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Effect of Concentration and Reaction (pH) on the Germicidal Activity of Chloramine–T

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The Public Health Service Milk Ordnance and Code (*1*) and the corresponding Public Health Service Ordinance and Code for Restaurants (*2*), recommend the use of hot water at 170° F. for a period of 2 minutes as a satisfactory bactericidal treatment for milk or food utensils. In lieu of hot water, hypochlorite (chlorine) compounds are permitted in a minimum concentration of 50 parts per million with an exposure period of 2 minutes. Chloramines, of which the most important commercially for food utensils is chloramine–T,[1] are permitted to be used in a similar manner to hypochlorites. An exact concentration in terms of parts per million is not stipulated, but the recommendation is made that it be bactericidally equivalent to 50 parts per million of available chlorine (av. cl.) as hypochlorite. The studies reported here were designed to determine the concentration of available chlorine as chloramine–T which is bactericidally equivalent to 50 ppm av. cl. as hypochlorite.

The procedure used for these studies was a modification of that reported by Weber and Black (*3*, *4*). The modifications included: (1) Alteration of time intervals to meet the needs for very short (a few seconds) and very long (several hours) survival curves; (2) higher dilutions for plating at each time interval, up to and including 10^{-6}; (3) preparation of a survival curve by graphing logarithm of the percent of survivors against time, and determining as the killing time that required to kill 99.9999 percent of the exposed organisms; (4) preparing 8 ml. rather than 9 ml. M/320 buffered phosphate dilution blanks and aseptically adding 1 ml. of a solution of the required concentration of sodium thiosulphate for chlorine neutralization just prior to use. This was made necessary, because, in working with extremely alkaline compounds, it was necessary to add appreciable amounts of HCl to the buffered blanks to neutralize immediately the alkali so

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[1] Sodium paratoluene sulfonchloramide.

that surviving bacteria would not be killed, and in autoclaving blanks containing sodium thiosulphate in the presence of this acid, the sodium thiosulphate was decomposed.

The test organism used was *Micrococcus pyogenes* var. *aureus*. Available chlorine concentrations for both hypochlorites and chloramines were determined by use of the sodium thiosulphate (starch-iodide) titration, standardized against N/100 potassium dichromate (*5*, *6*). In these experiments only initial chlorine concentrations were determined. All chlorine solutions were made in distilled water. Residual chlorine in 9 ml. dilution blanks was periodically checked with ortho-tolidine (*5*) for possible incomplete neutralization of the chlorine. Determinations of pH were made electrometrically by means of a glass electrode. All tests reported in this study were made at 25° C. Petri dishes were poured with tryptone glucose extract agar and incubation was at 35° C. for 48 hours. Other details are described elsewhere (*3*, *4*).

Preliminary studies were made with seven chloramine–T products in order to classify the compounds. One compound representing each "class" was selected for more detailed study. Generally, each killing time reported is the arithmetic mean of two or more, in a few cases, one, determinations made from a graph of the logarithm of percent of survivors against time. Some of the compounds studied were buffered more completely than others. Determinations of pH for duplicate experiments generally did not show much variation but slight variations were averaged (arithmetically) in order to establish points on the graphs (fig. 1 and 3).

Results

The results of this study are presented in the table and figures 1, 2, and 3. The table shows that, with hypochlorites at a concentration of 50 ppm, at pH 8.9 and below, the killing time was less than 5 seconds; at pH 10 (buffered, using carbonates), 0.40 minutes; and at pH 11.2, 1.85 minutes. The number of organisms employed was about 100 million or less per milliliter and would appear to be comparable to that to be expected on a heavily contaminated food utensil (*3*).

In the case of chloramine–T with 50 ppm av. cl. at pH 6.3, the killing time was 0.78 minutes; at pH 6.4, 2.15 minutes; at pH 7.4, 11.6 minutes; at pH 8.3, 31.5 minutes; and at pH 10.5, the killing time was prolonged to 372 minutes (6.2 hours). Each of these pH levels represents a different commercial product except that the one used at pH 6.3 is the same as that at pH 10.5 with the pH lowered by the addition of dilute HCl.

At a chloramine–T concentration of 250 ppm, a similar effect of pH was noted. At pH 6.9, the killing time was slightly less than 1 minute; at pH 8.3, 15.5 minutes; and at pH 11.3, 258 minutes. Again

Effect of pH and concentration on killing time of chloramine–T, compared with hypochlorite (25° C.; Micrococcus pyogenes var. aureus)

Chlorine compound	pH	Initial concentration av. cl. ppm	Killing time 99.9999 percent
			Seconds
Hypochlorite	8. 5	50	<5
	8. 9	50	<5
			Minutes
	10. 0	50	0. 40
	11. 2	50	1. 85
Chloramine–T	6. 3	50	*0. 78
	6. 4	50	2. 15
	7. 4	50	11. 6
	8. 3	50	31. 5
	10. 5	50	372
	6. 9	250	0. 97
	8. 3	250	15. 5
	11. 3	250	258
	7. 2	500	1. 26
	8. 3	500	8. 5
	11. 5	500	141
	7. 5	1, 000	1. 6
	8. 2	1, 000	4. 4
	11. 7	1, 000	66
	8. 1	1, 500	2. 4
	8. 3	1, 500	2. 7
	11. 9	1, 500	52

* pH adjusted.

with 500 ppm available chlorine introduced as chloramine–T at pH 7.2, the killing time was 1.26 minutes; at pH 8.3, 8.5 minutes; and at

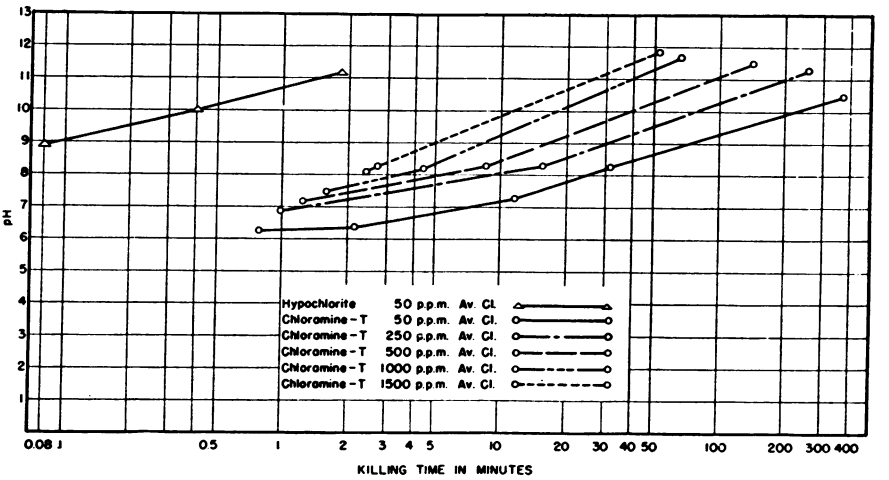

Figure 1. Effect of pH and concentration of chloramine–T on killing time (99.9999 percent) of *Micrococcus pyogenes* var. *aureus*:25° C.

