0	60	2.7	....	....	....	....	60	No symptoms; feels O. K........
OUT OF CHAMBER AT END OF 65 MINUTES AND STARTED INHALATION OF OXYGEN.										
68	32.0	33.0	....	....	....	....	....	....	....	
89	19.0	22.0	....	....	....	....	....	....	....	
110	15.0	17.0	105	0.5	15	18	115	79	115	No symptoms........
STOPPED INHALATION AT END OF 110 MINUTES.										
165	....	12.0	165	0.5	15	....	....	....	....	....
225	11.0	7.0	225	.5	15	....	....	....	....	....
285	8.0	5.0	285	.5	15	....	....	....	....	....

[1] Analysis of CO_2-O_2 mixture:

	Cylinder 1.	Cylinder 2.
	Per cent.	*Per cent.*
CO_2........	9.4	10.8
O_2........	88.9	87.5
N_2........	1.7	1.7

[2] Analysis of oxygen used: O_2, 98.5 per cent; N_2, 1.5 per cent.

TABLE II.—*Rate of elimination of carbon monoxide as obtained by different methods of treatment.*

Test No. 2, Mar. 1, 1922. Began 10 a. m. Conditions of test: Concentration of CO, parts per 10,000—start, 13.8; end, 13; average, 13.4: duration of exposure, 72 minutes; duration of treatment, 45 minutes.]

SUBJECT, SAYERS. TREATMENT, CO_2-O_2 MIXTURE. VOLUME USED, 1,238 LITERS.[1]

Carbon monoxide saturation of blood, per cent.			Carbon monoxide in alveolar air.		Respiration.		Pulse.		Personal data.		Remarks.
Time (min.).	Spectrophotometer.	Tannic acid.	Time (min.).	Parts per 10,000.	Time (min.).	Rate per minute.	Time (min.).	Rate per minute.	Time. (min).	Symptoms.	
0		0.0			0	18	0	44	0	Slight cold; otherwise O. K. on entrance	
30	21.0	18.0	40	1.2	20	18			45	No symptoms	
					50	18	45	72	50	Definite tightness across forehead, increasing on walking.	
									55	Slight feeling of dizziness. Headache beginning.	
60	35.0	36.0	68	3.6	76	20			60	Dizziness increased	
									70	Dizziness and headache	
OUT OF CHAMBER AT END OF 72 MINUTES. BEGAN INHALATION.											
88	20.0	20.0			85	[2] 37	86	88			
118	8.0	10.0	120	0.8	105	[2] 34					
STOPPED INHALATION AT END OF 117 MINUTES.											
195	6.0	9.0	195	0.5	195	19	195	85			
255	5.0	7.0	255	.6	255	18	255	78			
300	6.0	6.0	300	.6	300	19	300	82	300	Headache lingers; frontal in type	
360	3.0	5.0	3C0	.7							
420		4.0	420	.6	420	18	420	72			

SUBJECT, McCONNELL. TREATMENT, NORMAL AIR. (SEE REMARKS.)

0	...	0.0	...	...	0	14	0	84	0	Feels O. K. on entrance	It was planned to have McConnell take oxygen and Massey take normal air, but the latter subject collapsed after 2 minutes, and the oxygen was transferred to him, leaving McConnell to take air.
30	23.0	20.0	35	...	...	...	...	...	35	Slight tightness across forehead and slight dizziness.	
...	...	...	...	...	45	18	45	90	60	Above symptoms increasing	
70	...	36.0	65	...	...	...	72	108	72	Dizzy headache	

OUT OF CHAMBER AT END OF 72 MINUTES.

85	29.0	33.0	...	...	...	...	...	...	...	...	
113	...	28.0	120	2.1	105	14	90	94	...	...	
195	18.0	22.0	195	1.4	195	16	195	108	...	...	
255	16.0	18.0	255	.8	255	18	255	96	...	...	
									250	Headache persists	
300	10.0	13.0	300	.7	300	16	300	90	...	...	
360	6.0	11.0	360	.6	360	16	360	84	...	...	
420	5.0	10.0	420	.6	420	16	420	84	...	...	

SUBJECT, MASSEY. TREATMENT, OXYGEN. VOLUME USED, 610 LITERS.[3]

0	...	0.0	...	...	0	18	0	120	0	Slight sore throat, little "under par"	Although Massey did not feel severe symptoms they were evident to an outside observer. He seemed dazed and dizzy, but walked out unassisted and remarked that he was all right except a little dizzy. The collapse which followed a few minutes later was quick and complete.
30	25.0	20.0	40	2.4	...	...	...	...	45	No symptoms	
							50	100	50	Dizzy on standing	
									60	No symptoms while sitting	
65	34.0	38.0	67	3.3	...	...	...	...	70	Dizzy	

[1] Analysis of CO_2–O_2 mixture:

	Cylinder 1.	Cylinder 2.	Cylinder 3.
	Per cent.	*Per cent.*	*Per cent.*
CO_2	8.9	9.1	8.0
O_2	88.8	88.9	90.6
N_2	2.3	2.0	1.4

[2] Breathing rapid and deep.

[3] Analysis of oxygen used: O_2, 98.5 per cent; N_2, 1.5 per cent.

TABLE II.—*Rate of elimination of carbon monoxide as obtained by different methods of treatment*—Continued.

SUBJECT, MASSEY. TREATMENT, OXYGEN. VOLUME USED, 610 LITERS—Continued.

OUT OF CHAMBER AT END OF 72 MINUTES. BEGAN INHALATION OF OXYGEN AT 75 MINUTES.

Carbon monoxide saturation of blood, per cent.			Carbon monoxide in alveolar air.		Respiration.		Pulse.		Personal data.		Remarks.
Time (min.).	Spectrophotometer.	Tannic acid.	Time (min.).	Parts per 10,000.	Time (min.).	Rate per minute.	Time (min.).	Rate per minute.	Time (min.).	Symptoms.	
					83	21	....	....	74	Collapses while sitting on window sill......	
90	22.0	25.0	....	....	95	19	....	....	....	Placed on floor and given oxygen to breathe. Unconscious for 2 or 3 minutes.	
117	....	16.0	120	1.0	109	18	102	71	....	....	
STOPPED INHALATION AT END OF 117 MINUTES.											
195	10.0	12.0	195	1.2	195	....	195	88	....	....	
255	8.0	10.0	255	1.0	255	18	255	86	250	Feeling O. K. except a little tired. In a much better condition than either Sayers or McConnell.	
300	6.0	9.0	300	1.1	300	16	300	100	....	....	
360	4.0	8.0	420	.7	360	16	360	80	....	....	
420	....	7.0	....	.5	420	18	420	80	....	....	

Table III.—*Rate of elimination of carbon monoxide as obtained by different methods of treatment.*

[Test No. 3, Mar. 3, 1922. Began 12.45 p. m. Condition of test: Concentration of CO, parts per 10,000; start, 15; end, 14; average, 14.5; duration of exposure, 69 minutes; duration of treatment, 45 minutes.]

SUBJECT, SAYERS. TREATMENT, OXYGEN FROM GIBBS BREATHING APPARATUS. VOLUME, 682 LITERS, INCLUDING BAD LEAK.[1]

Carbon monoxide saturation of blood, per cent.			Carbon monoxide in alveolar air.		Respiration.		Pulse.		Personal data.	
Time (min.).	Spectrophotometer.	Tannic acid.	Time (min.).	Parts per 10,000.	Time (min.).	Rate per minute.	Time (min.).	Rate per minute.	Time (min.)	Symptoms.
0	....	0	....	....	8	16	0	72	0	Slight cold at time of entrance.
38	18	20	....	....	30	18	30	74	30	No symptoms except a little tightness across forehead.
		....	45	2.3	....	....	....	....	45	Slight headache on walking. Yawned several times.
							57	75	57	Slight dull headache. Dizzy on standing.
63	34	35	68	2.6	....	....	....	....	....	
Out of Chamber at End of 69 Minutes. Began Inhalation of Oxygen.										
93	18	20	100	1.5	77 82	14 18	77	82	....	
Stopped Inhalation at End of 99 Minutes.										
120	15	15	....	....	....	....	....	....	....	
180	13	11	180	1.1	180	16	180	68	180	Single, very fleeting sharp pain in head. No continuous headache, palpitation or dizziness.
240	9	8	240	.8	240	17	240	72	....	
300	....	6	300	.6	300	17	300	72	300	Feels fine.
360	8	5	360	.8	360	17	360	70	....	
420	8	4	420	.8	420	17	420	76	....	
480	7	3	480	.5	480	....	480	....	....	

SUBJECT, McCONNELL. TREATMENT, OXYGEN; VOLUME USED, 478 LITERS.[2]

0	....	0	....	....	0	16	0	90	0	Good condition at time of entrance.
30	22	20	....	....	30	18	30	84	....	
									30	No symptoms.
			45	2.8	....	....	....	....	42	Tightness, slight dizziness on standing.
60	35	35	68	3.5	50	16	50	90	50	Slight headache, sleepy, yawning a great deal.
									60	Definite headache.
Out of Chamber at End of 69 Minutes. Began Inhalation of Oxygen.										
90	22	25	....	....	75 97	16 19	75	88	....	
115	18	17	115	....	103	18	104	86	....	

[1] Analysis of oxygen used: O_2, 98.5 per cent; N_2, 1.5 per cent.
[2] Analysis of oxygen used: O_2, 98.5 per cent; N_2, 1.5 per cent.

Table III.—*Rate of elimination of carbon monoxide as obtained by different methods of treatment*—Continued.

SUBJECT, McCONNELL. TREATMENT, OXYGEN; VOLUME USED, 478 LITERS—Contd.

STOPPED INHALATION AT END OF 114 MINUTES.

Carbon monoxide saturation of blood, per cent.			Carbon monoxide in alveolar air.		Respiration.		Pulse.		Personal data.	
Time (min.).	Spectrophotometer.	Tannic acid.	Time (min.).	Parts per 10,000.	Time (min.).	Rate per minute.	Time (min.).	Rate per minute.	Time (min.).	Symptoms.
180	16	11	180	1.1	180	18	180	84	180	Headache remains but not as severe as at same time in test No. 2.
240	11	9	240	....	240	16	240	84	....	
300	9	7	300	1.2	300	16	300	90	....	
360	....	6	360	.9	360	16	360	84	....	
420	5	5	420	.8	420	18	420	91	....	
480	....	3	480	.5	480	16	480	84	....	Feels o. k.
1,200	....	0	....	....	....	....	....	....	....	

SUBJECT, MASSEY. TREATMENT, CO_2–O_2 MIXTURE. VOLUME USED, 772 LITERS.[3]

0	....	0	....	....	....	18	0	80	0	In good condition at time of entrance.
43	22	20	45	1.8	30	16	30	90	....	
									50	Slight unnatural feeling in head.
65	34	32	69	3.4	60	....	60	94	57	Tight feeling in head.

OUT OF CHAMBER AT END OF 69 MINUTES.

87	21	24	....	....	72	[4] 22	....	....	....	
113	10	10	115	1.2	80	[4] 26	....	....	....	
					95	[4] 28	....	....	....	
					100	[4] 27	....	....	....	
					107	[4] 30	....	....	....	

STOPPED INHALATION AT END OF 114 MINUTES.

180	8	8	180	1.0	180	16	180	88	180	Distinct headache.
240	....	7	240	1.1	240	18	240	82	....	
300	8	5	300	.8	300	18	300	96	....	
360	7	5	360	.7	360	20	360	84	....	
420	5	4	420	.6	420	18	420	90	....	
480	7	3	480	.5	480	16	480	84	....	
1,200	....	0	....	....	....	....	....	....	....	

[3] Analysis of CO_2–O_2 mixture:

	Cylinder 1.	Cylinder 2.
	Per cent.	*Per cent.*
CO_2	8.2	8.3
O_2	90.8	89.1
N_2	1.0	2.6

[4] Respiration much deeper than normal.

INTERPRETATION AND DISCUSSION OF RESULTS.

In a consideration of the efficacy of each of the three methods of treatment used, the average of the percentage saturation found in the blood was plotted against the time in minutes, which resulted in

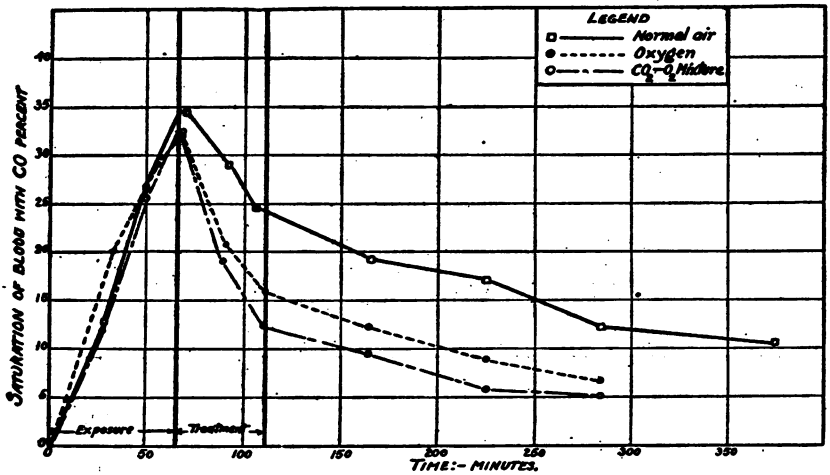

FIG. 2.—Curves showing comparative elimination of carbon monoxide from the blood by inhalations of air, oxygen, and a mixture of CO_2 in oxygen.

the curves represented by Figures 2, 3, and 4. Owing to the rather high concentration, and the like conditions of exposure for all three subjects, there was little difference to be found in the saturation; but

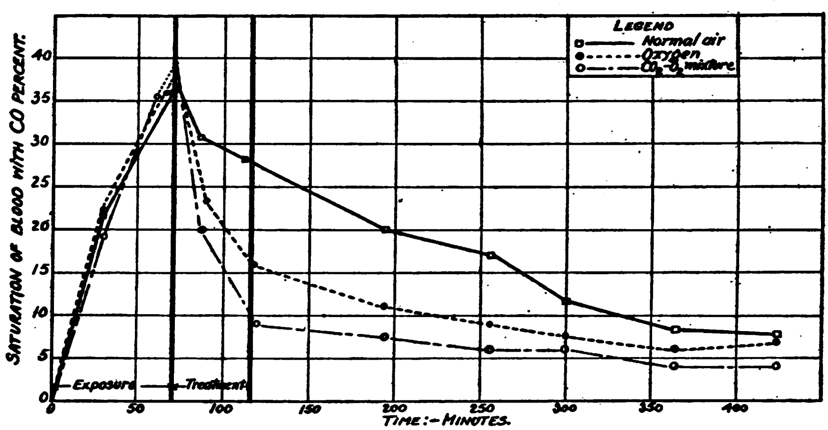

FIG. 3.—Curves showing comparative elimination of carbon monoxide from the blood by inhalations of air, oxygen, and a mixture of CO_2 in oxygen.

the data were recorded for comparison with the development of symptoms. As regards elimination, however, a very decided difference is indicated.

Although we did not attain in each case exactly the same degree of saturation of the blood with CO, a general comparison of the portions of the curves of each test which fall in the area of treatment shows that there is a difference in the rate of elimination of CO for each of the methods used, and that the speed of the exchange increases in the following order: Air, oxygen, CO_2–O_2 mixture.

The difference between the air and oxygen results is explainable on the basis of the relative percentage of oxygen in the inhaled gas, that is, 20.9 as against 98.5. The lung ventilation remained practically normal in the use of both air and oxygen; and the effect is primarily one of increasing the tension of O_2 in the lungs and in solution with plasma. This same factor explains the increased efficiency of the CO_2–O_2 mixture over the elimination with air, but it does not account for the further increase of the CO_2–O_2 over that of the O_2. By a comparison of the respiration rates and volumes breathed, as given in Tables I, II, and III, it will be noted that there is an increase in each case in which the CO_2–O_2 mixture was used;

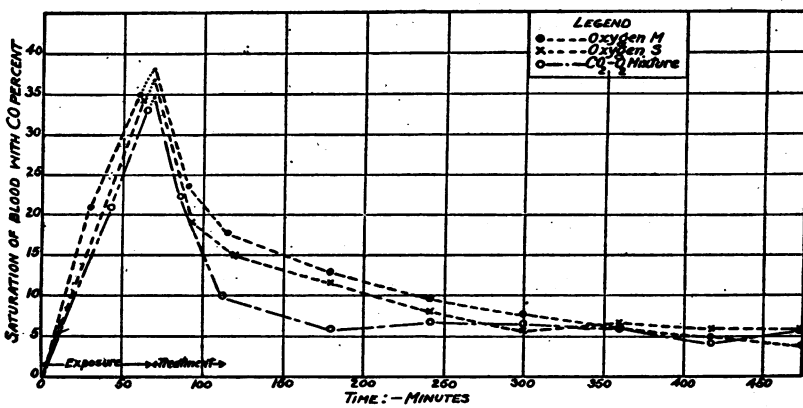

FIG. 4.—Curves showing comparative elimination of carbon monoxide from the blood by inhalations of oxygen and a mixture of CO_2 in oxygen.

and, further, by comparing these increases it is interesting to find that the elimination was fastest when these increases were the greatest. Although the tension of O_2 in the inhaled air was only approximately 90 per cent of that when O_2 alone was used, a more rapid elimination was secured by the increased lung ventilation, which compensated for the decrease in the O_2 by a more frequent change of the lung gases and the physical effects accompanying it.

A study of all the curves shown on Figures 2, 3, and 4 reveals a regularity in the elimination which would be expected from the type of the reaction. After the curves had been plotted, it was seen that they were of the exponential type, in which the log of any saturation percentage of Hb combining with CO (S′) after a time had elapsed, was equal to the log of the initial saturation (S) plus a constant (b log e) times the time (t) ($\text{Log } S' = \text{Log } S + (b \log e) \cdot t$). This constant (which is negative) was determined by the method of averages for comparative curves and portion of curves with the following results:

TABLE IV.—*Constants determined by method of averages for different treatment portions of elimination curves, Figures 2, 3, and 4.*

Treatment.	Test No.	From average of determined blood saturations, b=	From blood saturations calculated from alveolar air, b=
Air treatment	1	−0.00160	−0.00193
	2	−.00196	−.00195
Oxygen treatment	1	−.00659	(1)
	2	−.00754	(1)
	3	−.00665	(1)
CO_2–O_2 treatment	1	−.00918	(1)
	2	−.0132	(1)
	3	−.0127	(1)

[1] Sufficient data not taken.