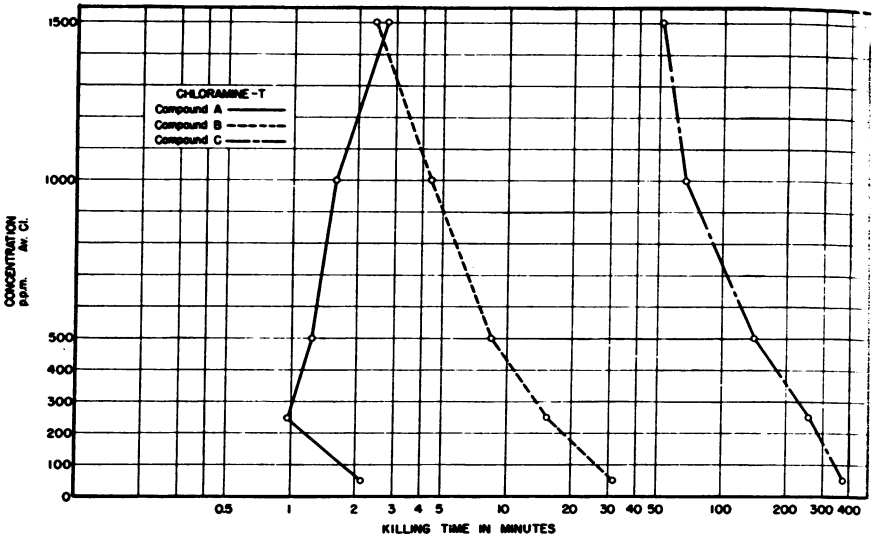

Figure 2. Effect of concentration of chloramine–T on killing time (99.9999 percent) of *Micrococcus pyogenes* var. *aureus*:25° C.

pH 11.5, 141 minutes. Similarly, when using 1,000 ppm av. cl· introduced as chloramine–T, the killing time ranged from 1.6 minutes at pH 7.5 to 66 minutes at pH 11.7. The results employing 1,500 ppm av. cl. as chloramine–T likewise show the pronounced effect of pH upon the killing time. Each of the three pH levels represents a different commercial product. It is of interest to note that the slight rise in pH from 8.1 to 8.3 appears to be reflected in a slightly increased killing time from 2.4 to 2.7 minutes. This trend is maintained as is evident at pH 11.9 with a killing time of 52 minutes.

The data from the table are presented graphically in figure 1 (semi-log) with pH on the arithmetic scale and killing time on the logarithmic scale. The logarithmic scale was employed here so that killing times from only a few seconds up to 372 minutes (6.2 hours) could be plotted on the same graph. The pronounced effect of pH on the killing time at each concentration of available chlorine introduced as chloramine–T is evident. Somewhat similar results may be observed with 50 ppm av. cl. as hypochlorite, but for a given pH the killing times are much shorter.

Figure 2 shows the effect of increased concentration of each of three commercial chloramine–T compounds on the killing time. With compound A, 50 ppm av. cl. resulted in a killing time of 2.15 minutes, and, with an increased concentration to 250 ppm av. cl., the killing time was shortened to only 0.97 minute. However, with the addition of more chloramine–T to 500, 1,000, and 1,500 ppm av. cl., the killing time was gradually increased to 1.26, 1.6, and 2.7 minutes, respectively. Paradoxically, above 250 ppm av. cl., the higher the concentration, the

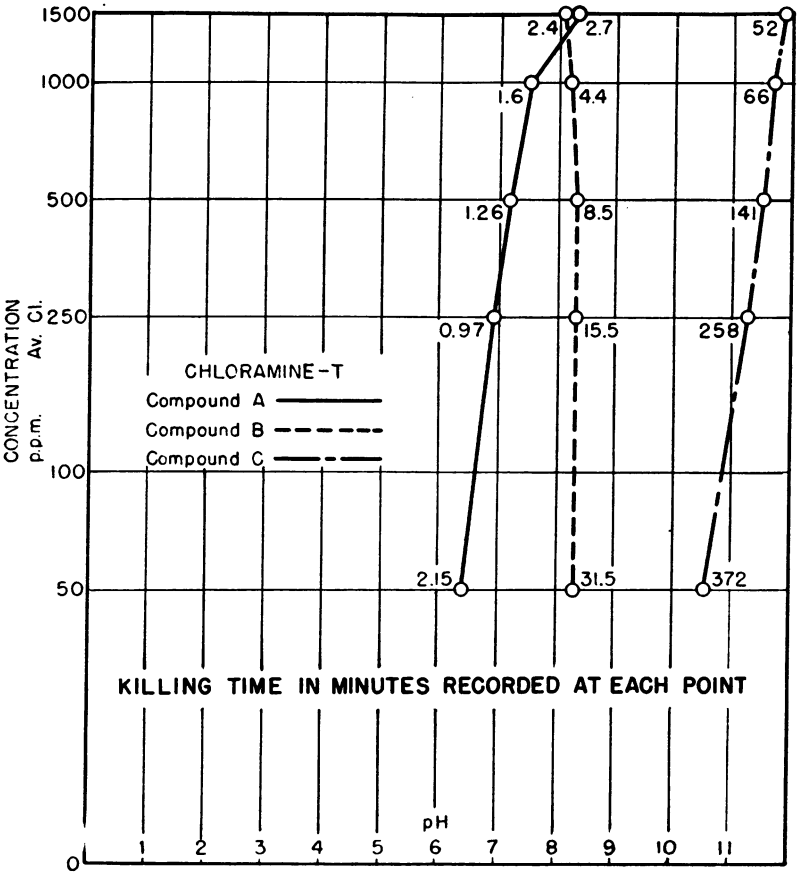

Figure 3. Effect of concentration of chloramine–T on pH:25° C. (*Micrococcus pyogenes* var. *aureus*).

longer the killing time, although even at the highest concentration used (1,500 ppm) the killing time was only slightly longer than at 50 ppm. Compound B showed a gradual reduction in killing time as the concentration was increased. At a concentration of 50 ppm av. cl., the killing time was 31.5 minutes, and at 1,500 ppm this was reduced to 2.4 minutes. Chloramine–T compound C also showed a reduction in killing time from 372 minutes with 50 ppm av. cl. to 52 minutes at a concentration of 1,500 ppm.

Figure 3 shows the effect of concentration on pH with each of the three chloramine–T compounds reported in figure 2. Killing times are reported in minutes on the graph. Compound A varies from pH 6.4 with 50 ppm av. cl. to pH 8.3 at 1,500 ppm. Compound B is rather well buffered and ranges only from pH 8.3 at 50 ppm to pH 8.1 at 1,500 ppm. With compound C, the reaction changes from pH 10.5 at 50 ppm to pH 11.85 at 1,500 ppm.

Discussion

The problem of just what concentration of available chlorine introduced as chloramine–T should be used in order to be bactericidally equivalent to 50 ppm av. cl. introduced as hypochlorite is made more difficult in that two factors exert influence on the killing time: pH and concentration of available chlorine. Generally, one might expect that by increasing the concentration of available chlorine (chloramine–T) the desired result could be attained, namely, a shortened killing time within the limits of those found for hypochlorites. This is not necessarily the case, however, especially with poorly buffered chloramine–T products. If the chloramine–T is not adequately buffered, as more of the compound is added to increase the concentration of available chlorine, the solution becomes more alkaline. This shift in pH in some cases may actually offset any shortening in killing time resulting from an increased concentration of available chlorine. This is exactly what occurred with chloramine–T compound A (fig. 2). Both pH and concentration influenced the killing time. At 50 ppm, the reaction (pH 6.4) was favorable for a rapid kill (fig. 3). At a concentration of 250 ppm, the pH was 6.9 and the killing time was 0.97 minute, more favorable than at the lower concentration. However, as the concentration of available chlorine was increased above 250 ppm, the killing time was actually increased due to the apparently greater influence of pH than concentration of available chlorine. Chloramine–T compound B (fig. 2) showed a shortened killing time with increase in concentration, and the compound behaved somewhat differently from compound A. The explanation of this becomes apparent from figure 3 where it may be noted that compound B is highly buffered and shows no appreciable change in pH from a concentration of 50 ppm to 1,500 ppm av. cl. Compound A, however, is poorly buffered, and this gradual increase in alkalinity with increasing available chlorine concentration is sufficient to have a demonstrable effect on the killing time. Chloramine–T compound C behaved somewhat like compound B but is not so well buffered and is considerably more alkaline, thus accounting for the longer killing time at each concentration studied.

The pronounced effect of pH on the germicidal activity of hypochlorites and inorganic chloramines (ammonia-chlorine combinations) has been reported by a number of investigators (*7*, *8*, *9*, *10*, *11*, *12*), but rather limited detailed studies are available on the organic chloramines such as chloramine–T (*13*, *14*).

With hypochlorites, Johns (*7*) reported in 1934 that there was no difference in germicidal activity between 25 and 10 ppm av. cl., but that 2 ppm was distinctly more active than 10 ppm. The explanation for this was believed to lie in the effect of pH on the proportion of the

total "available chlorine" existing in the form of hypochlorous acid; hypochlorous acid was believed to be the germicidally active component. In 1935 Charlton and Levine (*13*) reported that: " 'available chlorine' was not found to be a direct measure of the germicidal efficiency of the calcium hypochlorite studied. A solution containing 1,000 ppm available chlorine was only slightly more germicidal than the same solution diluted with distilled water to 100 ppm (the reaction was changed by diluting from pH 11.3 to pH 10.4) and very much less efficient than 20 ppm of the same disinfectant at a reaction of pH 8.3." Rudolph and Levine (*8*) reported in 1941 that the more dilute solutions showed greater germicidal activity. They reported that the effect on the killing time induced by change in reaction exceeds the effect of dilution. Weber and Levine (*11*) in 1944 buffered hypochlorites at various reactions to demonstrate the effect of reaction (pH) for a constant chlorine concentration, and the effect of concentration at a constant reaction. The effect of pH on the germicidal activity of inorganic chloramines was demonstrated in 1944 by Weber and Levine (*11*) employing spores and by Butterfield et al. (*12*, *15*) in 1946 using vegetative cells. Generally, as the alkalinity increases the germicidal activity is decreased, especially with vegetative cells. Some exceptions to this have been noted with spores (*11*, *16*).

In 1935, Charlton and Levine (*13*) reported results of studies employing high concentrations of chloramine–T (1,000 to 4,000 ppm av. cl.) against spores. The pH range studied was limited to 6.0 to 8.8, and it was observed that increasing the acidity markedly reduced the killing time.

For the studies reported here, the "yardstick" by which chloramine–T compounds have been measured is the killing time required for 50 ppm av. cl. introduced as hypochlorite. The table and figure 1 show that, with this concentration of hypochlorite at pH 8.5 and also at pH 8.9, the killing time was less than 5 seconds. An exact time was not determined. (Similar results were noted at pH 7.6 and 7.9.) At pH 10 the killing time was 0.40 minute, and at pH 11.2, 1.85 minutes were required. From figure 1 it is evident that in terms of speed of reaction none of the chloramine–T products is as rapid as 50 ppm av. cl. (hypochlorite) at pH 10 (or below). However, if the killing time of 1.85 minutes at pH 11.2 is taken as a "yardstick" then chloramine–T products appear to be sufficiently rapid if the pH is sufficiently low and the concentration is adequate at the given pH. It is obvious then that, as with hypochlorites, two factors must be considered in determining the use of a chloramine–T as a bactericide, (1) pH and (2) concentration. Grossly then from figure 1 it appears that chloramine–T is appreciably less rapid in germicidal action than is hypochlorite.

Some studies of chlorine compounds reported in the literature have

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been designed to demonstrate factors in addition to bactericidal activity such as the effect of organic matter and detergency (*7, 16, 17, 18*). The experiments reported here have all been carried out in the absence of organic matter to demonstrate only bactericidal activity. It is generally recognized, however, that, in the presence of organic matter, a reduction in germicidal activity will be effected. It has been demonstrated with hypochlorites that if the organic matter (at least certain types) is added slowly so that the available chlorine concentration remains considerably above that of the organic matter, the organic matter may be oxidized and the remaining hypochlorite which has not been reduced in the oxidation will remain an effective germicide (*19, 20*). Most investigations reveal that chemical germicides cannot be relied upon in the presence of more than just traces of organic matter. The surface of any food utensil should be thoroughly cleaned before applying a chemical bactericide.

Conclusions

It is evident from the results reported that the effect of reaction (pH) on the germicidal activity of chloramine–T is tremendous. Figure 1 shows that compounds with pH ranges higher than about 7.5 generally appear to be too slow to be of practicable usage in food utensil disinfection where only short periods of exposure are to prevail. Even increasing the concentration of these compounds up to as much as 30-fold (from 50 to 1,500 ppm) does not sufficiently shorten the killing time so that these chloramine–T compounds are quite equal in speed of reaction to even the more alkaline hypochlorites.

From figure 1 it appears that, if only about 250 ppm av. cl. is to be maintained, the reaction must not be more alkaline than about pH 7.0. If chloramine–T compounds more alkaline than about pH 7.0 but less alkaline than pH 7.5 are to be used, then it would appear that concentrations of available chlorine should be increased up to 500 or even 1,000 ppm av. cl. These concentrations may be economically impracticable. If short exposure periods are required, then it would seem that only chloramine–T solutions not more alkaline than pH 7.0 or 7.5, in adequate concentrations, would be sufficiently rapid in germicidal action.

Whether or not this type of germicide could be sufficiently buffered so that it would not become more alkaline and less efficient in natural alkaline waters or whether it would have sufficient detergent action should be determined. Also there is the distinct possibility that chloramine–T solutions in the range of pH 7.5 to 7.0 or more acidic would be too corrosive (*21*) for metals to be of any practical value. If greater detergency action is needed, alkaline solutions would appear to offer some advantage; however, the exposure times required would be

much greater. The possibility of combining synthetic neutral compatible detergents with neutral chloramine–T compounds could be considered, but the possibility of metallic corrosion must not be overlooked.

If chloramine–T compounds are to be employed for bactericidal treatment of food and milk equipment, the pH, in addition to concentration of available chlorine, should be determined and from this information an adequate exposure period provided. Generally, this exposure period should be considerably greater than for hypochlorites under similar conditions. Although chloramine–T compounds would appear to have only limited usage where rapid germicidal action is required, they may be the germicide of choice under certain special conditions where long exposure periods are practicable.