The values obtained for these constants plainly demonstrate the importance of increased tension of O_2 in the lungs, as shown by a comparison of those for air treatment with those when O_2 was used. The ratio is nearly 1: 4, or approximately that of the relative proportions of O_2 in the inhaled air.

Further, a consideration of the constants for the CO_2–O_2 treatment emphasizes the importance of lung ventilation during elimination and indicates the value of depth of breathing as against mere increase of rate; for in tests 1 and 3 (Figs. 2 and 4) the rate increase was practically the same, but the volume used by the subject in test 3 was considerably larger, which agrees with the elimination as determined from the blood saturations. In test 2 (Fig. 3), it is much in evidence that the maximum efficacy of the treatment had been passed, as the exceedingly large increase in volume used did not result in a proportional increase in the rate of elimination. However, the constants obtained from all three of the experiments show a marked increase in the elimination of CO by treatment with the CO_2–O_2 mixture.

In order to condense the data from the different experiments, and represent the average efficacy of each of the three methods, curves were prepared, using arbitrary values for S′ in the equation found to represent the curve of elimination ($S' = S + (b \log e \times t)$), the constants b log e used being the averages found for each respective treatment (from Table IV). The curves obtained are shown in Figure 5.

These curves, in addition to their use in comparing the elimination resulting from the different treatments, are also of value in making fair estimates of the time required for the removal of carbon monoxide from the blood, should the saturation at any time be known. For example, supposing that a victim was found to have his hemoglobin 52 per cent saturated with CO, and had no other treatment than air, he would, under normal conditions, have approximately 4 per cent COHb remaining at the end of 600 minutes. Of course, as the initial saturation increases to the stage causing respiratory failure, the elimination will vary accordingly somewhat from the curves given, as they were determined from experiments during which the initial saturation did not exceed 42 per cent. In cases in which persons have reached a state of coma as a result of

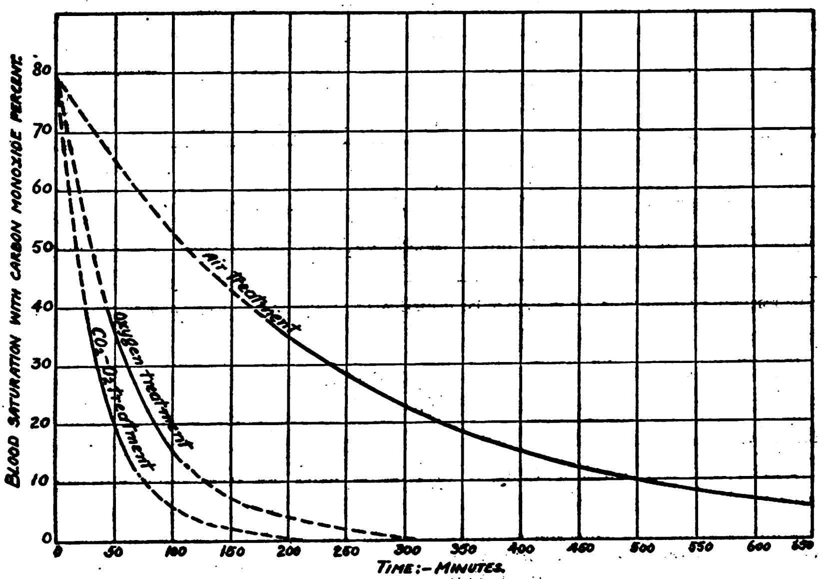

FIG. 5.—Average curves showing comparative elimination of carbon monoxide from the blood by the three different treatments. The data from which the curves were plotted were derived by means of the equation $S'=S+(b \log e)t$, using averages of the constants $b \log e$.

asphyxiation, the experience of other investigators[10] bears out the importance of the respiratory factor in that the curve will not initially follow that of a rectangular hyperbola. During the period of feeble respiration the elimination will be slower, but will increase directly with the respiration until the latter has assumed a fairly normal rate, and the curve of elimination will then merge with that of the form shown on Figure 5. However, the period of feeble respiration is comparatively short, and the speed of elimination is very rapid after the victim has been removed to fresh air, both of which factors affect the curves.

A series of results obtained from various sources by experimentation was compared with the results found by estimation from the curves, with the following results:

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[10] Personal communication from Y. Henderson.

TABLE V.—*Comparison of experimental data with values from Figure 5 of this report.*

[From data represented on curves of tests 1, 2, and 3, Figs. 2, 3, and 4, described in this report. Men used as subjects.]

Time (minutes).	Blood saturation.		Time (minutes).	Blood saturation.	
	By analysis.	From average, curve Fig. 5.		By analysis.	From average, curve Fig. 5.
Test 1—air treatment:			Test 2—air treatment:		
Initial	35.5	35.5	Initial	37.0	37.0
20	29.0	32.5	13	32.0	34.5
37	24.5	30.5	40	28.0	31.0
95	19.0	24.0	118	21.0	22.5
155	17.0	18.5	183	17.0	17.0
215	12.5	14.5	228	11.5	12.0
305	10.5	10.0	348	7.5	9.0
Test 1—oxygen treatment:			Test 1—(CO_2–O_2) treatment:		
Initial	33.0	33.0	Initial	34.0	34.0
24	20.5	22.0	24	19.0	18.0
45	15.0	16.0	45	12.5	11.0
Test 2—oxygen treatment:			Test 2—(CO_2–O_2) treatment:		
Initial	38.5	38.5	Initial	42.0	42.0
18	23.5	28.0	18	20.0	27.0
45	16.0	19.0	45	9.5	12.0
Test 3—oxygen treatment:			Test 3—(CO_2–O_2) treatment:		
Initial	42.0	42.0	Initial	35.0	35.0
16	24.0	32.0	18	22.5	22.0
45	17.5	20.0	45	10.0	11.0

[Data from an unpublished report of a similar investigation on carbon monoxide treatment by Dr. H. R. O'Brien. Men used as subjects.]

Oxygen treatment:			(CO_2–O_2) treatment:		
Initial	39.5	39.5	Initial	36.0	36.0
13	35.0	32.0	14	24.0	25.0
33	26.0	23.5	25	19.0	18.0
Oxygen treatment:			36	16.0	14.0
Initial	35.0	35.0	(CO_2–O_2) treatment:		
16	25.0	26.0	Initial	46.0	46.0
35	18.0	20.0	17	28.0	30.0

TABLE VI.—*Comparison of data obtained by other investigators* [1] *with those obtained from Figure 5 of this report.*

Time.	Blood saturation.		Time.	Blood saturation.	
	By analysis.	From average, curve Fig. 5.		By analysis.	From average, curve Fig. 5.
Air treatment:			Air treatment:		
Initial	22.0	22.0	Initial	18.0	18.0
150 minutes	5.0	12.0	60 minutes	12.0	14.0
Initial	26.0	26.0	120 minutes	5.0	11.0
60 minutes	17.0	20.0	Initial	20.0	20.0
Initial	27.8	....	30 minutes	17.0	18.0
30 minutes	27.5	24.5	60 minutes	14.0	16.0
70 minutes	16.2	20.5	90 minutes	8.0	14.0
100 minutes	19.6	18.0	Initial	21.0	21.0
175 minutes	5.1	14.0	180 minutes	8.0	10.0
210 minutes	0.0	12.0	Initial	18.0	18.0
250 minutes	0.0	10.0	60 minutes	6.4	14.0

[1] Data from Appendix 4, New York State Bridge and Tunnel Commission Report of Tunnel Gas Investigations Problem No. 2, "Physiological effects of exhaust cases." By Yandell Henderson. (Men used as subjects).

The results of this comparison indicate that with fairly normal respiration the average curves (Fig. 5) may be used to estimate within practical limits the rate of elimination, as a variation of 5 per cent is not exceedingly significant from the standpoint of physiological effects. In Table VI, the agreement is not as close as in Table V; but it must be remembered that, owing to the comparatively low initial saturations, the results are more likely to be affected by experimental error.

Thus it seems that the efficacy of the three methods of treatment is within the allowable limits, represented by the average curves, (Fig. 5) calculated from the data obtained as a result of this investigation. The blood was analyzed by two methods and by two different observers, with further confirmation in two instances by calculation of blood saturation from an analysis of alveolar air. The results of the different experiments are well in agreement with each other, there being no unexplainable erratic variations. The curves are similar in type to those representing the amount of a substance remaining in a reacting system after an interval of time. The constants appear to be interrelated in respect to oxygen tension and, to an extent, in respect to lung ventilation, which is in agreement with the work of other investigators.[11] It is unfortunate that no dependable data were obtainable for the elimination of CO as it occurs when the initial saturation is above 45 per cent; but owing to the risk incurred, it is not desirable to go higher in experimental work, as death might even occur at that point should the subject be in a poor physical condition. These high saturation results can only be obtained by chance observations of cases of accidental poisoning; and when such have been obtained, the dotted portions (50 to 80 per cent) of the curves shown on Figure 5 can be checked.

DISCUSSION OF SYMPTOMS RELATIVE TO METHOD OF TREATMENT.

The symptoms experienced by subjects during the exposure are considered mild to moderate. Headache and dizziness were the most common symptoms and were exaggerated on exercising. The pulse rate and the rate of respiration during the exposure remained practically normal, as shown in Tables I, II, and III. This was true also during treatment and after treatment, with the exception of the rate of respiration when breathing CO_2–O_2 mixtures.

The subjects who recovered in air had severe headaches, and were physically unfit for several hours after exposure, usually during the remainder of the day. The rate of elimination of CO from the blood when the subject was breathing air was very gradual, 5 to 6 hours

[11] The treatment of CO asphyxiation by means of oxygen, plus CO_2 inhalation. By Yandell Henderson and H. W. Haggard. Jour. Amer. Med. Assn., vol. 79, Sept. 30, 1922. pp. 1137-1145.

being required to reduce the hemoglobin saturation from about 35 per cent to 10 per cent.

The breathing of oxygen diminished or relieved the symptoms, and there were either no after effects or they were very much less severe than when recovery took place in normal air. This was especially striking in the case of subject Massey (Table II), who collapsed and was given oxygen for 45 minutes, after which he experienced no untoward symptoms other than feeling a little tired. The rate of elimination of carbon monoxide from the blood caused by breathing oxygen was much more rapid than that resulting from breathing normal air. By referring to the curves, it will be noted that the breathing of pure oxygen causes the elimination of the CO about four times as fast as the breathing of normal air.

The breathing of 8 to 10 per cent of CO_2 in O_2 caused deeper and more rapid respiration, which became tiring after a few minutes. The rate of respiration and the volume breathed increased from 50 to 100 per cent, varying with the subject. The after symptoms were more severe than those following the breathing of oxygen, but not as severe as when elimination was produced by normal air. The use of 8 to 10 per cent of CO_2 in O_2 caused the elimination of the CO from the blood five to six times as fast as the breathing of normal air; in other words, the CO_2 mixtures removed the CO from 25 to 50 per cent faster than did the pure O_2.

Henderson [12] states that breathing CO_2–O_2 mixtures produces an increase in blood pressure. This probably causes an increase of flow of blood through the lungs, and would thus aid in the elimination of CO; but in victims that have been gassed over a comparatively long period (several hours), the heart might perhaps be weakened and unable to withstand the increased stimulation for breathing caused by the CO_2. However, if the patient is under constant observation, overstimulation may be avoided by temporarily stopping the use of the CO_2–O_2 mixture; or, as suggested by Henderson and Haggard,[13] this effect may be prevented by the administration of atropin. Lower percentages of CO_2 in O_2 produce less effect; and it is hoped that a safe percentage may be found for routine first-aid work.

SUMMARY.

1. Recovery from carbon-monoxide poisoning depends to a great extent upon early elimination of carbon monoxide from the blood.
2. The rate of elimination of carbon monoxide from the blood depends upon the percentage of oxygen in the air breathed, also upon the rate and depth of respiration.

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[12] Administration of carbon dioxide after anesthesia and operation. By Yandell Henderson. Jour. Amer. Med. Assn., vol. 76, No. 10, Mar. 5, 1921, p. 672.

[13] Personal communication.

3. Pure oxygen causes the elimination of carbon monoxide about four times as fast as normal air, when breathed by persons who have been gassed until 35 or 40 per cent of the hemoglobin in their blood has been combined with carbon monoxide.

4. Breathing a mixture of oxygen containing 8 to 10 per cent of carbon dioxide causes deep and rapid respiration.

5. Breathing a mixture of oxygen and carbon dioxide (8 to 10 per cent) causes the elimination of carbon monoxide about five to six times as fast as normal air, when breathed by persons who have been gassed until 30 to 40 per cent of their blood has been combined with carbon monoxide.

6. It is recommended that all victims not under a physician's care be caused to breathe oxygen in the purest form available for at least 20 to 45 minutes.

7. It is recommended that physicians use the carbon dioxide-oxygen mixture where possible, and note the results, but when this mixture is not available that they use pure oxygen.

Acknowledgments.—Acknowledgment is made to Dr. W. J. McConnell and Dr. B. Massey, who aided in making these tests; to Mr. S. H. Katz, Mr. G. W. Jones, and other chemists of the Bureau of Mines gas laboratory, and to Mr. G. S. McCaa, and Mr. J. H. Zorn, of the mine safety service, all of the Pittsburgh experiment station of the Bureau of Mines; also to Dr. Yandell Henderson, of Yale University, who kindly read and criticized the report.

STUDIES ON THE PERMEABILITY OF LIVING AND DEAD CELLS.

III. THE PENETRATION OF CERTAIN ALKALIES AND AMMONIUM SALTS INTO LIVING AND DEAD CELLS.

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LIVING CELLS.

It has frequently been stated that penetration of the so-called weak bases is much more rapid than that of the strong bases. Warburg (1), Harvey (2), and others noted that NH_4OH readily penetrated living cells while NaOH and allied bases did not. Since ideal conditions for measuring the rate of penetration of bases into living plants by direct determinations were afforded through the structure of the marine alga, *Valonia ventricosa,* it was thought of interest to test these conclusions.

Methods for direct analysis of the sap of living plants have been developed by Osterhout (3) and by the writer (4) in the experiments on the fresh-water alga, *Nitella*. The internodes of this plant are cœnocytic cells, consisting of a central vacuole filled with cell-sap and surrounded by a delicate layer of protoplasm and by the cell-wall. It is possible to pierce the cell-wall by means of a sharp-pointed glass capillary pipette and collect the sap in an uncontaminated condition. The quantities of sap obtainable from *Nitella* are, however, very minute as compared with the amount of sap which can be obtained from *Valonia*, which was used for this investigation. *Valonia* is a spherical one-celled (cœnocytic) marine alga. The plants are of various sizes, usually yielding 5 c. c. to 10 c. c., but sometimes as much as 50 c. c. of sap. This quantity affords adequate amounts for accurate determinations. By expressing the cell-sap (free from contamination) into Pyrex tubes and adding indicators, the pH value of the sap of successive plants can be determined at intervals, and the rate of change in pH noted.

This method is described in full in a previous paper (5) on penetration of acids and will not be repeated here. It was found in the study on acids (5) that two sets of determinations for pH values of the sap were necessary: one set with and one without free CO_2. The free CO_2 present in the cell varied as the surrounding medium was changed and as the time during which these conditions prevailed.

When CO_2 is blown out of the cell-sap, there is removed not only the CO_2 which is free at the time that aeration begins but also a certain amount of CO_2 previously present in a combined state, which takes the place of the CO_2 already removed, until an equilibrium is attained which is compatible with the CO_2 tension of the so-called CO_2–free air which is introduced. Attention is called to this statement in order to avoid misunderstanding about the identity of the CO_2 of the sap which is removed through aeration.

Since analytical methods can not distinguish anhydrous CO_2 from H_2CO_3, these terms are both included in the term "free CO_2."

In addition to making "CO_2–free" determinations, it was also necessary, in this investigation, to remove the free NH_3 of the sap in those experiments where ammonium salts or bases were used. For this purpose the expressed sap was aerated with air which was also NH_3–free. The system used for aeration, therefore, consisted of a train of three flasks containing, respectively, NaOH, H_2SO_4, and distilled water.

The pH of normal sap with free CO_2 is from 6.2 to 6.4; and without free CO_2 it is 6.6 to 6.8 The sodium salt of brom thymol blue was used as an indicator. No correction was made for the salt error, which appears not to have been determined, but which in any case would not affect the conclusions derived from these data.

Atkins (6) has also estimated the reaction of some marine algæ and found it to be neutral or slightly alkaline. Since his estimations were made upon plants which had been cut or crushed and stained with indicators, the usual criticisms concerning injured tissues or extracted plant juices would apply to his results. He also took no account of the free CO_2 which probably escaped during the manipulation, thereby causing an increase in alkalinity which was not taken into account.

Hoagland and Davis (7) found by direct methods like those used by the writer that the pH of the sap of a California species of *Nitella* is 5.2. It is of interest to compare this with the pH of *Nitella* at Woods Hole, Mass., and at Washington, D. C., which was found by the writer (8) to be 5.7. These differences are probably due to specific or environmental differences. Similarly the pH of the sap of *Valonia* at Miami, Fla. (6.2 to 6.4), was found by the writer (5) to be higher than that of the Bermuda *Valonia* (6.0) as reported by Crozier (9).

Hoagland and Davis(7) proposed to find out directly by analyzing the cell-sap of *Nitella* whether changes in the alkalinity of the surrounding medium affected the pH of the sap. The water in which the plants were growing was allowed to become alkaline to pH 9.4 through the agency of photosynthesis. The authors noted no subsequent change in the pH of the sap and no exosmosis of chlorides, which always accompanies injury. Atkins (10), on the other hand, using the same method for producing alkalinity in sea water, found that injury occurred in the case of the superficial cells of *Ceramium rubrum*, but not in the case of *Ulva*.