Summary

A study has been made to determine the concentration of available chlorine introduced as chloramine–T which is bactericidally equivalent to 50 ppm av. cl. introduced as hypochlorite, as prescribed in the Milk Ordinance and Code and also the Ordinance and Code Regulating Eating and Drinking Establishments as recommended by the Public Health Service. The pH as well as the concentration of available chlorine should be determined in order to prescribe exposure periods for germicidal treatment of utensils with chloramine–T. Generally, considerably longer exposure periods are required for chloramine–T than are necessary for hypochlorite. Concentrations of chloramine–T of at least 250 ppm at a reaction not greater than about pH 7.0, or 500 to 1,000 ppm at reactions not more alkaline than pH 7.5, would appear to be as rapid in germicidal action (in the absence of organic matter) as 50 ppm of the slower (alkaline) hypochlorites. Commercial chloramine–T products generally are not adjusted to such a low pH, and there is some doubt as to the feasibility of using a chlorine germicide of such low pH.

ACKNOWLEDGMENT

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Successful Two-stage Resection of Carcinoma of the Upper Thoracic Esophagus with Reanastomosis

By H. L. Skinner, M. D., and Milton S. Lloyd, M. D.*

Cancer of the esophagus has long tested the resources of surgeons, and it has only been in the past decade that any significant success in the treatment of this disease has been noted. Important contributions in this field have been made by Adams and Phemister (*1*), Eggers (*6*), Churchill and Sweet (*4*), Garlock (*7, 8*), Wookey (*16*), DeBakey and Ochsner (*5*). Satisfactory results with esophago-gastric procedures in lesions from the arch of the aorta and downward have been reported, and the stomach has been carried higher and higher until anastomosis has been performed in the neck.

Surgical excision of carcinoma of the upper thoracic esophagus and cervical esophagus presents a complex technical problem because of the difficulty in providing a suitable replacement for the resected esophagus when extensive excision is necessary. It is also noted that patients who have had a large portion of their stomachs brought up into the chest in esophago-gastric surgery are not entirely free of symptoms. In this type of surgery where the lower two-thirds of the esophagus is completely dissected, it has been observed that the color of the esophagus and its integrity seem to remain good for a long period of time. Also, because it is not necessary as a rule to tie off any vessels of consequence in dissecting the lower two-thirds of the esophagus, it was felt that if the esophagus was not transected at the time of operation, it would be possible to utilize the lower portion of the esophagus for reanastomosis in the cervical region for lesions above the arch of the aorta, and also in the cervical area.

In May 1949, a case of carcinoma of the esophagus just above the arch of the aorta was referred to one of the authors, Lloyd, who suggested that this should be a suitable operable case for this type of procedure. This report gives an account of surgical problems encountered in a successful two-stage resection of carcinoma of the upper third of the esophagus and subsequent end-to-end anastomosis in the cervical region.

Since thoracic surgery has come within the realms of elective surgery, two schools have developed. One of them, influenced by the high infection risk, attempted mobilization of the esophagus to convert the operation into an extrathoracic procedure, while the other attempted suture in situ by various methods. Many workers have concerned themselves with resection and suture of the esophagus. Successful intrathoracic suture of the resected esophagus has been

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reported as far back as 1901. Sauerbruch felt that the procedure of choice was the use of the Murphy button. Omi, in 1913, reported on nine experimental end-to-end sutures of the thoracic esophagus with seven cases successful. He used three layers of silk sutures, the first in the mucosa, the second in the muscle and adventitia, the third layer being an external continuous Lembert. Apparently, the failures were due to difficulty in avoiding a fatal degree of tension on the suture line. In 1923, Miller and Andrus (*10*), working on animals, demonstrated that the lower portion of the esophagus could be freed and that it was developed enough so that a successful anastomosis could be performed to the stomach as is the procedure today in surgery of the lower part of the esophagus. In recent years there have been reports in the literature on resection of short lesions in the esophagus with end-to-end esophageal reconstruction and replacement in the posterior mediastinum. Experimental work has also been done showing that end-to-end reconstruction of the esophagus is feasible if there is not too much tension on the suture line.

Case Presentation

R. H., 49-year-old white male, was admitted to the hospital May 9, 1949. Chief complaint: dysphagia. His illness began in March 1949. A few days later while eating he suddenly was unable to swallow food and had to cough it up, but immediately afterwards he resumed eating without difficulty. He consulted a physician who referred him for X-ray examination (fig. 1). The results of this examination prompted the referral to one of the authors, Lloyd, for an esophagoscopy which revealed an early squamous cell carcinoma of the upper third of the esophagus. Physical examination was essentially normal, and there were no tumor masses palpable in either side of the neck. Laboratory examinations showed a hyperchronic secondary anemia.

On May 16, 1949, the first-stage resection of the esophagus was performed through a posterio-lateral incision by resecting the 6th rib and a portion of the 5th rib, opening the pleura, and completely dissecting free the espohagus from the opening in the diaphragm to just below the level of the clavicle. Meticulous care was used in freeing the esophagus, and it was necessary only to ligate two branches from the intercostal vessels just above the arch of the aorta. The esophageal opening in the diaphragm was then incised and the stomach freed. The left gastric artery was ligated well away from the anastomotic branches along the lesser curvature and cardiac end of the stomach. It was not necessary to ligate the left gastroepiploic artery. Having mobilized the esophagus and the stomach, an incision was made in the neck along the anterior border of the left sternocleidomastoid muscle, retracting the large vessels laterally and the thyroid medially,

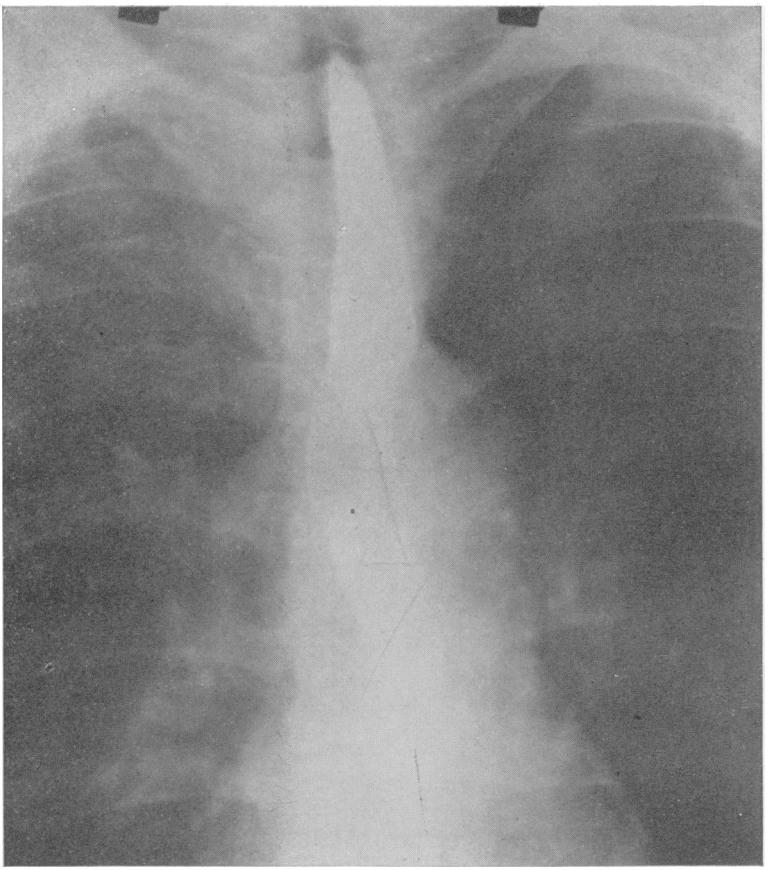

Figure 1. Original X-ray showing defect level of 2d rib anteriorly.