Jacobs (11) subjected flowers of a hybrid *Rhododendron*, containing a natural indicator which turned blue in the presence of alkali, to solutions of ammonium sulphate, chloride, and acetate. Although these solutions were acid, nevertheless the petals of the plant became blue, showing that they had become alkaline.

Hoagland and Davis (7) tested, by the direct method, the reaction of the cell-sap of *Nitella* when placed in solutions of ammonium sulphate, chloride, and nitrate in concentrations of 0.005 M. They found that penetration of ammonium caused a change in pH from 5.2 to 6.2 in 24 hours or less. This change is much less than that obtained by the writer in the case of *Valonia*. Differences may be due to the effects of the salt content of the surrounding medium or to the composition of the sap itself. The influence of Ca on delaying the outward diffusion of chlorides is mentioned by Hoagland and Davis.

TABLE I.—*Viability of Valonia in sea water after exposure to 0.03 M solutions.*

Substance.	Length of life in days after exposure to solutions for indicated time in hours.							
	½	1½	2	2½	3½	4	4½	5½
NH_4OH	11	2	1	>1	....	....	....	....
NH_4Cl	17	....	....	10	....	....	....	6
$(NH_4)_2SO_4$	30	....	....	....	....	28	....	....
$(NH_4)_2CO_3$	....	....	14	....	....	....	....	....
NaOH	29	....	....	....	22	....	....	....
KOH	29	....	....	....	22	....	....	....

Injury in *Valonia* was determined by the time of survival after the plants had been taken out of the test solution and replaced in sea water alone (see Table I). Exosmosis of chlorides could not be used as a test in this case because of the predominance of chlorides in the sea water itself.

All of the bases and salts were dissolved in sea water. The bases precipitated a slight amount of magnesium; but this was found not to affect the viability of the plants. It was necessary to use sea water, as the plants died rapidly when artificial sea water, such as that used by Harvey, was substituted. Experiments were performed in which the pH of all the solutions was the same, and others in which the molar concentrations were alike.

The concentrations of the bases used, and the resultant pH of the solutions are given in Table II. There seems to be a buffer effect at a pH of about 10.0.

The temperature at which these experiments were done was about 24° C. This is the temperature of the sea water at Miami, and of the running sea water in the laboratory.

The plants were placed in the solutions for various periods of time. They were then removed, carefully and quickly dried on filter paper, and punctured by means of a pointed glass rod. The cell-sap is under pressure and is readily expressed into a Pyrex tube containing indicator. The pH is then noted.

TABLE II.—*Concentrations and pH values of solutions of bases and ammonium salts in sea water.*

Concentration.	pH.				
	Base.		Salt.		
	NH_4OH	NaOH or KOH	$(NH_4)_2SO_4$	NH_4Cl	$(NH_4)_2CO_3$
0.0015 M	9.0	10			
.003 M	9.4	10.4			
.006 M	10.0	12			
.009 M	10.0	12			
.030 M	10.0	12	7.5	7.6	7.8

The salts of ammonium, owing to the unequal dissociation of their acid and alkali radicals, are specially favorable for noting the effects of the anion and cation separately upon the changes of the sap of *Valonia.* NH_4Cl and $(NH_4)_2SO_4$ have an acid reaction because the acid radical is more highly dissociated than the alkali radical, while $(NH_4)_2CO_3$ has an alkaline reaction for the opposite reason. It was thought that by removing the free CO_2 and free NH_3 from the expressed sap of cells placed in these solutions (by aerating with CO_2–free–NH_3–free air), it would be possible to detect and measure the rate of entrance of the anion, i. e., chloride, sulphate, hydroxide, or carbonate, by noting the divergence of the pH from normal.

The experiments of the writer on the changes in pH value of the sap of *Valonia* as affected by ammonium salts confirm, in general, those noted by previous observers. They include, however, not only the end-point of the reaction as obtained by these writers, but also the time curve showing the rate and manner of penetration, and the relative penetration of the different ions.

Figure 1 shows the penetration of NH_4OH into the cell sap of *Valonia.* The buffer effect at pH 10, noted when ammonia is added to sea water, is not apparent in the cell sap. In the highest concentration of NH_4OH (0.03 M), even though the pH of the surrounding liquid does not exceed 10, that of the cell sap becomes 11.5 Curves C all show the pH of the sap as it is expressed, before removing any of the free CO_2 and NH_3. Curve D shows that the pH of the sap is 6.6 after removal of free CO_2 and NH_3. This is the same pH as that of normal cells. Therefore no permanent chemical combinations with the components of the sap are made, although reversible combinations may occur. The abscissæ represent time in minutes and the ordinates represent the pH values. The dotted lines indicate the lapse of intervals of time too great to be included on the same scale of abscissæ. These data show that the concentration of free base tends to become equalized very quickly on both sides of the semipermeable membrane, the pH value of the sap being taken to indicate the amount of free base penetrating. Because of buffer action the pH of the sea water was constant (10) in the 3 highest concentrations of ammonia, but the pH of the sap itself became higher than 10 in the two highest concentrations because no such buffer effect was present.

When qualitative tests with Nessler's reagent were made upon the sap of cells which had been in ammonium solutions, a heavy precipi-

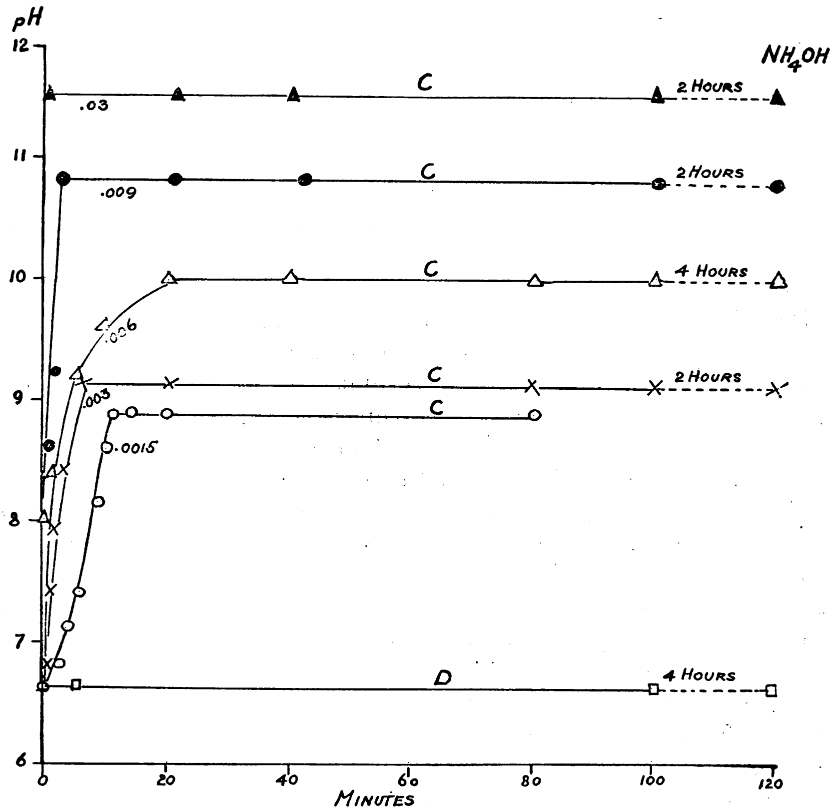

FIG. 1.—The penetration of NH_4OH into the cell-sap of *Valonia*. Curves C all show the pH of the sap as soon as it has been expressed after having been in solutions of the indicated concentrations for the periods of time represented. The dotted lines represent periods of time too great to be shown on the same scale. Curve D represents for all four of the above solutions the pH of the sap after the free CO_2 and NH_3 have been removed by aeration.

tate was obtained which showed the presence of ammonia. There seemed to be a slight precipitate in normal sap, but not nearly comparable with the precipitate in the sap of treated cells. The odor of ammonia as the sap was expressed was also unmistakable. It is hoped that quantitative estimations on ammonia penetration by means of the Nessler reaction will be carried out in the near future.

Figure 2 shows the penetration of NH_4Cl. Curve C shows that the pH of the sap becomes 8.6 in one hour and remains at this pH for 5 hours. Curve D shows that when the free NH_3 is removed from the

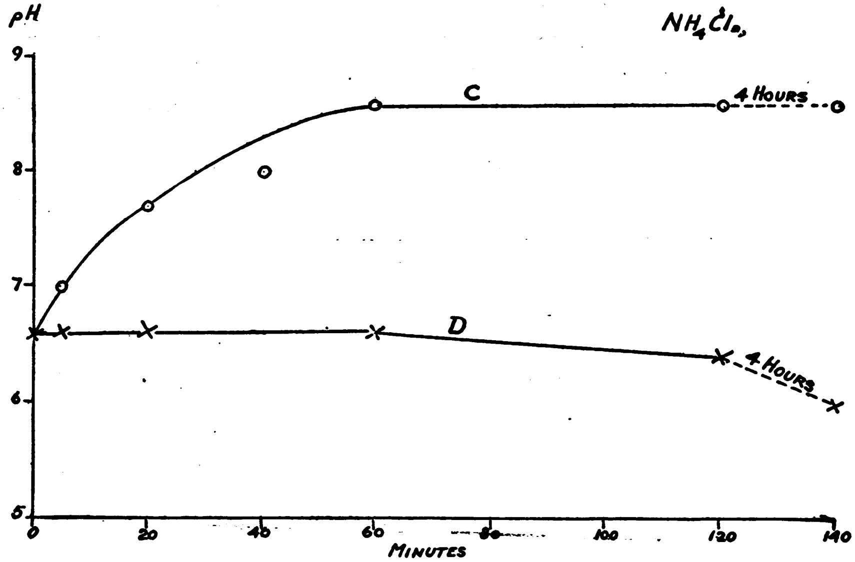

Fig. 2.—The penetration of NH_4Cl. Curve C shows the pH of the sap as it is expressed, and curve D the same after aeration.

sap, a pH of 6.6, which is normal, still persists. After one hour, however, the aerated sap shows a greater acidity than pH 6.6, owing, probably, to the penetration of HCl from the dissociated NH_4Cl.

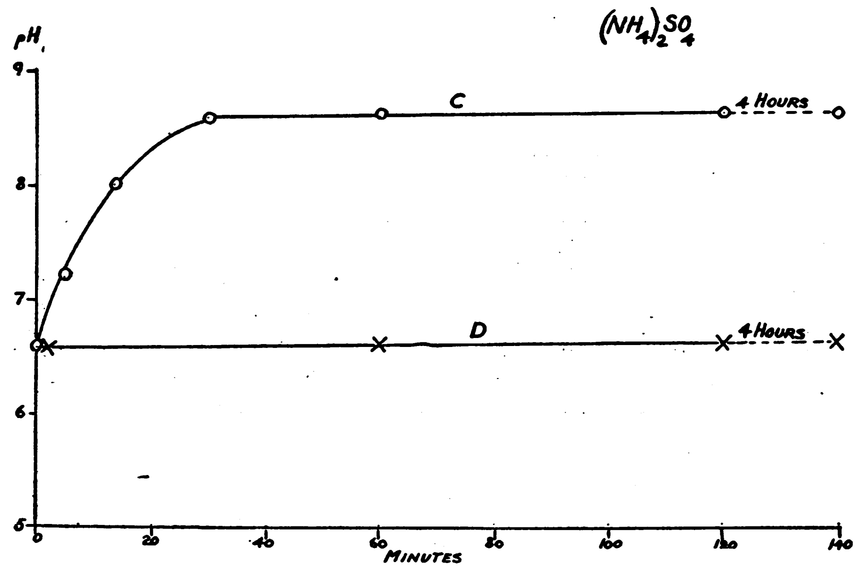

Fig. 3.—The penetration of $(NH_4)_2SO_4$. Curve C shows the pH of the sap as it is expressed, and curve D after aeration.

Figure 3 shows the effects of $(NH_4)_2SO_4$. As in Figure 2, the sap shows an increase in alkalinity to pH 8.6 in 30 minutes, a more rapid increase than in the case of NH_4Cl. This pH remains constant for 4 hours (curve C). Curve D shows that when the free NH_3 is removed from the sap the pH is 6.6. No increase in acidity is noted, owing, probably, to the impermeability of the cell to the sulphate ion. This observation is of interest, as it confirms the previous data of the writer on the slow rate of penetration of sulphuric acid into *Valonia.*

The results with ammonium carbonate were thought to be of special interest, inasmuch as both ions of this compound have such facility in entering the sap. Figure 4 shows the effects of the penetration of both the NH_4 and CO_3 ions. When the sap from a cell which has been 20 minutes in the solution is first expressed, the

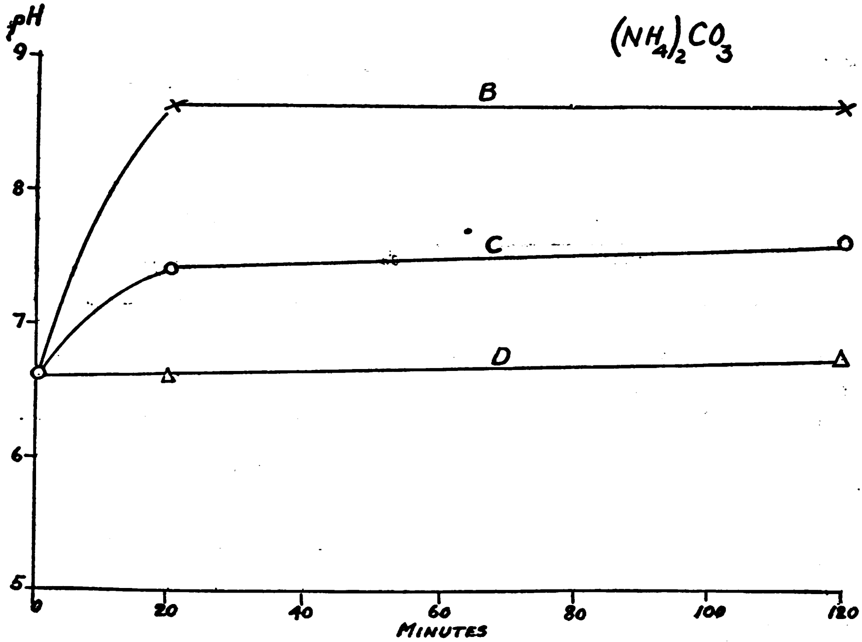

FIG. 4.—The penetration of $(NH_4)_2CO_3$. Curve C represents the pH value of the sap as it is expressed; curve B during, and curve D at the end of aeration. Curve B represents in each case the maximum of alkalinity due to the removal of an excessive proportion of CO_2.

pH is 7.5 (curve C). It is possible to follow the effect of the CO_2-free-NH_3-free air upon the expressed sap by noting the color changes in the indicator as the compressed air is blown through the sap. Since the sap at first became more alkaline (pH 8.6), the CO_2 was evidently more easily removable than the NH_3. Even after the cell had been 2 hours in an $(NH_4)_2CO_3$ solution the sap still behaved the same as shown in curve B. As some NH_3 also is undoubtedly removed, together with the CO_2, the degree of alkalinity probably does not indicate the full amount of NH_3 which has penetrated. Continued aeration of the sap removed the free NH_3 and produced a lower pH. When pH 6.6 was reached, no more change in pH by aeration could be produced. This is the same pH as that of the normal sap, represented by curve D, and suggests that now all the entering salt has been removed. As no one indicator covered the entire pH range, it was necessary to obtain these readings by taking out samples of sap at intervals and adding the appropriate indicator.

Figure 5 shows a typical experiment with NaOH and KOH in concentrations of 0.003 M (pH 10.4). Lesser concentrations produced an even slower rate of change in pH of the sap. The use of higher concentrations (0.03 M), the pH of which was about 12, did not materially increase the rate of change of pH of the sap. Curves A show the pH of the sap when the free CO_2 is not removed and curves B show the pH when the free CO_2 is removed. In the case of NaOH,

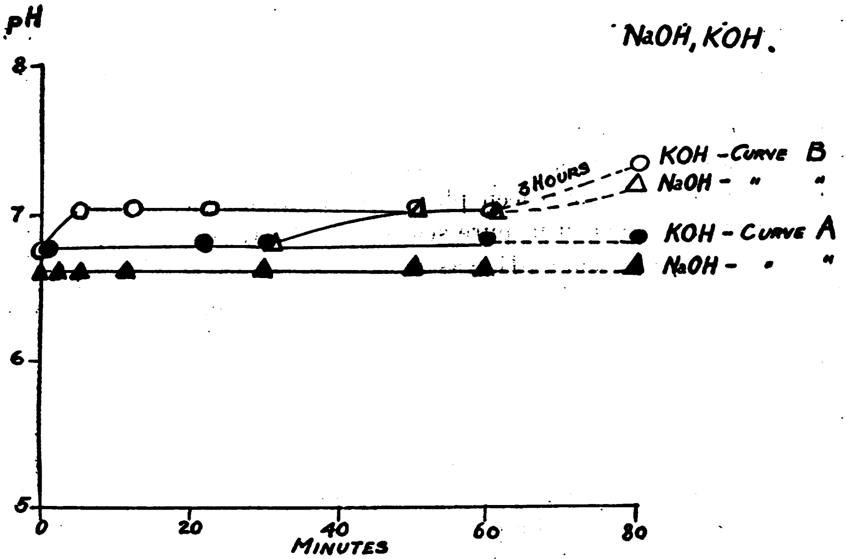

FIG. 5.—The penetration of NaOH and KOH (0.003 M). Curves A show the pH of the sap as it is expressed, and curves B after the free CO_2 has been removed.

curve A remains at a normal pH throughout the experiment, whereas the corresponding curve KOH shows a slightly greater alkalinity, as though there might have been some penetration of potassium into the sap. Again, curve B shows a more rapid increase in pH in the case of KOH than in the case of NaOH, the pH becoming 7.0 in five minutes while there is no change in the case of NaOH. The ultimate pH of the former is also slightly greater than that of the latter. The rate of penetration of K compounds is, therefore, under similar conditions, slightly more rapid than that of those containing Na. This observation agrees with the observations reported on various K and Na compounds by the writer (12) in a previous publication. It is is also evident that the penetration of the strong bases NaOH and KOH is much less rapid than that of NH_4OH, even when the concentration of the latter is much lower. This observation agrees with the observations of former investigators on the relative rates of penetration of strong and weak bases.