and ligating the inferior thyroid artery. The espohagus in the cervical area was then identified and dissected free until the upper portion of the esophagus could be brought out through the neck incision. The lesion was placed in the mid-portion of an esophageal loop which was left underneath the subcutaneous tissue. Silk sutures were placed in the muscle of the esophagus and sutured to the sternocleidomastoid muscle for identification at the secondary operation and also to localize the site of resection at that time. Before closure of the wounds, the elevated portion of the stomach in the chest cavity was plicated to form a tube-like structure. The diaphragm was then closed and the stomach attached to the diaphragm with interrupted sutures. Before the operation a Levine tube had been placed down through the esophagus into the stomach. This was left in place for feeding purposes (fig. 2). Both wounds were then closed. The lung was re-expanded and a catheter left in place in the pleural space for 48 hours using negative pressure suction. The postoperative course was quite

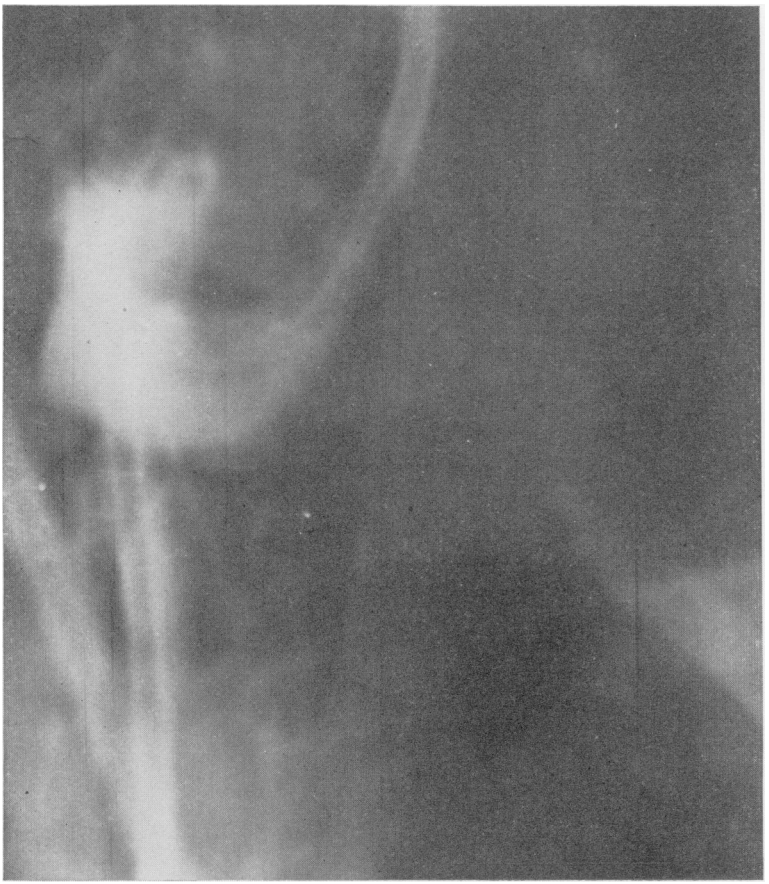

Figure 2. Showing exteriorization of esophagus in neck with Levine tube in place.

smooth. Between the first- and second-stage operations, swallowing through the esophageal loop took place in a normal manner. This supports the view that the mechanism of swallowing depends more upon the propulsive force generated in the mouth than upon peristalsis of the esophageal muscle.

On May 23, 1949, a second-stage resection was carried out by opening the skin incision in the neck and removing the esophageal tumor. This was done under local anesthesia. The anastomosis of the two ends was successful on the posterior surface, but the anterior surface proved difficult because of a thin and friable distal esophagus, and a small area of necrosis apparently caused by erosion of the Levine tube. The anastomosis was done with one layer of No. 000 intestinal cotton. The pathological report revealed a 9 cm. segment of esophagus after fixation and the microscopic section showed squamous cell cancer, grade II (fig. 3). The incision in the neck was closed

Figure 3. Photograph of actual lesion of esophagus removed.

with drainage, and a thin mucoid discharge came through the drainage tract for 5 days and then ceased. The patient was fed by Levine tube for 8 days postoperative. On the 8th day, X-ray by barium swallow showed a fistulous tract and blind pocket under the skin. The tube was removed and the patient was given a fortified liquid diet which he was able to swallow without much difficulty. The diet was progressed to soft foods and after 2 weeks the patient again experienced difficulty in swallowing to the extent that clear liquids were troublesome. The wound had become tender and indurated, and it was necessary to drain a paraesophageal abscess. This drained for approximately a week after which the wound healed. X-ray at this time showed marked angulation of the esophagus (fig. 4). The patient was again readmitted July 25, 1949, and, under general anesthesia, the esophagus was freed above and below the elbow seen on X-ray, and from the scar formation present around the old fistulous tract

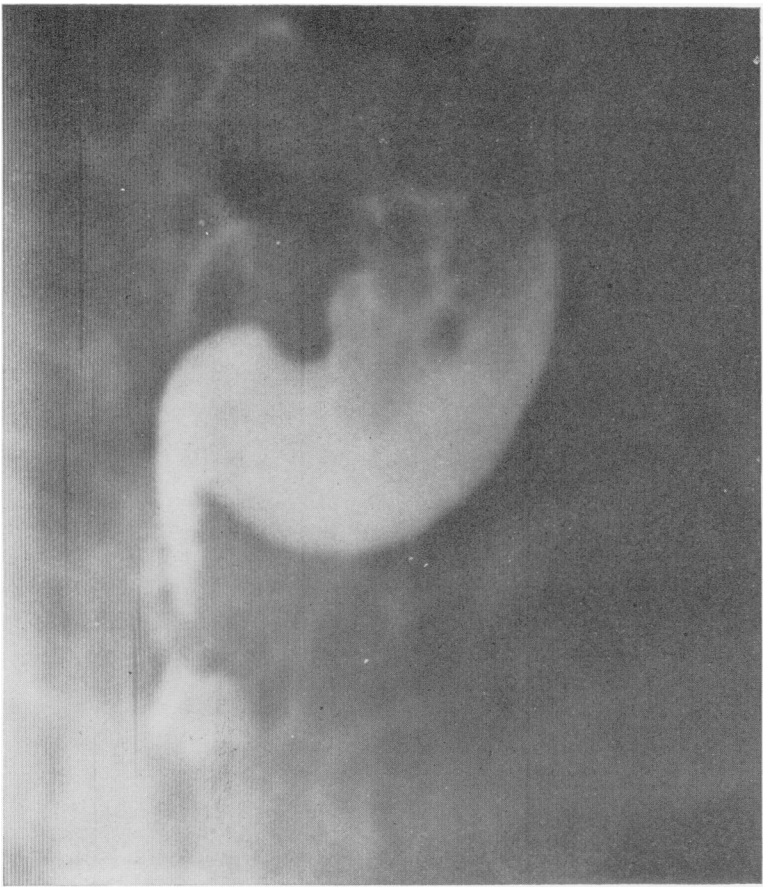

Figure 4. Angulation of esophagus after resection and anastomosis.

and the undersurface of the sternum. The esophagus was normal in appearance and had a rich blood supply. A reanastomous was necessary to eliminate angulation and align the esophagus. This was done by using No. 000 intestinal cotton in one layer. The esophagus was then placed in its normal position. Since the distal portion had a tendency to fall upward against the sternum, a portion of the sternocleidomastoid muscle was excised in its mid-portion, left attached to the sternum and rolled underneath the sternum, and sutured to hold the esophagus in its normal position. The wound was closed with drainage, but was healed by the 5th day. Barium swallow at this time showed the esophagus well aligned and without a fistulous tract. Follow-up of patient 8 months later reveals that his weight and blood count are normal, and he offers no complaint. He is able to eat any type of food and is working daily (fig. 5).