The viability of *Valonia* in these various solutions is given in Table I. This shows that NH_4OH is the most toxic of the substances used, and that NH_4Cl is more toxic than $(NH_4)_2SO_4$. The slower rate of penetration of the sulphate ion has probably some influence upon the degree of toxicity. This is of interest when compared with the results obtained by the writer in the study on acids, in which it was found that HCl is more toxic than H_2SO_4, and also that sulphuric acid has a slower rate of penetration than hydrochloric acid.

With ammonium salts it was found that the pH of the sap before removal of free NH_3, as represented by curves C, never exceeded that of the solution in which the cell was placed. This may be contrasted with the case of concentrated solutions of NH_4OH in sea water, which was sufficiently alkaline to call into play a buffer mechanism in the sea water but not in the sap, and as a consequence allowed the pH of the sap to exceed that of the outside solution.

When cells which had been placed in solutions of NH_4OH in sea water were again placed in pure sea water, the time required for the sap to regain its normal pH was about two or three times as long as that necessary for the original change in the opposite directions.

In order to determine whether the penetration of the Na ion was affected by the OH ions, two other types of solution were tested: one a solution of Na silicate in sea water (pH 9.3), and the other a solution of Na butyrate in sea water (pH 7.9). The same molecular concentration was used for these solutions as for the hydroxides. Na silicate undergoes hydrolytic dissociation to a considerable extent. There was no change in the pH of the sap after 3 hours in this solution. Presumably neither sodium nor silicate ions penetrated. In the case of Na butyrate, hydrolytic dissociation also occurs; but it might be thought that in this case, since the butyrate ion enters so readily (5), the Na ion also would enter. No change in the pH of the sap took place within 18 minutes. After 27 minutes, however, the pH became 7.5; but there was by this time decided injury of the cell, which cytolyzed when taken out and placed in sea water. It seems that in this case the butyrate ion entered without or with very little Na. These experiments agree in showing that Na has little penetrating power at any H ion concentration within the pH range 7.9 to 12. It is apparently the impermeability to Na which excludes NaOH from the cell sap. In the case of NH_4OH, it was not possible to determine whether penetration of the whole molecule takes place or only of the NH_3.

DEAD CELLS.

When dead cells were placed in solutions of the salts of ammonium used in these studies, their sap rapidly assumed the pH of the surrounding solution: $(NH_4)_2SO_4$, pH 7.5; NH_4Cl, pH 7.8; and $(NH_4)_2CO_3$, pH. 7.8 (illustrated in Table III). When the expressed sap from dead cells previously exposed to these solutions is placed in Pyrex tubes and aerated by CO_2-free–NH_3-free air, the pH of the resulting sap becomes, respectively, 5.2, 5.2, and 8.8. These values are the same as those assumed by the solutions of these salts in sea water when they are aerated in the same way. In the case of the sulphate and chloride, the acidity is due to the nonvolatile acid left behind, and in the case of the carbonate, to the nearly complete removal of both ions. Both the sap and the solutions were aerated for from 4 to 5 hours, at the end of which time further change had practically ceased.

TABLE III.—*pH of sap of dead cells of Valonia and of surrounding solution before and after aeration with compressed CO_2-free–NH_3-free air. Cells were 30 minutes in solution.*

Substance.	Sap of *Valonia*.		Solution in sea water.	
	Before aeration.	After aeration.	Before aeration.	After aeration.
$(NH_4)_2SO_4$(0.03 M)	7.6	5.2	7.5	5.2
NH_4Cl(0.03 M)	7.6	5.2	7.6	5.2
$(NH_4)_2CO_3$(0.03 M)	7.8	8.8	7.8	8.8
NH_4OH(0.03 M)	10.0	9.4	10.8	9.4
NaOH(0.003 M)	10.2	10.2	10.4	10.4
KOH(0.003 M)	10.2	10.2	10.4	10.4
Sea water	8.6	9.1	8.6	9.1

DISCUSSION.

These experiments further corroborate the theory that the internal H ion concentration of a cell is not determined primarily by that of the surrounding solution, but that the relative H ion concentration of the sap and the environing fluid may be profoundly affected by the nature of the acid or basic radicals present. This is emphasized by the comparable experiments on NaOH and NH_4OH, which had practically the same H ion concentration and yet had decidedly different effects upon the pH of the sap of *Valonia*.

Although some writers have called attention to the importance of other than H ions, the importance of the H ions seems to have been quite often overestimated. Before definite conclusions as to the influence of H or OH ions can be stated, the effects of the chemical nature of the accompanying anions or cations must be determined.

The great permeability of *Valonia* to CO_2 and NH_3 or their derivative ions raises some interesting questions. The case of ammonia is particularly interesting, since Vanzetti (14) has shown that when an ammonium salt in aqueous solution is allowed to diffuse against gravity, the acid radical travels more rapidly than the base, and is in excess in the forefront of diffusion. This finding is in accord with the migration velocities of the ions as determined by the method of transference numbers. Yet when NH_4Cl, for example, passes through the cell wall and the protoplasm of *Valonia*, the basic radical penetrates with very much the greater rapidity: the cell sap becomes alkaline and ammonia is found to be present in it. In the case of carbon dioxide the anion penetrates with a relative rapidity strikingly in excess of that which would be predicted from the transference numbers.

It will be evident that in trying to account for these differences we must look for distinguishing features—presumably chemical—in the behavior of the two ions concerned, which would place them in peculiar relationship to the protoplasm. Two possible explanations are of particular interest: One is based upon the pseudo-acid and pseudo-basic characters of CO_2 and NH_3, and the other upon the possibility that they may exist in solution in the form of dissolved gas molecules.

In the latter case we would have to consider the passage of particles which are electrically neutral, and might, for this reason, more easily pass through the wall and protoplasm. Such particles would presumably carry relatively little water of hydration, which would give them additional facility of penetration.

The explanation based upon the pseudo-acid or pseudo-basic nature of the substances in question has been suggested by Loeb (15), who explains the accelerating effect of sodium acetate upon the penetration of acetic acid into *Fundulus* eggs as the result of an increase in the tautomeric or "aci-" form of the acid. This form is considered to be capable of penetrating the cell with some ease. The same possibility of transformation into an "aci-"form exists for carbonates and ammonium compounds; and it may be supposed that for this reason *Valonia* and other forms are particularly permeable to these "aci-"tautomers.

The penetration of acids, bicarbonates, alkalies, and ammonium salts depends at least in part, then, upon the chemical nature of the ions present. For this reason it is interesting to note the fact, expressed by Loeb (13) (p. 707) that on the acid side of the isoelectric point, "the effect of salts on the membrane potentials, osmotic pressure, swelling of gelatine chloride, and that type of viscosity which is due to the swelling of protein particles, depends only on the valency and not on the chemical nature of the anion of the salt."

By analogy with the experiments of Loeb, one might reasonably inquire whether those properties (swelling, viscosity, etc.) which depend only on the valency of the ions present have anything to do with the regulation of permeability. Certainly in the case of *Valonia* the chemical nature of the ions plays the determining part.

SUMMARY.

1. Ammonium salts (NH_4Cl, $(NH_4)_2SO_4$, and $(NH_4)_2CO_3$ and NH_4OH cause an increase in the pH value of the sap of *Valonia.* This increase is due to penetration of free NH_3, which may be removed by aeration of the sap with NH_3-free air.

2. Increased acidity of the NH_3-free sap, presumably due to the penetration of anions, was observed to be greater in the case of chlorides than in the case of sulphates.

3. Both ions of $(NH_4)_2CO_3$ penetrate and can be removed by aeration, the CO_2 being more rapidly removed than NH_3.

4. Different concentrations of NH_4OH having the same pH in sea water may produce different pH values of the sap.

5. KOH penetrates very slowly but still slightly faster than NaOH.

6. The sap of dead cells assumes the same pH value as the solutions in which they are placed. Their aerated sap has a pH value equivalent to the pH after aeration of the solutions in which they are placed.

Acknowledgments.—The writer takes pleasure in acknowledging the courtesies afforded by the Miami Aquarium Association where this work was done, and in expressing much gratitude to the authorities of the Carnegie Institute of Washington, who made arrangements for collecting the plants.

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THE AMERICAN DIETETIC ASSOCIATION TO MEET IN OCTOBER.

The American Dietetic Association will hold its sixth annual meeting at Indianapolis on October 15, 16, and 17.

Among the speakers will be the following:

Mrs. Octavia Hall Smillie, president of the association.
Miss Effie Raitt, University of Washington.
Dr. Russel Wilder, Mayo Clinic.
Dr. A. C. Clowes, Eli Lilly Co.
Dr. Ruth Wheeler, University of Iowa.
Dr. Louis Burlingham, Barnes Hospital.
Dr. Amy Daniels, University of Iowa.
Mrs. Gertrude Gates Mudge, and
Miss Lydia Roberts, University of Chicago.

The program will cover every phase of applied dietetics.

DEATHS DURING WEEK ENDED AUGUST 25, 1923.

Summary of information received by telegraph from industrial insurance companies for week ended August 25, 1923, and corresponding week of 1922. (From the Weekly Health Index, August 28, 1923, issued by the Bureau of the Census, Department of Commerce.)

	Week ended Aug. 25, 1923.	Corresponding week, 1922.
Policies in force	54,748,544	49,858,834
Number of death claims	9,600	7,691
Death claims per 1,000 policies in force, annual rate	9.1	8.0

Deaths from all causes in certain large cities of the United States during the week ended August 25, 1923, infant mortality, annual death rate, and comparison with corresponding week of 1922. (From the Weekly Health Index, August 28, 1923, issued by the Bureau of the Census, Department of Commerce.)

City.	Week ended Aug. 25, 1923.		Annual death rate per 1,000, corresponding week, 1922.	Deaths under 1 year.		Infant mortality rate, week ended Aug. 25, 1923.[2]
	Total deaths.	Death rate.[1]		Week ended Aug. 25, 1923.	Corresponding week, 1922.	
Total	5,935	10.6	10.7	884	953	....
Akron, Ohio	20	5.0	5.5	5	4	59
Albany, N. Y.[3]	32	14.2	15.3	5	5	111
Atlanta, Ga	61	14.3	15.6	9	6	....
Baltimore, Md.[3]	164	11.1	11.8	28	36	82
Birmingham, Ala	51	13.6	9.8	6	5	....
Boston, Mass	170	11.5	11.6	20	34	57
Bridgeport, Conn	26	9.4	9.8	6	6	83
Buffalo, N. Y	114	11.1	10.6	22	24	92
Cambridge, Mass	18	8.4	9.4	4	3	71
Camden, N. J.[3]	34	14.3	10.3	8	5	132

[1] Annual rate per 1,000 population.
[2] Deaths under 1 year per 1,000 births—an annual rate based on deaths under 1 year for the week and estimated births for 1922. Cities left blank are not in the registration area for births.
[3] Deaths for week ended Friday, Aug. 24, 1923.

Deaths from all causes in certain large cities of the United States during the week ended August 25, 1923, infant mortality, annual death rate, and comparison with corresponding week of 1922. (From the Weekly Health Index, August 28, 1923, issued by the Bureau of the Census, Department of Commerce)—Continued.

City.	Week ended Aug. 25, 1923.		Annual death rate per 1,000, corresponding week, 1922.	Deaths under 1 year.		Infant mortality rate, week ended Aug. 25, 1923.
	Total deaths.	Death rate.		Week ended Aug. 25, 1923.	Corresponding week, 1922.	
Chicago, Ill	504	9.1	11.0	70	101	63
Cincinnati, Ohio	120	15.4	9.8	22	11	145
Cleveland, Ohio [3]	164	9.6	10.3	24	23	69
Columbus, Ohio	59	11.8	9.3	6	9	62
Dallas, Tex	31	9.1	13.6	5	3	....
Dayton, Ohio	36	11.3	11.6	5	2	82
Denver, Colo	66	12.7	11.5	10	6	....
Des Moines, Iowa	25	9.3	....	2	....	....
Detroit, Mich	196	10.3	8.1	34	30	68
Duluth, Minn	14	6.9	....	2	....	46
Erie, Pa	18	8.3	13.3	0	5	0
Fall River, Mass. [3]	24	10.3	16.4	4	11	57
Flint, Mich	26	11.5	....	5	....	99
Fort Worth, Tex	13	4.7	10.9	2	4	....
Grand Rapids, Mich	25	8.9	8.0	6	3	95
Houston, Tex	35	11.8	9.7	5	2	....
Indianapolis, Ind	103	15.7	13.9	23	13	177
Jacksonville, Fla	23	12.0	10.7	3	3	....
Jersey City, N. J	71	12.0	9.7	14	15	94
Kansas City, Mo	72	10.7	13.5	10	12	....
Los Angeles, Calif	204	16.0	13.6	27	26	101
Louisville, Ky	50	10.1	11.8	5	7	54
Lowell, Mass	29	13.1	11.4	6	7	104
Lynn, Mass	15	7.6	....	1	....	26
Memphis, Tenn	58	17.8	17.4	4	6	....
Milwaukee, Wis	98	10.5	6.3	20	9	99
Minneapolis, Minn	52	6.6	9.0	6	6	33
Nashville, Tenn. [3]	34	14.6	13.0	6	3	....
New Bedford, Mass	25	10.0	9.4	12	5	187
New Haven, Conn	34	10.2	10.4	5	4	65
New Orleans, La	103	13.3	15.3	6	26	....
New York, N. Y	1,132	10.0	9.4	170	173	68
Bronx Borough	140	8.7	7.0	15	11	53
Brooklyn Borough	365	8.8	7.9	58	57	62
Manhattan Borough	502	11.5	11.7	83	94	81
Queens Borough	84	8.2	7.5	9	9	48
Richmond Borough	41	16.8	14.7	5	2	91
Newark, N. J	94	11.2	11.5	16	15	75
Norfolk, Va	24	7.9	8.3	4	2	71
Oakland, Calif	53	11.5	5.8	4	4	51
Omaha, Nebr	63	16.1	10.9	11	6	119
Paterson, N. J	18	6.7	11.3	0	8	0
Philadelphia, Pa	407	11.0	10.7	73	80	95
Pittsburgh, Pa	106	9.0	13.0	18	24	63
Portland, Oreg	40	7.6	10.1	5	4	51
Providence, R. I	50	10.8	11.5	12	12	98
Richmond, Va	54	15.6	12.0	12	7	147
Rochester, N. Y	60	9.8	10.7	2	12	16
St. Louis, Mo	177	11.5	9.3	17	15	....
St. Paul, Minn	46	9.9	7.2	6	5	55
Salt Lake City, Utah [3]	19	7.8	7.2	1	2	16
San Antonio, Tex	39	11.0	15.2	8	10	....
San Francisco, Calif	114	11.0	11.7	4	9	24
Seattle, Wash	52	8.6	7.6	3	5	27
Spokane, Wash	20	10.0	13.0	0	8	0
Springfield, Mass	19	6.9	9.3	1	5	14
Tacoma, Wash	12	6.2	....	2	....	50
Toledo, Ohio	59	11.5	12.2	7	9	71
Trenton, N. J	23	9.4	12.5	4	6	68
Utica, N. Y	20	10.1	....	2	....	42
Washington, D. C	99	11.8	12.9	13	19	74
Wilmington, Del	20	8.9	13.1	7	5	142
Worcester, Mass	45	12.2	10.5	6	6	69
Yonkers, N. Y	22	10.7	7.9	5	2	108
Youngstown, Ohio	31	12.2	13.8	8	6	109

[3] Deaths for week ended Friday, Aug. 24, 1923.

PREVALENCE OF DISEASE.

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

UNITED STATES.

CURRENT STATE SUMMARIES.

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

Reports for Week Ended Sept. 1, 1923.

ARKANSAS.

	Cases.
Chicken pox	14
Diphtheria	10
Influenza	11
Malaria	232
Measles	29
Mumps	4
Paratyphoid fever	4
Pellagra	7
Poliomyelitis	2
Scarlet fever	1
Trachoma	3
Tuberculosis	4
Typhoid fever	42
Typhus fever	4
Whooping cough	28

COLORADO.

(Exclusive of Denver.)

Chicken pox	7
Diphtheria	13
Measles	56
Mumps	1
Pneumonia	1
Scarlet fever	1
Tuberculosis	66
Typhoid fever	21
Whooping cough	17

CONNECTICUT.

Chicken pox	4
Diphtheria	30
Dysentery (amebic)	1
Dysentery (bacillary)	2
German measles	3
Influenza	2
Malaria	2
Measles	13

CONNECTICUT—continued.

	Cases.
Mumps	5
Pneumonia (lobar)	4
Poliomyelitis	8
Scarlet fever	36
Tetanus	1
Trichinosis	4
Tuberculosis (all forms)	23
Typhoid fever	9
Whooping cough	37

DELAWARE.

Diphtheria	2
Influenza	3
Measles	1
Pneumonia	1
Scarlet fever	8
Tuberculosis	3
Typhoid fever	6

FLORIDA.

Dengue	2
Diphtheria	11
Malaria	26
Pneumonia	1
Scarlet fever	1
Typhoid fever	8

GEORGIA.

Cerebrospinal meningitis	1
Dengue	1
Diphtheria	27
Hookworm disease	6
Malaria	55
Measles	23
Mumps	2
Paratyphoid fever	1
Pneumonia	11
Scarlet fever	6

GEORGIA—continued.

	Cases.
Septic sore throat	1
Smallpox	4
Tuberculosis	11
Typhoid fever	21
Typhus fever	1
Whooping cough	14

ILLINOIS.

Cerebrospinal meningitis:	
Adams County	1
Diphtheria:	
Cook County	61
Scattering	37
Influenza	1
Lethargic encephalitis—Chicago	1
Pneumonia:	
Chicago	63
Scattering	10
Poliomyelitis:	
Chicago	9
Kankakee County	1
McLean County	1
Madison County	1
Mason County	1
Piatt County	1
Scarlet fever:	
Cook County	16
Scattering	32
Smallpox:	
Macon County	8
Location not given	1
Typhoid fever:	
Cook County	12
Scattering	71
Whooping cough	127

INDIANA.