This case is interesting not only from the technical problem in-

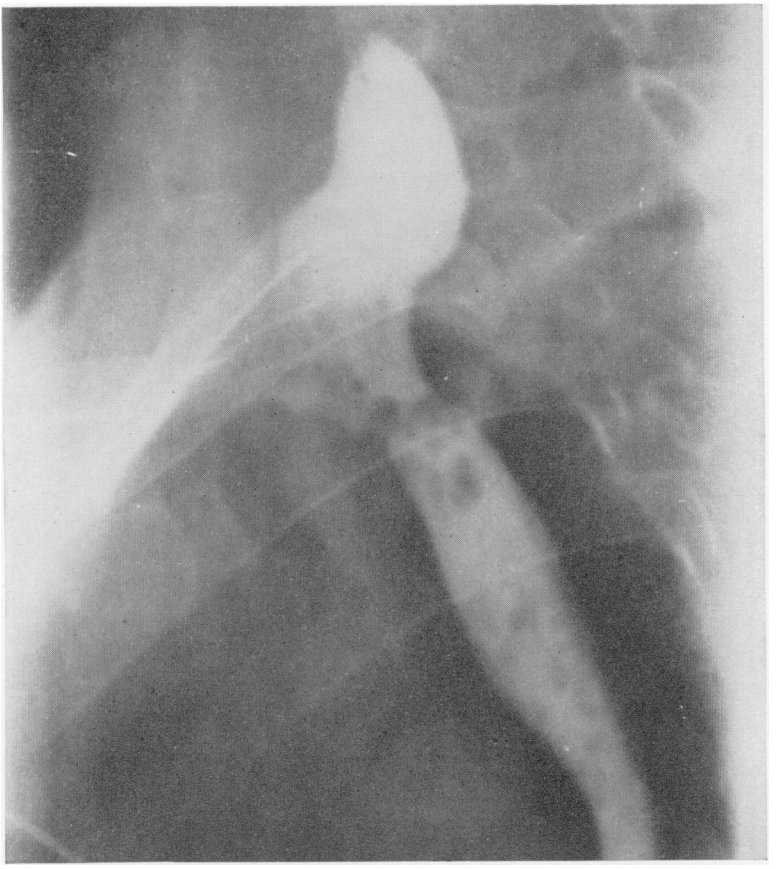

Figure 5. X-ray of esophagus 5 months postoperative.

volved in telescoping 4 inches of the esophagus through the mediastinum, but also in regard to blood supply of the esophagus. Apparently, the esophagus was able to revascularize itself from surrounding structures, as a rich blood supply to this distal portion was noted on the 3d operation approximately 2 months after the original procedure. This was not the case 1 week after the original procedure. This observation justifies nontransection of the esophagus at the primary operation, since a blood supply is provided from both directions until a new source of nourishment can be established. Erosion of the anterior wall of the esophagus from the feeding tube may be avoided by placing the lower end of the esophagus under the loop in the neck instead of over it. This would avoid pressure by the tube against the under-surface of the sternum.

It would appear that this operation may be indicated in those cases where the stomach cannot be brought up into the cervical region and may be applied to all lesions supra-aortic and in the cervical area.

It has the advantage of restoring the continuity of the esophagus, thereby preserving the propulsive force of the swallowing mechanism, plus the peristaltic force of the distal esophagus. The ulcerations of the esophagus which are sometimes seen in these cases postoperatively should be obviated.

It also provides for an anastomosis in the cervical area when the blood supply of the proximal and distal esophagus has been assured, and when the mediastinum is sealed off.

Another advantage is that it does not disturb the vagus nerves and set up the subsequent physiological disturbance which may occur after they are sectioned.

It has the disadvantage of displacing the abdominal viscera for a short distance in the chest, and it is a two-stage procedure. However, the latter should not be of too much importance if the desired result is obtained.

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Colorado Tick Fever

Isolation of Virus Strains by Inoculation of Suckling Mice

By J. W. Oliphant, M. D., and R. O. Tibbs*

The experimental transmission of the virus of Colorado tick fever to man and from man to the golden hamster by means of blood serum obtained from patients during the first or second febrile period of the disease was reported by Florio, Stewart, and Mugrage (*1*). The sera were stored in the cold until used. It was necessary to make repeated passages in the hamster before frank signs of illness or death appeared. Koprowski and Cox (*2*) isolated a strain of virus by intracerebral passage of human serum in DBA mice. The serum was obtained from a Colorado patient during the second febrile period and was chilled on ice during transit to New York.

Primary isolation of the virus by inoculation of albino Swiss mice has not previously been reported. Ten strains of the virus were isolated in this laboratory during 1948 and 1949 by intraperitoneal inoculation of Swiss mice 3 to 5 days old, using either blood serum or emulsified blood clot. The blood specimens were received from physicians in Colorado, Wyoming, Oregon, and Utah during April, May, June, and July. The stated interval between onset of illness and date the blood specimens were obtained varied from 1 to 10 days. The specimens were all forwarded to this laboratory by ordinary mail with a provisional diagnosis of Colorado tick fever. In some instances Rocky Mountain spotted fever or Q fever was also suspected.

The inoculum consisted either of undiluted emulsified blood clot or serum given intraperitoneally in a dose of 0.1 ml. to each of a litter (usually 4 to 7) of mice 3–5 days old. If signs of illness were not seen by the fifth day, some of the animals were sacrificed, the brains pooled, and a 20 percent emulsion made in 10 percent normal rabbit serum-saline. After centrifugation for 10 minutes at 1,000 rpm, 0.1 ml. of the supernate was injected intraperitoneally into each of a fresh litter of mice 3–5 days old. In five instances signs of illness were seen in the first generation of mice at intervals varying from 5 to 8 days after inoculation. In all series definite signs of illness appeared in animals of the first passage, consisting of excitability, hyper-irritability, and muscular incoordination. Terminal stupor and death usually occurred within 48 hours after appearance of illness. All virus strains were tested by neutralization test

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for identity, either against known immune hamster serum (Florio 2 strain[1]) in baby mice or against hamster antisera prepared with the new strains and tested for neutralization of Florio 2 virus in mice about 21 days old. In all cases neutralization of 10 LD_{50} or more of virus was demonstrated. In performing the tests, the source of virus was infected mouse brains harvested about 5 days after inoculation. The brains were emulsified with 10 percent normal rabbit serum-saline to make a 20 percent suspension. After centrifugation at 1,000 rpm, serial tenfold dilutions of the supernate were made with 10 percent normal rabbit serum-saline. To aliquots of these dilutions were added equal quantities of immune serum or normal rabbit serum (controls). Groups of five normal mice were inoculated with each mixture. When the test was done in baby mice, the inoculum used was 0.1 ml. injected intraperitoneally. When 21-day-old mice were used, the inoculum was 0.03 ml. given intracranially.