Diphtheria	18
Measles	14
Scarlet fever	12
Smallpox	7
Tuberculosis	26
Typhoid fever	31

IOWA.

Diphtheria	23
Poliomyelitis	6
Scarlet fever	18
Smallpox	5
Typhoid fever	5

KANSAS.

Chicken pox	2
Diphtheria	29
Malaria	4
Measles	24
Mumps	6
Pneumonia	8
Poliomyelitis	11
Scarlet fever	54
Smallpox	3
Tuberculosis	43
Typhoid fever	52
Whooping cough	60

LOUISIANA.

	Cases.
Dengue	51
Diphtheria	28
Malaria	2
Measles	4
Poliomyelitis	2
Scarlet fever	5
Smallpox	1
Tuberculosis	29
Typhoid fever	24
Whooping cough	4

MARYLAND.

Chicken pox	1
Diphtheria	47
Dysentery	8
Influenza	4
Lethargic encephalitis	1
Malaria	1
Measles	50
Mumps	2
Pneumonia (all forms)	24
Poliomyelitis	1
Scarlet fever	27
Tetanus	1
Tuberculosis	53
Typhoid fever	48
Whooping cough	49

MASSACHUSETTS.

Cerebrospinal meningitis	1
Chicken pox	18
Conjunctivitis (suppurative)	8
Diphtheria	124
Dysentery	1
German measles	2
Influenza	3
Lethargic encephalitis	2
Malaria	1
Measles	35
Mumps	20
Ophthalmia neonatorum	13
Pellagra	1
Pneumonia (lobar)	11
Poliomyelitis	10
Scarlet fever	54
Septic sore throat	6
Trichinosis	1
Tuberculosis (all forms)	90
Typhoid fever	21
Whooping cough	77

MICHIGAN.

Diphtheria	124
Measles	46
Pneumonia	49
Scarlet fever	104
Smallpox	15
Tuberculosis	280
Typhoid fever	26
Whooping cough	105

MINNESOTA.

(Exclusive of Minneapolis, St. Paul, and Duluth.)

Diphtheria	24
Measles	14
Poliomyelitis	1

MINNESOTA—continued.

	Cases.
Scarlet fever	34
Smallpox	5
Tuberculosis	51
Typhoid fever	5
Whooping cough	8

MISSISSIPPI.

Diphtheria	23
Scarlet fever	4
Typhoid fever	33

MISSOURI.

(Exclusive of St. Louis, Cape Girardeau, and Springfield.)

Diphtheria	26
Epidemic sore throat	1
Influenza	5
Measles	16
Mumps	1
Poliomyelitis	1
Scarlet fever	28
Smallpox	1
Trachoma	7
Tuberculosis	6
Typhoid fever	25
Whooping cough	27

MONTANA.

Diphtheria	8
Poliomyelitis—Great Falls	1
Rocky Mountain Spotted fever—Hamilton	1
Scarlet fever	5
Smallpox	7
Typhoid fever	10

NEW JERSEY.

Cerebrospinal meningitis	4
Chicken pox	6
Diphtheria	54
Influenza	4
Malaria	2
Measles	16
Paratyphoid fever	2
Pneumonia	30
Poliomyelitis	18
Scarlet fever	17
Typhoid fever	21
Whooping cough	55

NEW MEXICO.

Diphtheria	20
Measles	4
Scarlet fever	3
Tuberculosis	29
Typhoid fever	29
Whooping cough	4

NEW YORK.

(Exclusive of New York City.)

Cerebrospinal meningitis	1
Diphtheria	75
Influenza	5

[1] Deaths.

NEW YORK—continued.

	Cases.
Measles	95
Pneumonia	42
Poliomyelitis	18
Scarlet fever	60
Smallpox	3
Typhoid fever	38
Whooping cough	152

NORTH CAROLINA.

Chicken pox	6
Diphtheria	96
German measles	1
Measles	126
Scarlet fever	33
Septic sore throat	5
Smallpox	20
Typhoid fever	60
Whooping cough	284

OREGON.

Chicken pox	7
Diphtheria	11
Measles	4
Pneumonia	[1] 5
Scarlet fever	5
Septic sore throat	[1] 1
Smallpox	9
Tuberculosis	11
Typhoid fever	9
Whooping cough	9

SOUTH DAKOTA.

Chicken pox	1
Diphtheria	5
Measles	7
Poliomyelitis	3
Scarlet fever	12
Tuberculosis	5
Whooping cough	4

TEXAS.

Dengue	8
Diphtheria	12
Dysentery (epidemic)	1
Measles	7
Mumps	3
Pellagra	1
Pneumonia	2
Scarlet fever	7
Smallpox	17
Trachoma	1
Typhoid fever	50
Tuberculosis	16
Whooping cough	23

VERMONT.

Chicken pox	5
Diphtheria	2
Measles	22
Mumps	5

VERMONT—continued.

	Cases.
Pneumonia	1
Poliomyelitis	3
Scarlet fever	2
Whooping cough	15

VIRGINIA.

	Cases.
Poliomyelitis:[1]	
Halifax County	[1] 2
Stafford County	[1] 4
Fauquier County	[1] 1
Caroline County	[1] 1
Bland County	1
Wythe County	1

WASHINGTON.

	Cases.
Chicken pox	5
Diphtheria	7
Measles	8
Mumps	2
Scarlet fever	26
Smallpox	7
Tuberculosis	23
Typhoid fever	23
Whooping cough	44

WEST VIRGINIA.

	Cases.
Diphtheria	7
Scarlet fever	16
Typhoid fever	11

Reports for Week Ended August 25, 1923.

DISTRICT OF COLUMBIA.

	Cases.
Chicken pox	1
Diphtheria	3
Measles	2
Scarlet fever	2
Tuberculosis	26
Typhoid fever	6
Whooping cough	13

NEBRASKA.

	Cases.
Diphtheria	16
Measles	1
Mumps	1
Pneumonia	1
Poliomyelitis	1
Scarlet fever	12

NEBRASKA—continued.

	Cases.
Tuberculosis	3
Typhoid fever	3
Whooping cough	5

NORTH DAKOTA.

	Cases.
Chicken pox	2
Diphtheria	16
Measles	24
Poliomyelitis	1
Scarlet fever	27
Smallpox	3
Trachoma	5
Tuberculosis	6
Typhoid fever	10
Whooping cough	12

SUMMARY OF CASES REPORTED MONTHLY BY STATES.

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

State.	Cerebrospinal meningitis.	Diphtheria.	Influenza.	Malaria.	Measles.	Pellagra.	Poliomyelitis.	Scarlet fever.	Smallpox.	Typhoid fever
June, 1923.										
Oklahoma	1	16	30	10	215	7	......	34	90	55
July, 1923.										
Colorado	1	159	......	......	291	......	......	62	2	42
District of Columbia	1	11	......	......	73	......	......	23	1	12
Hawaii	......	24	154	......	11	......	......	6	......	17
Idaho	......	1	......	......	12	......	......	3	3	......
Iowa	......	58	1	......	60	......	......	61	22	8
Kansas	......	89	1	......	435	1	8	90	17	108
Michigan	9	371	......	......	2,027	2	3	453	118	77
Mississippi	......	60	89	13,287	664	880	4	14	22	327
Montana	......	11	......	......	49	......	......	37	21	19
Ohio	6	382	4	4	960	......	9	442	124	223
Oregon	......	27	2	......	22	......	......	39	62	5
South Dakota	1	32	......	......	159	......	5	50	3	8
Virginia	7	95	140	376	1,680	29	57	72	23	358

[1] Onset previous to week of report.

PLAGUE.

San Francisco, Calif.

A case of bubonic plague was reported at San Francisco, Calif., August 27, 1923. The patient was taken ill August 18, 1923. She had been in San Francisco only three days prior to the onset of the disease, and previously resided in Pacific Grove, Monterey County. The diagnosis has been confirmed bacteriologically.

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923.

CEREBROSPINAL MENINGITIS.

The column headed "Median for previous years" gives the median number of cases reported during the corresponding week of the years 1915 to 1922, inclusive. In instances in which data for the full eight years are incomplete, the median is that for the number of years for which information is available.

City.	Median for previous years.	Week ended Aug. 18, 1923. Cases.	Week ended Aug. 18, 1923. Deaths.
California:			
Pasadena	0	1	
San Francisco	0	1	1
Illinois:			
Chicago	1	2	2
East St. Louis	0	1	1
Massachusetts:			
Lynn	0	1	
Springfield	0	1	
New Jersey:			
Newark	0	2	
New York:			
Albany	0	1	
New York—Continued.			
Buffalo	0	1	
New York	5		2
Ohio:			
Canton	0		1
Pennsylvania:			
Allentown	0	1	
Johnstown	0	1	
Texas:			
Dallas	0		1
West Virginia:			
Huntington	0		1
Wheeling	0	1	

DIPHTHERIA.

See p. 2099; also Current State summaries, p. 2089, and Monthly summaries by States, p. 2092.

INFLUENZA.

City.	Cases. Week ended Aug. 19, 1922.	Cases. Week ended Aug. 18, 1923.	Deaths, week ended Aug. 18, 1923.
California:			
Los Angeles	18	3	
San Francisco	2		
Santa Ana	1		
Stockton			1
Connecticut:			
New London		2	1
Florida:			
Tampa	3		
Illinois:			
Chicago		1	2
Kentucky:			
Louisville	1		
Maryland:			
Baltimore		1	1
Cumberland	1		
Massachusetts:			
Boston		5	
Brookline			1
Michigan:			
Detroit	1		1
Missouri:			
Kansas City	1		
New Jersey:			
Newark	5	5	
New York:			
Buffalo		1	
New York	8	4	2
Ohio:			
Cleveland		1	
Toledo			1
Youngstown			1
Pennsylvania:			
Pittsburgh		1	1

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

LEPROSY.

City.	Cases.	Deaths.
New York:		
New York	1	

LETHARGIC ENCEPHALITIS.

Nebraska:		
Omaha		1
Wisconsin:		
Milwaukee	1	

MALARIA.

City.	Cases.	Deaths.
Alabama:		
Birmingham	4	
Mobile	1	
Tuscaloosa	2	
Arkansas:		
Little Rock	6	
California:		
Los Angeles	1	
Pasadena	1	
Connecticut:		
New Britain	1	
Florida:		
St. Petersburg	1	
Tampa	1	
Georgia:		
Brunswick	3	
Macon	6	
Savannah		1
Valdosta		1
Illinois:		
Chicago	1	
Louisiana:		
New Orleans	2	
Maryland:		
Baltimore	3	
Massachusetts:		
Pittsfield	1	
New York:		
New York	17	
Tennessee:		
Memphis	21	2
Texas:		
Dallas	1	1
Virginia:		
Norfolk	1	

MEASLES.

See p. 2099; also Current State summaries, p. 2089, and Monthly summaries by States, p. 2092.

PELLAGRA.

City.	Cases.	Deaths.
Alabama:		
Birmingham	1	
Tuscaloosa	1	
Arkansas:		
Little Rock	1	
Georgia:		
Atlanta		1
Savannah		1
Louisiana:		
New Orleans	1	1
Minnesota:		
Rochester	1	1
North Carolina:		
Durham		1
Wilmington		1
Oklahoma:		
Oklahoma		1
South Carolina:		
Charleston		1
Columbia		3
Tennessee:		
Nashville		2
Texas:		
Houston		1
San Antonio		1

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

PNEUMONIA (ALL FORMS).

City.	Cases.	Deaths.
Alabama:		
Birmingham	6	1
Mobile		1
California:		
Eureka		1
Long Beach		2
Los Angeles	18	2
Oakland		3
Pasadena		1
Sacramento		3
San Francisco	4	3
Colorado:		
Denver		2
Pueblo		1
Connecticut:		
Bridgeport	2	
Hartford	1	
New Haven		4
Norwalk		1
District of Columbia:		
Washington		7
Georgia:		
Atlanta		4
Augusta		3
Illinois:		
Aurora		1
Bloomington		1
Chicago	57	27
East St. Louis		2
Freeport		1
Galesburg		1
Jacksonville		1
Kewanee		1
Quincy	1	
Springfield	4	2
Indiana:		
East Chicago		1
Hammond		1
Indianapolis		3
Kokomo		1
Logansport		1
Michigan City		1
Muncie		1
South Bend		1
Iowa:		
Burlington	1	
Muscatine		1
Kansas:		
Wichita		3
Kentucky:		
Covington		1
Louisville		6
Louisiana:		
New Orleans		5
Maine:		
Biddeford	1	
Portland	1	
Maryland:		
Baltimore	14	10
Cumberland		2
Massachusetts:		
Arlington	1	
Boston	9	2
Chelsea		1
Fall River		3
Lowell		4
Lynn	1	
Medford		1
New Bedford		6
Salem		2
Springfield	5	3
Michigan:		
Detroit		20
Grand Rapids	2	1
Hamtramck		2
Highland Park	2	
Muskegon		1
Minnesota:		
Duluth		1
Minneapolis		1
St. Paul		4
Missouri:		
Kansas City		4
St. Joseph		3
Montana:		
Great Falls		1
Helena		1
Nebraska:		
Omaha		3
New Jersey:		
Atlantic City		2
Hoboken		1
Newark	13	6
Phillipsburg		1
Trenton		3
New York:		
Albany	1	
Buffalo		3
Cohoes		1
Hudson		1
Lackawanna		1
New York	105	70
Rochester	6	5
Rome		1
Schenectady		1
Syracuse		2
North Carolina:		
Winston-Salem		1
Ohio:		
Akron	1	
Canton		1
Cleveland	14	12
Columbus		2
Dayton	1	
Salem		2
Toledo		3
Youngstown		1
Oklahoma:		
Oklahoma		2
Oregon:		
Portland		4
Pennsylvania:		
Philadelphia	34	15
Pittsburgh		17
Rhode Island:		
Cranston		1
Pawtucket		1
Providence		2
South Carolina:		
Columbia		1
Greenville		1
Tennessee:		
Memphis		1
Nashville		3
Texas:		
Dallas		1
Galveston		2
Houston		2
San Antonio		2
Utah:		
Salt Lake City		1
Virginia:		
Norfolk		2
Petersburg		1
Richmond		3
Roanoke		1
West Virginia:		
Charleston		2
Wheeling		1
Wisconsin:		
Racine		1
Superior		1

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

POLIOMYELITIS (INFANTILE PARALYSIS).

The column headed "Median for previous years" gives the median number of cases reported during the corresponding week of the years 1915 to 1922, inclusive. In instances in which data for the full eight years are incomplete, the median is that for the number of years for which information is available.

City.	Median for previous years.	Week ended Aug. 18, 1923.		City.	Median for previous years.	Week ended Aug. 18, 1923.	
		Cases.	Deaths.			Cases.	Deaths.
Alabama:				New Jersey:			
Birmingham	0	1	1	Bayonne	0	2	
California:				Clifton	0	1	
Los Angeles	0	1		Jersey City	0	1	
Pasadena	0	1		Newark	0	1	
Connecticut:				New York:			
Waterbury	0	1		Albany	0	1	
Illinois:				New York	8	30	3
Chicago	3	3	1	Newburgh	0	2	
Oak Park	0	1		Ohio:			
Kansas:				Cleveland	0	1	
Topeka	0	12	1	Oklahoma:			
Massachusetts:				Tulsa	0	1	
Fall River	0	1		Pennsylvania:			
Northampton	0	1		Wilkes-Barre	0	2	
Michigan:				Tennessee:			
Detroit	0		1	Memphis	0	2	
Minnesota:				Wisconsin:			
Duluth	0	2		Milwaukee	0	3	
Nebraska:							
Omaha	0	9					

RABIES IN ANIMALS.

City.	Cases.	City.	Cases.
California:		New Jersey:	
Los Angeles	13	Bloomfield	2
Oakland	1	Clifton	1
Pasadena	2	West Orange	2
Kentucky:		Tennessee:	
Owensboro	1	Memphis	1
Massachusetts:		Texas:	
Arlington	2	Dallas	1

SCARLET FEVER.

See p. 2099; also Current State summaries, p. 2089, and Monthly summaries by States p. 2092.

SMALLPOX.

The column headed "Median for previous years" gives the median number of cases reported during the corresponding week of the years 1915 to 1922, inclusive. In instances in which data for the full eight years are incomplete, the median is that for the number of years for which information is available.

City.	Median for previous years.	Week ended Aug. 18, 1923.		City.	Median for previous years.	Week ended Aug. 18, 1923.	
		Cases	Deaths.			Cases.	Deaths.
Alabama:				Michigan:			
Mobile	0	1		Detroit	1	5	
California:				Flint	0	1	
Los Angeles	0	2		Jackson	0	1	
Georgia:				Pontiac	0	2	
Atlanta	0	3		Minnesota:			
Indiana:				Duluth	0	3	
Gary	0	2		Nebraska:			
Kentucky:				Omaha	2	1	
Louisville	0	1					

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

SMALLPOX—Continued.

City.	Median for previous years.	Week ended Aug. 18, 1923. Cases.	Deaths.	City.	Median for previous years.	Week ended Aug. 18, 1923. Cases.	Deaths.
New York:				Oregon:			
Buffalo	0	1		Portland	4	3	
Niagara Falls	0	9		Vermont:			
North Carolina:				Burlington	0	2	
Winston-Salem	0	7		Washington:			
Ohio:				Everett	0	3	
Middletown	0	1		Spokane	3	4	
Toledo	0	7		Vancouver	0	1	
Zanesville	0	1		Wisconsin:			
Oklahoma:				Kenosha	0	2	
Tulsa	0	1		Milwaukee	1	1	

TETANUS.

City.	Cases.	Deaths.	City.	Cases.	Deaths.
Alabama:			Michigan:		
Birmingham	1	1	Detroit		1
California:			Missouri:		
Los Angeles		1	St. Louis	1	1
Connecticut:			New Jersey:		
New Haven	1		Trenton		2
Georgia:			Ohio:		
Valdosta		1	Cleveland		1
Illinois:			South Carolina:		
Chicago	2	2	Charleston		1
Kentucky:			Tennessee:		
Lexington	1		Memphis	1	
Massachusetts:			Texas:		
Lawrence	1	1	Houston		1

TUBERCULOSIS.

See p. 2099; also Current State summaries, p. 2089.

TYPHOID FEVER.

The column headed "Median for previous years" gives the median number of cases reported during the corresponding week of the years 1915 to 1922, inclusive. In instances in which data for the full eight years are incomplete, the median is that for the number of years for which information is available.

City.	Median for previous years.	Week ended Aug. 18, 1923. Cases.	Deaths.	City.	Median for previous years.	Week ended Aug. 18, 1923. Cases.	Deaths.
Alabama:				Connecticut—Continued.			
Birmingham	10	13	1	New Haven	2	5	1
Mobile	1	4		New London	0	1	1
Arkansas:				District of Columbia:			
Little Rock	3	5		Washington	10	8	2
California:				Georgia:			
Long Beach	0	2		Atlanta	5	3	2
Los Angeles	4	12	1	Macon	1	2	
Oakland	2	2		Rome	1	2	
Pasadena	0	1		Savannah	3	1	
Sacramento	1	2	1	Valdosta	2	1	
San Bernardino	0	1		Illinois:			
San Diego	0	1	1	Chicago	9	2	3
San Francisco	3	4		Galesburg	0	1	
Colorado:				Kewanee	0	2	
Denver	4	3		Quincy	0	1	
Connecticut:				Indiana:			
Hartford	1	1	1	Anderson	0		1

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

TYPHOID FEVER—Continued.

City.	Median for previous years.	Week ended Aug. 18, 1923. Cases.	Deaths.
Indiana—Continued.			
Ft. Wayne	0		1
Hammond	0	1	
Indianapolis	3	1	
South Bend	0	3	
Kansas:			
Atchison	0	1	
Coffeyville	2	1	
Kansas City	1	1	
Lawrence	0	2	
Leavenworth	0	1	
Parsons	0	1	
Topeka	2	1	
Wichita	6	1	
Kentucky:			
Covington	0		1
Louisville	8	7	1
Owensboro	1	4	
Louisiana:			
New Orleans	7	4	
Maine:			
Portland	0	2	
Maryland:			
Baltimore	14	19	1
Cumberland	2	2	1
Frederick		1	
Massachusetts:			
Boston	4	7	
Brockton	0	1	
Cambridge	0		1
Everett	0	1	
Fall River	4	1	
Holyoke	0	1	
Lawrence	1	2	
Lynn	0	1	
Quincy	0	1	
Michigan:			
Detroit	9	1	
Highland Park	0	1	
Kalamazoo	0	1	
Minnesota:			
Duluth	1	1	
Mankato	0	1	
Minneapolis	3	1	
St. Paul	1		1
Missouri:			
Cape Girardeau	1		1
Kansas City	5	3	
St. Louis	10	4	1
Nevada:			
Reno	0	1	
New Jersey:			
Elizabeth	0	2	
Hoboken	0		1
Newark	3	1	
Summit	0	1	
Trenton	1	1	
West Hoboken	0	1	1
West Orange	0	1	
New York:			
Albany	2	3	
Buffalo	4	4	1
Elmira	1	21	
Hornell	0	1	
New York	41	30	6
Niagara Falls	0	2	
Rochester	0	1	
North Carolina:			
Greensboro	0	2	1
Raleigh	1	1	
Wilmington	1	2	
Winston-Salem	5	5	
Ohio:			
Chillicothe	0	1	
Cincinnati	4	7	
Cleveland	6	2	1
Columbus	2	7	1
Dayton	2	2	
New Philadelphia	0	3	
Toledo	3	1	2
Oklahoma:			
Oklahoma	1	2	
Tulsa	4	3	
Oregon:			
Portland	1	1	
Pennsylvania:			
Braddock	0	2	
Chester	0	2	
Erie	1	2	
Harrisburg	0	1	
Johnstown	1	1	
Mount Carmel	0	1	
Philadelphia	18	10	1
Pittsburgh	4		1
Washington	2	1	
Wilkes-Barre	0	2	
Rhode Island:			
Newport	0	1	
Providence	1	1	
South Carolina:			
Charleston	1	6	1
Columbia	2		1
Tennessee:			
Memphis	4	14	3
Nashville	8	4	
Texas:			
Dallas	5	3	2
El Paso	1	2	
Houston	0	2	
San Antonio		2	1
Utah:			
Salt Lake City	1	2	
Virginia:			
Charlottesville	0	1	
Lynchburg	3	1	
Norfolk	3	1	
Petersburg	0	4	
Richmond	3	3	
Washington:			
Everett	0	24	
Seattle	1	1	
Spokane	1	1	
Tacoma	1	2	
Walla Walla	8	1	
West Virginia:			
Charleston	5	5	4
Clarksburg	1		1
Wheeling	1	1	
Wisconsin:			
Marinette	0	1	
Milwaukee	1	3	
Wausau	0	4	

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS.

City.	Population Jan. 1, 1920.	Total deaths from all causes.	Diphtheria.		Measles.		Scarlet fever.		Tuberculosis.	
			Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Alabama:										
Birmingham	178,806	64	2		2		1		8	4
Dothan	10,034	0	1		1				1	
Mobile	60,777	21								1
Tuscaloosa	11,996		1						1	
Arkansas:										
Little Rock	65,142		3						4	
California:										
Bakersfield	18,638	5			1					1
Eureka	12,923	10	1		1					
Glendale	13,536	10								
Long Beach	55,593	17	2				1		2	1
Los Angeles	576,673	157	46	1	11		5	18	39	
Oakland	216,261	52	7				1		3	3
Pasadena	45,354	15					1		2	1
Richmond	16,843		1				2			
Riverside	19,341	4	2		1					
Sacramento	65,903	21	1	2	1		1		2	2
San Bernardino	18,721	9								2
San Diego	74,683	38	1		2		2		7	4
San Francisco	506,676	121	32	3	88		6		28	7
Santa Ana	15,485	6							1	
Santa Barbara	19,441	5								
Santa Cruz	10,917	4							1	
Stockton	40,296	9								
Vallejo	21,107	1								
Colorado:										
Denver	256,491	68	9	1	2		6			14
Pueblo	43,050	12	5				3			
Trinidad	10,906				1					
Connecticut:										
Bridgeport	143,555	28	6				3		4	7
Hartford	138,036	37	4						4	
Manchester (town)	18,370	3								
Milford (town)	10,193	2								
New Britain	59,316	5	2				1			
New Haven	162,537	28	2				1		6	
New London	25,688	6							2	1
Norwalk	27,743	8								1
Waterbury	91,715	20	3		1	1	5		1	
District of Columbia:										
Washington	437,571	118			2		1		22	11
Florida:										
Key West	18,749	4								
St. Petersburg	14,237	2								
Tampa	51,603	18	1						1	1
Georgia:										
Albany	11,555				1					
Atlanta	200,616	64	1		5		1		1	5
Augusta	52,548	27								1
Brunswick	14,413	6								
Macon	52,995		1						3	
Rome	13,252		3						2	
Savannah	83,252	30							4	3
Valdosta	10,783	4							1	
Idaho:										
Boise	21,393	2								
Illinois:										
Alton	24,682	5	1							
Aurora	36,397	12							4	1
Bloomington	28,725	7			2					
Blue Island	11,424	4	1	1	1					
Chicago	2,701,705	537	55	3	22	1	25		174	36
Cicero	44,995	4								
East St. Louis	66,767	19								
Elgin	27,454	5	1				1			1
Forest Park	10,768		1							
Freeport	19,669	8		1	1				1	1
Galesburg	23,834	7			1					
Jacksonville	15,713	13							1	1
Kewanee	16,026	7							1	
La Salle	13,050	3								
Oak Park	39,858	9			1				2	
Quincy	35,978	5							2	

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

City.	Population Jan. 1, 1920.	Total deaths from all causes.	Diphtheria.		Measles.		Scarlet fever.		Tuberculosis.	
			Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Illinois—Continued.										
Rock Island	35,177	4	1				1			
Rockford	65,651	15								1
Springfield	59,183	17							10	1
Indiana:										
Anderson	29,767	3	1				1			
Bloomington	11,595	5			1					1
Crawfordsville	10,139	2								
East Chicago	35,967	10							1	
Elwood	10,790	1								
Fort Wayne	86,549	15								2
Frankfort	11,585	3			1					
Gary	55,378	15					1			
Hammond	36,004	8	1		1		1			
Huntington	14,000	0								
Indianapolis	314,194	79	5	1	10		2		3	1
Kokomo	30,067	6								
La Fayette	22,486	15								
Logansport	21,626	4								
Michigan City	19,457	10								
Muncie	36,524	7			2					1
Newcastle	14,458	4								1
South Bend	70,983	9	1				2			1
Iowa:										
Burlington	24,057	3								
Muscatine	16,068	5								
Sioux City	71,227	0	2		2		1			
Kansas:										
Atchison	12,630		3							
Coffeyville	13,452	7								
Hutchinson	23,298		1		1					
Kansas City	101,177				2				3	
Lawrence	12,456	7								
Leavenworth	16,912	4			1					
Parsons	16,028						1			
Topeka	50,022	15			3				1	1
Witchita	72,217	20	3		2					
Kentucky:										
Covington	57,121	17			1					
Henderson	12,169	3								
Lexington	41,534	17	2		5					2
Louisville	234,891	75	1	1	4				12	4
Owensboro	17,424								1	
Paducah	24,735		1							
Louisiana:										
New Orleans	387,219	117	4						18	11
Maine:										
Auburn	16,985	4					1		1	
Bangor	25,978				2					
Bath	14,731	2								
Biddeford	18,008	4	1		1					
Lewiston	31,791	13								3
Portland	69,272	22	2	1	2		1			2
Sanford (town)	10,691	1								
Waterville	13,351		1							
Maryland:										
Baltimore	733,826	193	16	2	10	1	13		40	14
Cumberland	29,837	3					1			
Frederick	11,066	3	2							
Massachusetts:										
Amesbury (town)	10,036	0								
Arlington (town)	18,665	0	1							
Attleboro	19,731	1							1	
Beverly	22,561	1	3						1	
Boston	748,060	176	69	6	23		13		56	15
Braintree (town)	10,580	8							1	3
Brockton	66,254	6	2				1			1
Brookline	37,748	5			1					
Cambridge	109,694	29	6		1		1		3	6
Chelsea	43,184	8							1	1
Chicopee	36,214	5	1							
Clinton	12,979	2								
Danvers	11,108				1				2	
Dedham	10,792	1								

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

City.	Population Jan. 1, 1920.	Total deaths from all causes.	Diphtheria.		Measles.		Scarlet fever.		Tuberculosis.	
			Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Massachusetts—Continued.										
Easthampton	11,261		2				2			
Everett	49,120	5	2				1		1	1
Fall River	120,485	40	4	1			4	1	3	
Fitchburg	41,029	7	1		1				2	
Framingham	17,033	6			2		1			
Gardner	16,971	8		1					1	2
Greenfield	15,462	5			2				1	1
Haverhill	53,884	3			3		2			
Holyoke	60,203	10	5				1		3	1
Lawrence	94,270	19	2	1	4				4	2
Leominster	19,744	3			1		1			
Lowell	112,759	35			2		6	1	1	1
Lynn	99,148	18	1	1	1				1	
Malden	49,103	8					2			
Medford	39,038	5	2		1		1		1	
Methuen	15,189	3							2	
New Bedford	121,217	32	3						4	2
Newburyport	15,618	6								
Newton	46,054	7	3		1		1		4	
North Adams	22,282	4	1							
Northampton	21,951	4								
Pittsfield	41,763	9	2				3		5	2
Plymouth	13,045	3								
Quincy	47,876	5	2				2		3	
Salem	42,529	4	3		1		4			2
Saugus	10,874	0	1		1				1	
Somerville	93,091	10	1	1			2		2	
Southbridge	14,245	1								
Springfield	129,614	26	2				3		1	1
Taunton	37,137	10			2					1
Wakefield	13,025	1								
Waltham	30,915	8	1	1					2	2
Watertown	21,457	5	1							
West Springfield	13,443	1								
Westfield	18,604	1					1			
Winthrop	15,455	3	1		1					
Woburn	16,574	4								
Worcester	179,754	33	4				2		4	2
Michigan:										
Alpena	11,101		1		2					
Ann Arbor	19,516	14	1							
Battle Creek	36,164		3							
Benton Harbor	12,233	1	1				1			
Detroit	993,678	216	24	2	14		22	1	73	21
Flint	91,599	37	17	3	9		3			1
Grand Rapids	137,634	28	4	1	5		4		3	1
Hamtramck	48,615	12	1							
Highland Park	46,499	8			1					
Holland	12,188				2					
Jackson	48,374	11			2		3		1	1
Kalamazoo	48,487	9	8	1	4		1			
Marquette	12,718	4	8	2						
Muskegon	36,570	6	3				1			1
Pontiac	34,273	7	4		1		1			1
Sault Ste. Marie	12,096	4								1
Minnesota:										
Duluth	98,917	23						1		2
Hibbing	15,089	3					14			
Minneapolis	380,582	71	12		3		16		15	1
Rochester	13,722	27			2					
St. Paul	234,698	43	5				5		16	2
Missouri:										
Cape Girardeau	10,252	9								
Kansas City	324,410	76			1		2		5	7
St. Joseph	77,939	34			1					1
St. Louis	772,897	208	10	1	1		12		40	7
Springfield	39,631	15								2
Montana:										
Anaconda	11,668	2								
Billings	15,100	3								
Great Falls	24,121	6			1		1		1	1
Helena	12,037	6								
Missoula	12,668	4								

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CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

City.	Population Jan. 1, 1920.	Total deaths from all causes.	Diphtheria.		Measles.		Scarlet fever.		Tuberculosis.	
			Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Nebraska:										
Lincoln	54,948	7	1	1			1			
Omaha	191,601	52	14				1			3
Nevada:										
Reno	12,016	3	1				1			
New Hampshire:										
Concord	22,167	7					1			1
Dover	13,029	1								
Keene	11,210	1								
New Jersey:										
Asbury Park	12,400	5								
Atlantic City	50,707	12	2						2	
Bayonne	76,754								3	
Bloomfield	22,019	2								
Clifton	26,470	2							3	
Elizabeth	95,783		10				1		1	2
Englewood	11,627	4								1
Garfield	19,381		1							
Hoboken	68,166	12		1						
Jersey City	298,103		7				1		9	
Kearny	26,724	2							2	
Morristown	12,548	4								
Newark	414,524	82	9		8				24	8
Orange	33,268	2	1						4	
Passaic	63,841	6	2				1		1	
Paterson	135,875		1		1		1		2	
Perth Amboy	41,707	5							2	1
Phillipsburg	16,923	4	1							1
Plainfield	27,700							3	1	2
Trenton	119,289	32	3		2		2		2	1
Union (town)	20,651								1	
West Hoboken	40,074	2								
West New York	29,926	1								
West Orange	15,573	1								
New Mexico:										
Albuquerque	15,157	4							2	1
New York:										
Albany	113,344		3		3		6		4	
Amsterdam	33,524	15	1							
Buffalo	506,775	100	4	1	1		3		21	6
Cohoes	22,987	4			3		1		1	
Elmira	45,393	17	1		2					
Hornell	15,025	6					1			
Hudson	11,745	4							1	
Ithaca	17,004	1			3					
Lackawanna	17,918	6			8		3		2	2
Lockport	21,308	8							2	
New York	5,620,048	1,058	82	4	68		20		[1] 171	[1] 92
Newburgh	30,366	10	2							
Niagara Falls	50,760	11					2			
North Tonawanda	15,482	2			5		4			1
Olean	20,506	4					1			
Peekskill	15,868	5						1		
Poughkeepsie	35,000	5							2	1
Rochester	295,750	53	2	1			1		10	6
Rome	26,341	12			2					
Saratoga Springs	13,181	5								
Schenectady	88,723	17	7		11		2			1
Syracuse	171,717	48	2		3		9		5	3
White Plains	21,031	8			2					
North Carolina:										
Durham	21,719	4	1							
Greensboro	43,525	13	1				1			
Raleigh	24,418	3								
Rocky Mount	12,742	4								
Wilmington	33,372	14			2					
Winston-Salem	48,395	13	5		15				4	2
North Dakota:										
Fargo	21,961	0			1		1			
Grand Forks	14,010						2			
Ohio:										
Akron	208,435	22	3				4		1	
Ashtabula	22,082	5	1				3			

[1] Pulmonary only.