Difficulty of Adaptation to Older Mice. Considerable difficulty was experienced in adapting virus strains to older mice. Attempts were made to adapt two of the strains to 21-day-old white Swiss mice by repeated blind passage of brain suspensions at 5-day intervals to fresh animals using both intraperitoneal and intracranial inoculations. After seven blind passages of each strain, no evidence of illness appeared in the 21-day-old mice, but 3-day-old mice were readily infected by intraperitoneal inoculation of pooled brain suspension from the seventh passage animals. A third strain was finally adapted to 23-day-old mice by making passages of brain suspension to animals of gradually increasing age at each passage. Greatest difficulty was experienced in making the step from 16-day mice to older animals. It was found necessary to make the 16–17- and 17–18- day steps before the virus could be adapted to older mice.

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[1] Supplied by Dr. H. Koprowski, Lederle Laboratories.

INCIDENCE OF DISEASE

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

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED MARCH 25, 1950

Reported cases of influenza in the United States for the week ended March 25 were 26,505 compared with the corresponding total of 28,945 cases reported for the preceding week and 3,611 for the 5-year (1945–49) median. These figures exclude Kentucky which reported an estimated total of 11,609 cases for the week, compared with only 91 cases reported for the preceding week.

The cumulative total of reported influenza cases for the first 12 weeks of this year is 170,959 compared with the corresponding total of 52,478 cases for 1949 and 170,339 for 1946, the second highest during the last 5 years.

Relatively large increases for the latest week over the previous week were reported in Idaho (272 to 401), Illinois (4 to 29), Iowa (233 to 353), Maine (1 to 1,058), Maryland (61 to 116), Missouri (14 to 184), Montana (283 to 625), New Jersey (24 to 49), New York (49 to 86), North Dakota (17 to 43), Washington (5 to 18), and Wyoming (0 to 71).

The following States reported relatively large numbers of cases: Texas (8,750), Virginia (6,109), West Virginia (2,275), Arkansas (2,241), Maine (1,058), Oklahoma (1,022), and Tennessee (722). Ten States reported 5 cases or less. However, in some of these States influenza is not a legally reportable disease.

A special report from the U. S. Naval Training Center, Great Lakes, indicates that influenza has been prevalent there since mid-February. The weekly sick rate increased irregularly from 5.9 cases per 1,000 personnel for the week ended January 7 to 39.2 for the week ended March 11. Most of this increase is attributed clinically to mild and uncomplicated cases of influenza. Since March 11, however, the sick rate has declined.

Increases over the previous week were reported for the following diseases: measles (10,119 to 11,940), pneumonia (3,021 to 3,147), scarlet fever (1,880 to 1,993), typhoid and paratyphoid fever (39 to 48) and whooping cough (2,867 to 2,901). One case of smallpox was reported in Kentucky. Diphtheria cases decreased from 153 to 114 and poliomyelitis cases from 73 to 62.

Telegraphic case reports from State health officers for the week ended March 25, 1950

[Leaders indicate that no cases were reported]

Division and State	Diphtheria	Encephalitis, infectious	Influenza	Measles	Meningitis, meningococcal	Pneumonia	Poliomyelitis	Rocky Mountain spotted fever	Scarlet fever	Smallpox	Tularemia	Typhoid and paratyphoid fever [1]	Whooping cough	Rabies in animals
NEW ENGLAND														
Maine			1,058	42		6			11				32	
New Hampshire			2			6			5				9	
Vermont				37					9				29	
Massachusetts	4			335	1		1		177			2	211	
Rhode Island			5	2		4			11				28	
Connecticut			10	35	2	46			32			1	131	
MIDDLE ATLANTIC														
New York	5	4	[2] 86	1,272	9	419			[3] 165			6	150	13
New Jersey	3		49	1,004	1	96			70			1	129	
Pennsylvania	2			615	4	121	2		170			6	227	
EAST NORTH CENTRAL														
Ohio	10		3	352	5	125	2		247			1	241	8
Indiana	4		16	434		27	1		85		1	2	44	
Illinois	2		29	317	8	97	3		80		1		81	4
Michigan	8	6	14	2,474	2	85	3		208			1	304	1
Wisconsin			242	580	2	41			105				195	
WEST NORTH CENTRAL														
Minnesota	2		14	115	1	19	1		19				25	
Iowa	1		353	534	1	3			9				16	11
Missouri	1		184	50	2	127			22				24	
North Dakota			43	10		4			1					
South Dakota	1	1		7	1		2		15			1	3	
Nebraska			33	55	1	2			12				8	
Kansas	3		51	29	3	33	1		57				38	
SOUTH ATLANTIC														
Delaware				5					8				80	
Maryland	6		116	36	2	79			19			1	50	
District of Columbia	1		18	110		21			3					
Virginia	4		6,109	47	8	198	1		19				76	2

West Virginia	3	1	2,275	310	1	33	3		25			1	35	21
North Carolina	8			291	5				31			1	35	
South Carolina	4		369	54	1	36	2		1		1		4	9
Georgia	1		371	189	2	19			8		4		5	10
Florida			21	110	1	17	3		4				22	
EAST SOUTH CENTRAL														
Kentucky				376	7	46			22	1			11	17
Tennessee	6		722	138	4				34			5	29	9
Alabama	8	1	675	50	5	85	2		10		5		24	5
Mississippi	1		186	296	2	67	1		6		1	2	4	6
WEST SOUTH CENTRAL														
Arkansas			2,241	42	7	76	1		2		1	2	37	2
Louisiana	3		16	81	3	31	3		6			4	3	6
Oklahoma	2		1,022	14		63	3		19		1	2	23	3
Texas	11	1	8,750	425	11	929	15		24		1	4	215	35
MOUNTAIN														
Montana	2		625	132	2	2			7				1	
Idaho			401	36		13			2				4	
Wyoming			71	24									2	
Colorado			62	61	1	19	1		14				53	14
New Mexico			13	16		17			2				14	
Arizona	2		172	115		24			5				39	1
Utah	1		19	189		7	2		4		3		14	
Nevada						1							1	
PACIFIC														
Washington			18	105	2	6	1		69			1	41	
Oregon			19	26	2	32	1		16			1	37	
California	5	3	22	363	7	65	7		123			3	117	1
Total	114	17	26,505	11,940	116	3,147	62		1,993	1	19	48	2,901	178
Median, 1945-49	272	7	[4] 3,611	21,613	106		31		3,103	4	20	45	2,198	
Year to date, 12 weeks	1,900	150	[4] 170,959	86,141	1,113	30,709	1,199	11	21,573	14	273	558	31,035	183
Median, 1945-49	3,510	88	[4] 117,617	175,422	1,040		494	6	34,189	49	251	522	26,857	
Seasonal low week ends	(27th) July 9		(30th) July 30	(35th) Sept. 3	(37th) Sept. 17		(11th) Mar. 18		(32d) Aug. 13	(35th) Sept. 3		(11th) Mar. 18	(39th) Oct. 1	
Since seasonal low week	6,171		[4] 201,466	105,271	2,026		62		38,012	34		48	52,571	
Median, 1944-45 to 1948-49	11,076		[4] 161,155	210,368	2,011		31		59,663	103		45	51,137	

[1] Including cases reported as salmonellosis.
[2] New York City only.
[3] Including cases reported as streptococcal sore throat.
[4] Exclusive of Kentucky.

Alaska: Influenza 69, pneumonia 4, whooping cough 7.
Hawaii: Influenza 8, measles 1, meningococcal meningitis 1, scarlet fever 1, typhoid fever 1, paratyphoid fever 1.

PLAGUE INFECTION IN LEA COUNTY, NEW MEXICO

Under date of March 24, 1950, plague infection was reported proved in a pool of 60 fleas, *Anomiopsyllus* sp., taken from a wood rat, *Neotoma*, nest found 6 miles north then 2 miles west of Eunice, Lea County, New Mexico.

TERRITORIES AND POSSESSIONS

Puerto Rico

Notifiable diseases—4 weeks ended February 25, 1950.—During the 4 weeks ended February 25, 1950, cases of certain notifiable diseases were reported in Puerto Rico as follows:

Disease	Cases	Disease	Cases
Chickenpox	79	Tetanus, infantile	1
Diphtheria	19	Tuberculosis (all forms)	429
Dysentery	3	Typhoid fever	5
Influenza	41	Typhus fever (murine)	1
Malaria	6	Venereal diseases:	
Measles	13	Gonorrhea	95
Poliomyelitis	8	Syphilis	53
Tetanus	9	Whooping cough	314

DEATHS DURING WEEK ENDED MAR. 25, 1950

	Week ended Mar. 25, 1950	Corresponding week, 1949
Data for 94 large cities of the United States:		
Total deaths	10,500	10,146
Median for 3 prior years	10,146	
Total deaths, first 12 weeks of year	118,958	118,822
Deaths under 1 year of age	626	641
Median for 3 prior years	688	
Deaths under 1 year of age, first 12 weeks of year	7,551	8,042
Data from industrial insurance companies:		
Policies in force	69,847,313	70,534,114
Number of death claims	13,960	12,813
Death claims per 1,000 policies in force, annual rate	10.4	9.5
Death claims per 1,000 policies, first 12 weeks of year, annual rate	9.8	9.8

FOREIGN REPORTS

CANADA

Provinces—Notifiable diseases—Week ended March 11, 1950.—During the week ended March 11, 1950, cases of certain notifiable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Newfoundland	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox			17		294	295	24	36	46	112	824
Diphtheria					11	1			6		18
Dysentery, bacillary						3					3
Encephalitis, infectious						2					2
German measles			109		13	372		37	180	223	934
Influenza			42			223	7	5			277
Measles			19	2	512	624	18	35	30	174	1,414
Meningitis, meningococcal						2					2
Mumps			96		275	632	3	60	135	363	1,564
Poliomyelitis					1						1
Scarlet fever	2		2	2	66	44	10	3	67	11	207
Tuberculosis (all forms)	19		7	6	107	27	20	13	5	27	231
Typhoid and paratyphoid fever					5	2					7
Undulant fever					2	1					3
Venereal diseases:											
Gonorrhea	4		7	4	97	39	27	17	29	65	289
Syphilis	6	1	2	9	75	29	5	10	7	20	164
Whooping cough	1		18		59	62	6	2		61	209

NEW ZEALAND

Notifiable diseases—4 weeks ended January 28, 1950, and 4 weeks ended February 25, 1950.—During the 4 weeks ended January 28, 1950, and the 4 weeks ended February 25, 1950, certain notifiable diseases were reported in New Zealand as follows:

Disease	4 weeks ended Jan. 28, 1950		4 weeks ended Feb. 25, 1950		Disease	4 weeks ended Jan. 28, 1950		4 weeks ended Feb. 25, 1950	
	Cases	Deaths	Cases	Deaths		Cases	Deaths	Cases	Deaths
Diphtheria	4		6		Poliomyelitis	8		18	
Dysentery:					Puerperal fever			2	1
Amebic	3		1		Scarlet fever	76		72	
Bacillary	8		34		Tetanus	2	1	3	1
Erysipelas	5		9		Trachoma			3	
Food poisoning	14	1	17	2	Tuberculosis (all forms)	133	64	194	45
Influenza	3	2			Typhoid fever	8	1	10	
Meningitis, meningococcal	10		9	1	Undulant fever	1		4	
Ophthalmia neonatorum	1								

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

Note.—The following reports include only items of unusual incidence or of special interest and the occurrence of these diseases, except yellow fever, in localities which had not recently reported cases. All reports of yellow fever are published currently.

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

Cholera

Indochina (French).—During the week ended March 11, 1950, five cases of cholera with three deaths were reported in Battambang, Cambodia.

Pakistan.—Four cases of cholera, with three deaths, were reported in Chittagong during the week ended March 11, 1950.

Plague

Brazil.—Reports of plague in Brazil have been received as follows: July 1–31, 1949, 2 cases 1 death in Bahia State; August 1–31, 1949, 10 cases 4 deaths in Bahia State, 2 cases in Pernambuco State; September 1–30, 1949, 12 cases 2 deaths in Bahia State, 3 cases in Minas Gerais State, 1 case in Pernambuco State.

Peru.—During the month of December 1949, 14 cases of plague were reported in Peru—11 cases in Ayabaca Province and 1 case in Huancabamba Province, Piura Department; 1 case in Chota Province, Cajamarca Department; 1 case in Trujillo Province, Libertad Department. During the month of January 1950, 2 cases were reported in Peru, both in Ayabaca Province, Piura Department.

Smallpox

Arabia.—During the week ended March 11, 1950, 10 cases of smallpox were reported in Mecca, and 7 cases in Jedda.

Burma.—Two hundred and eighty-two cases of smallpox, with 157 deaths, were reported in Burma during the week ended March 11, 1950, including 90 cases 35 deaths in Rangoon and 72 cases 36 deaths in Bassein.

French Equatorial Africa.—For the period January 21–31, 1950, 74 cases of smallpox were reported in French Equatorial Africa.

Great Britain—Glasgow.—Information dated March 28, 1950, reported an outbreak of smallpox in Glasgow. As of that date four confirmed and four suspected cases had been reported in that city. The number of contacts was said to be large.

Indonesia—Java.—According to information dated March 21, 1950, an epidemic of smallpox has been reported in Surabaya, Java. Fifty-one cases were stated to have occurred on March 20, with the number increasing on March 21.

Mexico.—During the period February 12–25, 1950, 12 cases of smallpox were reported in Mexico City.

Palestine.—During the month of December 1949, 102 cases of smallpox were reported in Palestine, of which 28 occurred in Jerusalem, 33 in Hebron, and 27 in Jericho. During the month of January 1950, 63 cases were reported in that country, including 5 cases in Jerusalem, 20 cases in Hebron, and 31 cases in Jericho.

Typhus Fever

Egypt.—During the week ended February 25, 1950, 16 cases of typhus fever were reported in Egypt, including 4 cases in the port of Damietta.

Germany (United States Zone).—During the week ended January 21, 1950, one fatal case of typhus fever was reported in Bavaria, in the United States Zone of Germany. Two cases of this disease were reported in that area during the month of December 1949.

Libya.—During the month of February 1950, 19 cases of typhus fever were reported in Tripolitania.

Yellow Fever

Bolivia.—Information from Bolivia dated March 29, 1950, states that in the recent outbreak of yellow fever in that country 850 cases with 230 deaths were reported up to March 14. It is also stated that the outbreak was regarded as under control.

Sierra Leone.—One case of yellow fever has been reported in Sierra Leone. This case was stated to have had onset on January 27, 1950, and to have been confirmed on March 6.