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

City.	Population Jan. 1, 1920.	Total deaths from all causes.	Diphtheria.		Measles.		Scarlet fever.		Tuberculosis.	
			Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Ohio—Continued.										
Barberton	18,811	2					1			
Bucyrus	10,425	3	2				1		1	
Cambridge	13,104	1					1			
Canton	87,091	17	4		1				1	
Chillicothe	15,831	4								
Cincinnati	401,247	103	3		6		1		35	12
Cleveland	796,841	184	26	2	17		10		45	29
Cleveland Heights	15,236						1			
Columbus	237,031	57	4				1		9	4
Dayton	152,559	40	1				4		1	
East Cleveland	27,292	3								
East Youngstown	11,237	2				1				
Findlay	17,021	1							1	1
Freemont	12,468	1					1			
Hamilton	39,675	7			1		1			
Kenmore	12,683		1							
Lancaster	14,703	5	1				2			1
Lima	41,326	7	1		1		2		5	1
Lorain	37,295		4				4		3	
Mansfield	27,824	6								2
Martins Ferry	11,634	5								
Middletown	23,594	9							3	2
Newark	26,718	8								1
Niles	13,080	1								
Norwood	24,966	3	1				1		1	1
Piqua	15,044	3							3	
Salem	10,305	5								
Sandusky	22,897	2								
Springfield	60,840	9			1					1
Steubenville	28,508	4							2	
Toledo	243,164	61	14	3	2		10		5	3
Youngstown	132,358	20	14	1	4		6			1
Zanesville	29,569	11	1				2			
Oklahoma:										
Oklahoma	91,295	20					4			
Tulsa	72,075		1							
Oregon:										
Portland	258,288	41	5		2		2		9	2
Pennsylvania:										
Allentown	73,502		4							
Altoona	60,331		3							
Bethlehem	50,358		2				3		2	
Braddock	20,879								1	
Charleroi	11,516		1							
Connellsville	13,804		1				1			
Donora	14,131		3							
Erie	93,372		3		2				6	
Farrell	15,586		4							
Greensburg	15,033		1							
Harrisburg	75,917		1				1			
Johnstown	67,327		2				2		5	
Lancaster	53,150		1				2			
McKeesport	46,781								1	
Monessen	18,179		3							
Mount Carmel	17,469								2	
New Kensington	11,987		2							
Norristown	32,319				1		1			
Oil City	21,274						1			
Philadelphia	1,823,779	418	25		6		12		77	35
Pittsburgh	588,343	142	20	3	4		6	1		13
Plymouth	16,500		2							
Punxsutawney	10,311						1			
Scranton	137,783								5	
Sharon	21,747		1							
Steelton	13,428						1			
Washington	21,480		1							
Wilkes-Barre	73,833		1							
Woodlawn	12,495								1	
Rhode Island:										
Cranston	29,407	7	1							1
Cumberland (town)	10,077	2								
East Providence (town)	21,793						2			
Newport	30,255	5								

CITY REPORTS FOR WEEK ENDED AUGUST 18, 1923—Continued.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

City.	Population Jan. 1, 1920.	Total deaths from all causes.	Diphtheria.		Measles.		Scarlet fever.		Tuberculosis.	
			Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Rhode Island—Continued.										
Pawtucket	64,248	18								3
Providence	237,595	58					3			6
South Carolina:										
Charleston	67,957	30	1		1					4
Columbia	37,524	21			1				1	
Greenville	23,127	4	1							
Tennessee:										
Memphis	162,351	73	1						6	2
Nashville	118,342	40					2		8	10
Texas:										
Amarillo	15,494	11								
Beaumont	40,422	19	2							1
Corpus Christi	10,522	4								
Dallas	158,976	44	2				2	1	2	2
El Paso	77,560	22			1				5	9
Galveston	44,255	15	2				1			
Houston	138,276	37	2						7	3
San Antonio	161,379	53								4
Waco	38,500	11								
Utah:										
Provo	10,303	1								
Salt Lake City	118,110	23	4	1	2		1		11	1
Vermont:										
Burlington	22,779	3			2					
Virginia:										
Alexandria	18,060	5								1
Charlottesville	10,688	2								
Lynchburg	20,070	3	1		1		1			
Norfolk	115,777		1		4		1		3	5
Petersburg	31,012	2			2		1		1	
Richmond	171,667	45		1	28		1		3	1
Roanoke	50,842	9	1		1				1	1
Washington:										
Bellingham	25,585		1				1			
Everett	27,644				3					
Seattle	315,312		1		3		4		32	
Spokane	104,437		7		6		7			
Tacoma	96,965		4		1		5			
Vancouver	12,637		1							
West Virginia:										
Charleston	39,608	22	2		1		3			2
Clarksburg	27,869	8			1		1			
Fairmont	17,851						1			
Huntington	50,177	17	2							2
Martinsburg	12,515		1							
Morgantown	12,127		1							
Parkersburg	20,050	4	1							
Wheeling	56,208	12					4		3	2
Wisconsin:										
Appleton	19,561	2							1	
Beloit	21,284	6					1			
Eau Claire	20,906				2					
Fond du Lac	23,427	8					1			
Green Bay	31,017		2		1		3			
Janesville	18,293	3					1			
Kenosha	40,472	3	3		4					
Madison	38,378	4	1				1			
Manitowoc	17,563		1				1			
Marinette	13,610		1							
Milwaukee	457,147		6		1		10	1	11	8
Oshkosh	33,162	7								
Racine	58,593	8					1		2	
Sheboygan	30,955	4	6				1			
Superior	39,671	7					3			
Wausau	18,661		2		1					

FOREIGN AND INSULAR.

BRAZIL.

Influenza—Mortality from Certain Diseases—Porto Alegre.

According to information received under date of July 30, 1923, epidemic influenza in mild form was prevalent at Porto Alegre and in vicinity, State of Sao Paulo, Brazil, during the first quarter of the year 1923.

During the same period, mortality from certain diseases was reported at Porto Alegre as follows: Diphtheria, 4 deaths; epidemic cerebrospinal meningitis, 11 deaths; measles, 1 death; plague, 19 deaths; tuberculosis, 15 deaths; typhoid fever, 2 deaths. Population, 210,000.

CHILE.

Influenza—Smallpox—Typhus Fever—Valparaiso.

Under date of July 31, 1923, influenza in epidemic form was reported present in the district of Valparaiso.

During the week ended July 28, 1923, smallpox and typhus fever were reported at Valparaiso as follows: *Smallpox*, 10 deaths, with 25 cases in lazaretto; *typhus fever*, 4 deaths, with 45 cases in lazaretto.

CHINA.

Cholera—Shanghai.

Under date of August 28, 1923, cholera was reported moderately prevalent at Shanghai, China.

COLOMBIA.

Yellow Fever—Bucaramanga, Department of Santander.

Yellow fever was reported present at Bucaramanga, Department of Santander, Colombia, during the two weeks ended July 29, 1923. The first appearance of the disease at Bucaramanga was noted March 12, 1923.[1]

[1] Public Health Reports, Mar. 23, 1923, p. 650, and subsequent issues.

CUBA.

Communicable Diseases—Habana—Provinces.

Communicable diseases have been notified in Cuba as follows:

Habana.

Disease.	Aug. 11-20, 1923. New cases.	Aug. 11-20, 1923. Deaths.	Remaining under treatment Aug. 20, 1923.
Chicken pox	2		2
Diphtheria	2	1	1
Leprosy			[1] 12
Malaria	39		[2] 50
Measles	2		
Paratyphoid fever	1		3
Scarlet fever			1
Typhoid fever	10	1	[3] 45

[1] From abroad, 1. [2] From the interior, 21. [3] From the interior, 24.

Provinces—July 1-10, 1923.

Province.	Chicken pox.	Diphtheria.	Malaria.	Measles.	Paratyphoid fever.	Scarlet fever.	Typhoid fever.
Camaguey			16				1
Habana		3	62	2	1	1	20
Matanzas							10
Oriente	2		82				26
Pinar del Rio					2		
Santa Clara					5		14
Total	2	3	160	2	8	1	71

CZECHOSLOVAKIA.

Communicable Diseases—April-June, 1923.

During the three month period ended June 30, 1923, communicable diseases were reported in Czechoslovakia as follows:

Disease.	Cases.	Deaths.	Provinces reporting the greatest number of cases and deaths.
Cerebrospinal meningitis	63	18	Bohemia; cases, 26; deaths, 12.
Diphtheria	725	53	Bohemia; cases, 350; deaths, 21.
Malaria	45		Russinia; cases, 27.
Scarlet fever	1,891	182	Bohemia; cases, 652; deaths, 53.
Smallpox	16	4	Bohemia; cases, 15; deaths, 4.
Trachoma	1,323		Slovakia; cases, 782.
Typhoid fever [1]	836	96	Bohemia; cases, 362; deaths, 47.
Typhus fever	132	4	Russinia; cases, 98; deaths, 1.

[1] Paratyphoid A, 1 case; paratyphoid B, 20 cases; occurring in Province of Bohemia.

Other Diseases—Anthrax—Dysentery—Rabies—April-June, 1923.

During the period under report, 12 cases of anthrax, of which 4 cases occurred in the Province of Bohemia; 122 cases of dysentery with 5 deaths, of which 65 cases with 4 deaths occurred in the Province of Bohemia; and 8 fatal cases of rabies, of which 5 occurred in Bohemia, were reported in Czechoslovakia.

EGYPT.

Status of Plague.

During the week ended July 29, 1923, 47 cases of plague were reported in Egypt, as compared with 21 cases reported during the preceding week. Of these, two cases occurred at Port Said, the remaining 45 cases being distributed in four districts, with the greatest prevalence in the district of Touk, with 41 cases, of which 40 occurred in one locality. From January 1 to August 2, 1923, a total of 1,279 cases with 630 deaths was reported.

ESTHONIA.

Communicable Diseases—June, 1923.

During the month of June, 1923, communicable diseases were reported in the Republic of Esthonia as follows:

June, 1923.

Disease.	Cases.	Remarks.
Diphtheria	16	
Measles	228	
Scarlet fever	26	
Smallpox	4	
Tuberculosis	97	
Typhoid fever	26	Paratyphus fever, two cases. Recurrent typhus, one case.

GUADELOUPE (WEST INDIES).

Smallpox (Reported as Alastrim).

Under date of August 4, 1923, smallpox (reported as alastrim) was stated to be present in epidemic form in the island of Guadeloupe. About 3,000 cases were stated to be present at Pointe à Pitre. Along the coast from Basse Terre to Pointe à Pitre smallpox was reported present in small villages and was also reported present in the back country.

HAWAII.

Plague-Infected Rats—Hamakua—Honokaa.

The finding of plague-infected rats has been reported in Hawaii as follows: August 2, 1923, one plague rat trapped on the Hamakua Mill Co. plantation; July 30, 1923, two plague-infected rats reported, one trapped, one found dead, the infection in one instance being at the Honokaa Sugar Co. mill, and in the other at the village of Honokaa.

JAMAICA.

Smallpox (Reported as Alastrim).

During the two weeks ended August 4, 1923, 46 new cases of smallpox (alastrim) were reported in the island of Jamaica. Of these, seven cases occurred in the parish of Kingston.

Typhoid Fever—Kingston and Vicinity.

During the same period there were reported at Kingston four cases of typhoid fever, occurring during the week ended August 4, and in the surrounding country, 42 cases.

MEXICO.

Plague-Infected Rat—Tampico Suburb.

The finding of a plague-infected rat was reported at Dona Cecelia, a suburb of Tampico, August 8, 1923, making a total of five plague-infected rats found at Tampico since January 1, 1923.

POLAND.

Communicable Diseases—May 13–26, 1923.

During the period May 13 to 26, 1923, communicable diseases were reported in Poland as follows:

May 13–19, 1923.

Disease.	Cases.	Deaths.	Districts with greatest number of deaths.
Cerebrospinal meningitis	10	6	Lodz.
Diphtheria	44	1	Posen.
Measles	30	3	Stanislawow.
Scarlet fever	151	16	Do.
Smallpox	453	14	Lwow.
Tuberculosis	181	225	Do.
Typhoid fever	211	14	Lodz.
Typhus fever	315	35	Lwow.
Typhus fever, recurrent	29	....	Do.
Whooping cough	106	8	Warsaw.

May 20–26, 1923.

Disease.	Cases.	Deaths.	Districts with greatest number of deaths.
Cerebrospinal meningitis	10	6	Lodz.
Diphtheria	49	7	Stanislawow.
Measles	15	1	Do.
Scarlet fever	228	29	Tarnopol.
Smallpox	497	7	Lwow.
Tuberculosis	178	215	Do.
Typhoid fever	154	8	Lodz.
Typhus fever	270	15	Wilno.
Typhus fever, recurrent	22	1	Do.
Whooping cough	46	4	Lwow.

Dysentery—May 13–26, 1923.

During the period May 13 to 26, 1923, 52 cases of dysentery were reported in Poland, occurring in the two districts.

TUNIS.

Typhoid Fever—Bizerta.

Six cases of typhoid fever were present at Bizerta, Tunis, during the 10-day period ended July 20, 1923.

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

The reports contained in the following tables must not be considered as complete or final as regards either the list of countries included or the figures for the particular countries for which reports are given.

Reports Received During Week Ended September 7, 1923.[1]

CHOLERA.

Place.	Date.	Cases.	Deaths.	Remarks.
China:				
Shanghai	Aug. 28			Reported moderately prevalent.
India				June 17-23, 1923: Cases, 3,052; deaths, 1,330.
Bombay	July 1-7	1		
Calcutta	July 8-14	42	28	
Rangoon	do	2	1	

PLAGUE.

Place.	Date.	Cases.	Deaths.	Remarks.
Brazil:				
Porto Alegre				Jan. 1-Mar. 31, 1923: Deaths, 19.
Ceylon:				
Colombo	July 8-14	7	3	Plague rats, 4.
China:				
Amoy	July 15-21		1	
Egypt				Jan. 1-Aug. 2, 1923: Cases, 1,279; deaths, 630. July 23-29, 1923: Cases, 47; urban, 2; districts, 45 occurring in 4 districts; greatest number in one district, 41.
City—				
Port Said	July 23-29	2		
Hawaii:				
Hamakua				Aug. 2, 1923: One plague rat at Hamakua Mill Co. plantation.
Honokaa				July 30, 1923: Two plague rats; Honokaa Sugar Co. mill and Honokaa village.
India				June 17-23, 1923: Cases, 238; deaths, 173.
Bombay	July 1-7	1	1	
Karachi	July 15-21	14	13	
Madras Presidency	do	67	33	
Rangoon	July 8-14	42	37	
Mexico:				
Tampico				Aug. 8, 1923: At Dona Cecelia, a suburb of Tampico, one plague-infected rat found. From Jan. 1 to Aug. 8, 1923, plague-infected rats found, 5.

SMALLPOX.

Place.	Date.	Cases.	Deaths.	Remarks.
Arabia:				
Aden	July 22-28	2		
Brazil:				
Rio Grande do Sul				Jan. 1-Mar. 31, 1923: Present, with some mortality.
Chile:				
Valparaiso	July 22-28		10	July 30: 25 cases in lazaretto.
China:				
Amoy	July 15-21			Present.
Chungking	July 8-21			Present. Stated to be endemic.
Czechoslovakia				April-June, 1923: Cases, 16; deaths, 4.
Province—				
Bohemia	Jan. 1-Mar. 31	15	4	
Esthonia	June 1-30	4		
Guadeloupe (West Indies)	July 22-Aug. 4			Present in epidemic form. (Reported as alastrim.)
Pointe à Pitre	July 22-Aug. 4			Estimated from 2,000 to 3,000 cases.
India	June 17-23			June 10-23, 1923: Cases, 1,872; deaths, 625.
Bombay	July 1-7	11	8	
Calcutta	July 1-14	6	6	
Karachi	July 15-21	4		
Madras	do	2	2	
Rangoon	July 8-14	4	4	
Jamaica				July 22-Aug. 4, 1923: Cases, 46.
Kingston	July 22-Aug. 4	7		Parish.
Mexico:				
Guadalajara	Aug. 12-18		1	
Poland				May 13-26, 1923: Cases, 950; deaths, 21.

[1] From medical officers of the Public Health Service, American consuls, and other sources.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received During Week Ended September 7, 1923—Continued.

SMALLPOX—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Spain:				
Seville	July 19–25		1	
Valencia	July 28–Aug. 4	4		
Union of South Africa:				
Cape Province	June 24–30			Outbreaks.
Do	July 1–7			Do.
East London	July 8–14	1		
Orange Free State	June 24–30			Do.
Do	July 1–7			Do.
Yugoslavia				July 1–7, 1923: Cases, 8; deaths, 1.
Province—				
Bosnia-Herzegovina	July 1–7	1		
Croatia-Slavonia	do	4	1	
Serbia	do	2	1	
Belgrade	July 8–14		1	
Woiwodina	July 1–7	1		

TYPHUS FEVER.

Place.	Date.	Cases.	Deaths.	Remarks.
Chile:				
Valparaiso	July 22–28		4	July 30: 45 cases in lazaretto.
Czechoslovakia				Apr. 1–June 30, 1923: Cases, 132; deaths, 4. Paratyphoid A, 1; paratyphoid B, 20.
Province—				
Bohemia	Apr. 1–June 30	8		
Moravia	do	2		
Russinia	do	98	1	
Silesia	do	1	1	
Slovakia	do	23	2	
Esthonia				June 1–30, 1923: Recurrent typhus, one case; paratyphus, two cases.
Greece:				
Piræus	June 1–30	3		
Do	July 1–10	3		
Poland				May 13–26, 1923: Cases, 585; deaths, 50. Recurrent typhus: Cases, 51; deaths, 1.
Union of South Africa:				
Cape Province	June 24–30			Outbreaks.
Do	July 1–7			Do.
Yugoslavia				July 1–7, 1923: Cases, 4.
Province—				
Bosnia-Herzegovina	July 1–7	4		

YELLOW FEVER.

Place.	Date.	Cases.	Deaths.	Remarks.
Colombia:				
Bucaramanga	July 16–29			Present.

Reports Received from June 30 to August 31, 1923.[1]

CHOLERA.

Place.	Date.	Cases.	Deaths.	Remarks.
India				Apr. 15–June 16, 1923: Cases, 15,445; deaths, 12,765.
Bombay	June 3–30	34	23	
Calcutta	May 6–June 30	371	300	
Madras	June 3–30	2		
Do	July 1–7	1		
Rangoon	May 13–June 30	18	15	
Do	July 1–7	2	2	
Indo-China				Oct. 1–31, 1922: Cases, 92; deaths, 53. Preceding month: Cases: 24; deaths, 14. October, 1921: Cases, 100; deaths, 61.
City—				
Saigon	May 20–June 9	11	10	
Province—				
Annam	Oct. 1–31	68	39	Preceding month: Cases, 2; deaths, 1.
Cambodge	do	2	1	Preceding month: Cases, 3.
Cochin-China	do	21	13	Preceding month: Cases. 19; deaths, 13.
Tonkin	do	1		Preceding month: No cases.

[1] From medical officers of the Public Health Service, American consuls, and other sources.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received from June 30 to August 31, 1923—Continued.

CHOLERA—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Iraq (Mesopotamia):				
Bassorah	Aug. 21			Present. Port declared infected since Aug. 6, 1923.
Philippine Islands:				
City—				
Manila	June 10-16	2	1	Death in foreign case from Chingkang, China.
Province—				
Bulacan	May 17-23	1		
Capiz	May 27-June 2	1	1	
Cebu	Apr. 8-21	1	1	
Cotobato	Apr. 8-14	1	1	
Laguna	May 6-June 9	2	1	
Mountain	Mar. 25-31	1	1	
Pangasinan	June 24-30	2	2	
Russia (Soviet)				Jan. 1-May 15, 1923: Cases, 10.
Siam:				
Bangkok	May 13-June 23	9	10	

PLAGUE.

Place.	Date.	Cases.	Deaths.	Remarks.
Australia:				
Sydney	June 30	1	1	
Azores:				
St. Michael Island	May 6-26	12	5	In one locality.
British East Africa:				
Kenya—				
Kisumu	June 10-16	2	1	
Tanganyika	May 6-June 2	3	3	Territory.
Uganda	Apr. 1-30	7	5	
Canary Islands:				
Las Palmas	June 7	1		
Ceylon:				
Colombo	May 6-June 30	18	19	Plague rats, 38.
Do	July 1-7	8	8	Plague rats, 2.
China:				
Amoy	May 13-June 25		10	
Do	July 1-14		3	
Foochow	May 27-June 23			Present.
Do	July 8-14			Reported as endemic.
Hongkong	Apr. 29-June 30	63	40	
Do	July 1-7	7	4	
Manchuria—				
Yakoshih	May 31	1	1	Station on Eastern Chinese Railway. Occuring in tarabagan (marmot) hunter. Bubonic.
Nanking	June 17-30			Rodent plague present.
Do	July 1-21			Do.
Ecuador:				
Guayaquil				May 16-June 30, 1923: Rats examined, 13,800; found infected, 39. July 1-31, 1923: Rats examined, 9,300, found infected, 15.
Do	July 1-15	2	2	
Santa Ana (Manabi)	July 16-31	3	3	
Egypt				Jan. 1-June 21, 1923: Cases, 1,051; deaths, 548. May 1-29: Cases, 345. Jan. 1-June 24, 1923: Cases, 1,069. Jan. 1-July 26, 1923: Cases, 1,241; deaths, 619.
City—				
Alexandria	Jan. 7-June 24	35	15	May 1-29, 1923: Cases, 14.
Do	July 1-22	5		
Port Said	Jan. 7-June 24	24	12	May 1-29, 1923: Cases, 13.
Do	July 1-22	15		
Suez	Mar. 2-June 15	12	7	May 1-29, 1923: Cases, 3.
Do	July 16-22	1		
Province—				
Assiout	May 1-29	64		Deaths not reported.
Benisouef	do	7		Do.
Fayoum	do	14		Do.
Garbieh	do	2		Do.
Geizeh	do	3		Do.
Girgeh	do	123		Do.
Keneh	do	22		Do.
Menoufieh	do	34		Do.
Minieh	do	46		Do.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received from June 30 to August 31, 1923—Continued.

PLAGUE—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Hawaii:				
Hamakua				Plague-infected rats: Pohakea, May 23, 1923, 1 rat; vicinity of Pacific Sugar Co. mill, June 2, 1 rat.
Honokaa				July 20, 1923: One plague rat.
India				Apr. 29-June 16, 1923: Cases, 4,955; deaths, 3,783.
Bombay	Apr. 29-June 30	503	411	
Calcutta	May 6-June 9	13	13	
Karachi	May 13-June 30	110	85	Plague rats, 5.
Do	July 1-14	23	20	
Madras Presidency	May 13-June 30	254	141	
Do	July 1-14	43	24	
Rangoon	May 6-June 30	260	229	
Do	July 1-7	34	26	
Indo-China				Oct. 1-31, 1922: Cases, 93; deaths, 89. Preceding month: 70 cases; 68 deaths.
Province—				
Annam	Oct. 1-31	15	14	Preceding month, 15 deaths.
Cambodge	do	75	75	Preceding month, 51 deaths.
Cochin China	do	3		Preceding month, 4 cases, 2 deaths.
Iraq (Mesopotamia):				
Bagdad	May 1-31	222	143	
Java:				
East Java—				
Soerabaya	Apr. 1-June 23	488	488	May 1-31, 1923: Cases, 471; deaths, 471.
Do	June 17-23	1	1	
Soerakarta				May 16, 1923: Epidemic in five districts.
Madagascar				Apr. 1-June 15, 1923: Cases, 74; deaths, 71. Bubonic, pneumonic, septicemic.
Province—				
Tananarive	Apr. 1-June 15	56	53	
Tananarive	Apr. 16-June 15	20	20	
Mauritius Island				May 4-21, 1923: 2 cases.
Port Louis	May 4	1		
Mexico:				
Tampico				Apr. 15-21, 1923: 1 plague rat.
Palestine:				
Jaffa	June 19-July 16	10	1	Bubonic and septicemic.
Peru				May 1-June 30, 1923: Cases, 111; deaths, 68.
Locality—				
Ayabaca	May 16-June 30	15	13	
Callao	May 1-June 30	5	3	
Canete	May 16-June 30	3	2	
Cerro Azul	May 1-31	3	1	
Chiclayo	May 1-June 30	9	2	
Cutervo	May 1-15	2	1	
Huancabamba	May 1 June 30	34	25	
Huara	June 1-30	2	2	
Lima (city)	May 1-31	17	8	
Lima (country)	do	7	4	
Mollendo	June 1-30	1	1	
Salaverry	May 1-June 30	11	3	
Trujillo	do	2	3	
Russia				Jan. 1-May 15, 1923: Few cases in Far East regions.
Siam:				
Bangkok	Apr. 29-June 23	27	26	
Siberia				Sporadic cases of plague reported yearly in localities vicinity of stations Matsievskaya and Borzia, Transbaikal Railway.
Haramhor	May 6	1	1	Village in zone of endemic tarabagan (marmot) plague, Transbaikal Region.
Station No. 83				Station on Transbaikal Railway. Marmot plague during recent years.
Soktu				Do.
Straits Settlements:				
Singapore	May 6-June 30	6	8	
Syria:				
Beirut	May 12-June 20	3		

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received from June 30 to August 31, 1923—Continued.

SMALLPOX.

Place.	Date.	Cases.	Deaths.	Remarks.
Algeria:				
Algiers	May 1-31	2		
Arabia:				
Aden	May 27-June 2		2	
Do	July 8-21	4		
Bolivia:				
La Paz	Apr. 1-June 30	2	3	
Brazil:				
Pernambuco	May 6-June 16	5		
Do	July 1-7	8		
Rio de Janeiro	May 13-June 23	10	2	
Do	July 15-28	8		
British East Africa:				
Kenya—				
Mombasa	May 20-26	1		From vessel from Bombay.
Tanganyika	Apr. 29-June 9	3		Territory.
Uganda—				
Entebe	Apr. 1-30	4		
Canada:				
Alberta—				
Calgary	May 27-June 2	1		Infection from Deer Lodge, Mont.
British Columbia—				
Vancouver	May 27-June 30	33	1	
Do	July 1-14	5	1	
Victoria	Aug. 5-11	1		
Manitoba—				
Winnipeg	June 3-30	1		
Do	July 1-31	1		
New Brunswick—				
Kent County	July 1-7	1		
Ontario				June 1-30, 1923: Cases, 13. July 1-31, 1923: Cases, 14.
London	July 15-21	1		
Toronto	June 24-30	3		
Do	July 15-21	1		
Quebec—				
Quebec	June 10-16	1		Varioloid.
Saskatchewan—				
Moose Jaw	July 8-14	1		
Regina	June 24-30	3		
Ceylon:				
Colombo	May 6-June 2	23	1	
Chile:				
Concepcion	May 22-June 11		3	June 1-30, 1923: Cases, 2.
Valparaiso	May 7-June 23	6	121	June 10-16, 1923: 29 cases reported from 2 districts.
Do	July 1-14	12		
China:				
Amoy	May 13-June 23		3	June 19-25, 1923; Present.
Do	July 1-14			Present.
Antung	May 14-20	1		
Chungking	May 13-June 30			Present and endemic.
Do	July 1-7			Do.
Foochow	May 13-July 14			Do.
Hongkong	Apr. 29-June 30	98	82	
Do	July 1-7	6	11	
Manchuria—				
Dairen	May 21-27	1		
Harbin	May 7-June 24	5		
Do	July 1-7	1		
Mukden	May 13-20	1		
Nanking	May 13-June 23			Present.
Do	June 24-July 7			Do.
Shanghai	May 21-June 3	4		Foreign.
Do	July 2-8	1	2	Cases, foreign; deaths, Chinese.
Chosen (Korea):				
Chemulpo	May 1-31	1		
Fusan	May 1-June 30	4		
Gensan	May 1-31	1		
Seoul	May 1-June 30	42	13	
Cuba:				
Antilla	July 8-14		2	From Preston.
Czechoslovakia				Jan.-Mar., 1923: Cases, 15.
Ecuador:				
Alausi	July 16-31	3		
Guayaquil	May 16-31	1		
Egypt:				
Cairo	Mar. 12-May 6	17	4	
Finland				May 1-15, 1923: 1 case.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received from June 30 to August 31, 1923—Continued.

SMALLPOX—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Great Britain:				
Birmingham	June 18–30	3		
Bristol	June 28			Present.
Cardiff	June 3–30	6		
Gloucester	June 28			123 cases reported in hospital; present in rural districts. July 15, 1923: Present.
Do	July 12	19		
Nottingham	June 3–9	1		May 1–31, 1923: Cases, 211.
Do	July 8–14	1		
Greece:				
Athens	May 1–31	53		
Patras	Apr. 24–June 15		19	
Saloniki	Apr. 30–May 20	2	2	
Hungary	July 15–21	6		
India				Apr. 15–June 9, 1923: Cases, 5,914; deaths, 1,718.
Bombay	Apr. 22–June 30	298	141	
Calcutta	May 13–June 9	12	9	
Do	July 1–7	4	4	
Karachi	May 13–June 30	24	8	
Do	July 1–14	3	1	
Madras	May 13–June 23	91	16	
Do	July 8–14	10	5	
Rangoon	May 6–June 30	125	67	
Do	July 1–7	16	5	
Indo-China:				
Saigon	May 20–June 23	28	20	Including 100 surrounding square kilometers.
Iraq (Mesopotamia):				
Bagdad	Apr. 1–May 31	20		
Italy:				
Turin	May 28–June 3	1		
Do	July 2–15	2		
Jamaica				May 27–June 30, 1923: Cases, 226. July 1–7, 1923: Cases, 13. (Reported as alastrim.)
Kingston	May 27–June 30	39		
Do	July 1–7	12		
Japan:				
Kobe	May 28–June 10	2		
Do	July 2–8	1		
Java:				
East Java—				
Soerabaya	Apr. 22–June 30	187	22	
West Java—				
Batavia	May 5–June 8	17	3	Province.
Do	June 30–July 7		1	City and Province.
Latvia				Apr. 1–May 31, 1923: Cases, 8.
Mexico:				
Aguascalientes	July 8–14		1	
Chihuahua	June 11–24	7		
Guadalajara	July 22–Aug. 11		6	June 1–30, 1923: Cases, 15; deaths, 2.
Mexico city	May 19–June 30	164		Including municipalities in Federal district.
Do	July 1–21	84		
Palestine:				
Jaffa	June 5–11	1		
Persia:				
Tabriz	Apr. 1–14		1	District.
Teheran	Feb. 22–June 14		30	
Poland				Apr. 29–May 12, 1923: Cases, 15; deaths, 4.
Portugal:				
Lisbon	May 20–June 30	35	3	
Do	July 1–28	18	2	
Oporto	June 10–30	6	3	
Do	July 9–15	5	4	July 8–28, 1923: Cases, 7; deaths, 2.
Portuguese West Africa:				
Angola—				
Loanda	Apr. 1–21		2	
Rhodesia (British Africa):				
Northern Rhodesia	May 8–14	21	8	
Southern Rhodesia	May 3–16	4	2	
Siam:				
Bangkok	Apr. 29–June 23	79	43	
Sierra Leone:				
Kaballa	May 1–15	1		
Pujehun	May 16–31	1		In Sembehun district.
Spain:				
Barcelona	May 31–June 6		1	
Do	June 28–July 10		2	
Valencia	May 15–June 30	44	2	
Do	July 1–21	21	4	

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received from June 30 to August 31, 1923—Continued.

SMALLPOX—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Switzerland:				
Basel	May 27–June 30	4		
Do	July 8–14	1		
Berne	May 20–June 30	11		
Do	July 1–28	10		
Lucerne	May 1–June 7	36		
Do	July 1–31	14		
Zurich	May 20–June 23	10		
Do	July 15–21	6		
Syria:				
Aleppo	July 15–21	6		
Damascus	May 15–June 11	7		
Tunis:				
Bizerta	June 10–20	1		
Tunis	June 11–17	1		
Do	June 26–July 1	1		
Turkey:				
Constantinople	May 13–June 26		45	
Do	June 27–July 10		6	
Union of South Africa				May 1–31, 1923: Cases, 33; deaths, 1 (colored).
Cape Province				May 1–31, 1923: Cases, 32 (colored).
Do	May 6–June 16			Outbreaks.
Orange Free State	Apr. 29–May 14			Do.
Transvaal				May 1–31, 1923: 1 case.
Do	May 26–June 9			Outbreaks.
Yugoslavia:				
Serbia—				
Belgrade	June 10–16	1	1	
Zagreb	June 24–30	1		
On vessels:				
S. S. Kargola	May 20–26	1		At Mombasa, British East Africa. Vessel arrived from Bombay Mar. 25, 1923.
S. S. Makura	May 26	2		Two cases in quarantine (reported as alastrim). Vessel left Victoria, B. C., Apr. 28, 1923. Touched at Honolulu.

TYPHUS FEVER.

Place.	Date.	Cases.	Deaths.	Remarks.
Algeria:				
Algiers	May 1–June 30	66	19	
Argentina:				
Rosario	May 25–31		3	
Bolivia:				
La Paz	June 1–30	4		
Bulgaria:				
Sofia	Apr. 22–June 23	11	2	Paratyphus, 2 cases, 2 deaths.
Chile:				
Concepcion	May 22–June 18		3	
Talcahuano	May 13–19	1		
Valparaiso	May 7–June 23		26	June 11, 1923: 34 cases in Salvador Hospital.
Do	July 1–21		14	
China:				
Antung	May 28–June 24	12		
Do	July 16–22	1		
Hankow	May 19–25	1		
Manchuria—				
Harbin	May 6–13	1		
Mukden	May 14–20	2		
Czechoslovakia				Jan.–Mar., 1923: Cases, 191; deaths, 6.
Egypt:				
Alexandria	May 14–June 24	7	5	
Do	June 25–July 29	5	3	
Cairo	Apr. 12–May 6	20	10	
France:				
Marseille	Mar. 1–May 31		3	
Germany:				
Coblenz	May 27–June 2		1	
Hamburg	May 20–26	3		
Königsberg	May 13–June 2	2		
Stettin	May 27–June 9	1	1	
Great Britain:				
Bootle	Aug. 4	1		Vicinity of Liverpool.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued.

Reports Received from June 30 to August 31, 1923—Continued.

TYPHUS FEVER—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Greece				May 1-31, 1923: Cases, 876.
Athens	May 1-31	150	5	
Patras	Apr. 24-June 15		30	
Piræus	May 1-31	353	11	
Saloniki	Apr. 30-June 24	56	16	Apr. 30-May 27, 1923: Recurrent typhus: Cases, 3; deaths, 3.
Guatemala:				
Guatemala City	Apr. 1-June 30		5	
Hungary				Jan. 1-May 19, 1923; Cases, 318; deaths, 36. In 11 counties.
Budapest	Jan. 1-June 2	48	12	
Iraq (Mesopotamia):				
Bagdad	Apr. 1-30	2		
Japan:				
Nagasaki	July 2-8	1		
Latvia				Apr. 1-May 31, 1923: Cases, 186. Paratyphus, 4 cases.
Mexico:				
Mexico City	May 20-June 30	75		Including municipalities in Federal district.
Do	July 1-21	27		Do.
Guadalajara	June 1-30	1		
San Luis Potosi	July 29-Aug. 4		1	
Palestine:				
Jaffa	May 22-28	2		
Do	June 26-July 9	4		Relapsing fever, 1 case.
Jerusalem	May 22-28	1		
Persia:				
Tabriz	Apr. 1-14	2		
Teheran	Feb. 22-June 14		4	
Poland				Mar. 4-Apr. 7, 1923: Cases, 2,253; deaths, 172. Recurrent typhus: Cases, 338; deaths, 6. Apr. 29-May 12, 1923: Cases, 720; deaths, 61. Recurrent typhus: Cases, 188; deaths, 1.
Portugal:				
Oporto	June 10-16	1		
Do	July 1-21	3		
Rumania:				
Kishineff	May 1-June 30	41		
Russia				Jan. 1-Apr. 30, 1923: Cases, 106,854. (Corresponding period 1922: Cases, 847,516.) Feb. 1-28, 1923: Cases, 17,577. Recurrent, Jan. 1-Feb. 28, 1923: Cases, 43,540.
European Russia and autonomous republics.	Jan. 1-Apr. 30	93,999		
Siberia, Caucasus, and Central Asia.	do	9,921		
Waterways and railways	do	2,934		
Spain:				
Barcelona	June 21-27		1	
Madrid	May 1-31		1	
Syria:				
Aleppo	May 20-June 16	4	2	
Do	July 15-21	3	1	July 8-14, 1923: Present.
Beirut	May 1-10	1		
Tunis:				
Tunis	May 28-June 24	3	2	
Do	July 9-15	1	1	
Turkey:				
Constantinople	May 13-June 26		19	
Do	June 27-July 3		1	
Union of South Africa				May 1-31, 1923: Cases, 102; deaths, 21 (colored). White—Cases, 6. Total, 108 cases, 21 deaths.
Cape Province				May 1-31, 1923: Cases, 49 (colored); white, 5.
Do	Apr. 29-June 16			Outbreaks.
Natal				May 1-31, 1923; One case (colored).
Orange Free State				May 1-31, 1923: Cases, 45 (colored).
Do	May 6-June 16			Outbreaks.
Transvaal				May 1-31, 1923: Cases, 7.
Johannesburg	May 1-June 30	4	4	
Yugoslavia:				
Croatia—				
Zagreb	May 27-June 2	1		

YELLOW FEVER.

Place.	Date.	Cases.	Deaths.	Remarks.
Brazil:				
Bahia	May 13-June 30	25	6	
Do	July 1-7		2	
Colombia:				
Bucaramanga	June 25-July 15			Present.

X