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THE PHARMACOLOGICAL ACTION OF CERTAIN PHENOL ESTERS, WITH SPECIAL REFERENCE TO THE ETIOLOGY OF SO-CALLED GINGER PARALYSIS

(Second Report)

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In a preliminary communication (1) on the probable cause of the recent widespread epidemic of the condition clinically described as peripheral multiple neuritis, believed to have resulted from drinking adulterated fluid extract of Jamaica ginger, certain pharmacological and chemical evidence was presented to show that a phenol, firmly bound chemically with phosphoric acid, appeared to be the specific etiologic factor. The precise chemical nature of the phenolic compound was not revealed owing to the technical difficulties involved in its isolation and identification, though on the basis of the evidence presented it was suspected to be a phosphoric acid ester of one or more of the cresols.

Conclusive evidence on the pharmacological side of this problem was equally lacking, on account of the difficulties experienced in faithfully reproducing the human disease in the usual laboratory animals. After much indirect evidence had been obtained pointing to the cresol ester as the probable immediate cause of the paralysis in question, a successful experiment was conducted upon calves. This experiment showed that the human disease could be faithfully reproduced in the calf, as distinguished from the usual laboratory animals, and that the offending agent must have been contained in a technical grade of tricresyl phosphate, the peculiar action of which was either due to an impurity, to the substance itself, or to a combination thereof with some ginger constituent.

The obvious procedure to carry the solution of this problem to a satisfactory conclusion was to ascertain (*a*) whether an impurity in the technical tricresyl phosphate had anything to do with the paralysis, (*b*) which of the three isomers of tricresyl phosphate had this specific action on the motor nerve mechanism, (*c*) what the probable

reason was for the wide differences in species susceptibility, and lastly (*d*) what is the probable manner of action of the specific ester.

The present experiments are given in an attempt to answer at least in part some or all of these questions. Some of the results of this study indicate that the suggestion made in the first report to account for differences in species susceptibility solely on the ground of differences in hydrolytic cleavage of the ester in different animals is no longer tenable; and while this suggestion may still be in part correct, it will have to be modified to harmonize with the new facts.

EXPERIMENTAL

It will be recalled from the evidence presented in the first report (1) that the most important single lead to the solution of this problem was the observation that the suspected adulterated gingers presented uniformly and characteristically a toxicity in rabbits on oral administration which could not be accounted for by the ginger, the alcohol, or any of the well-known poisons. The chemical finding of phenols on saponification, acidification, and steam distillation directed our attention to stable phenolic compounds for two reasons: First because the phenols could only be obtained upon drastic hydrolysis; and, second, what is perhaps more important, because the symptom complex produced by the suspected ginger in rabbits resembled that of the systemic action of phenol or the cresols, yet differed from them in certain essential details. In particular the manifestations of the adulterated ginger poisoning in the rabbit were characterized by a long delay in the onset of the symptoms, sometimes by an interval of several days, and by evidence of very pronounced stimulation of the spinal cord, sometimes lasting over a period of several days before the condition finally terminated in respiratory paralysis. Furthermore, the toxicity of the suspected ginger extracts was far greater than could be accounted for by the phenols or cresols recoverable from them.

These early observations indicated, therefore, a plan of investigation aiming at a comparative qualitative and quantitative study of the toxicity and cumulative effects in the rabbit of as many phenolic esters as were procurable, for which there was no or only a limited amount of data in the literature.

Four phosphoric acid esters were procured for this work:

1. Triphenyl phosphate, technical grade; and
2. Tri-ortho cresyl phosphate, technical—both supplied by Celluloid Corporation, Newark, N. J.
3. Tri-ortho cresyl phosphate, C. P.; and
4. Tri-para cresyl phosphate, C. P.—both supplied by Eastman Kodak Co., Rochester, N. Y.

There is no reliable information available as to the degree of purity of the technical triphenyl phosphate, or the technical tri-ortho cresyl

phosphate. Some of the constants for the chemically pure tri-ortho cresyl phosphate as furnished by the manufacturers are as follows:

Specific gravity at 20° C	1.18
Boiling point at 20 mm. Hg	263–265° C.
Index of refraction	1.555

It is insoluble in water, and soluble in the usual lipoid solvents. P_2O_5 determination gave a value of 19.0, as against the theoretic value of 19.29 per cent. The Millon color value (11) of the cresol obtained by saponification, acidification, and steam distillation agreed with that given by a corresponding standard prepared from pure ortho cresol.

The toxicity and cumulative effects of these substances were studied in rabbits upon oral administration in a solution of approximately 80 per cent alcohol to make it comparable as far as possible with the adulterated ginger extracts previously used. The amount of alcohol administered at one time was usually not more than 6 c. c. per kilo and never more than 8 c. c. per kilo. For comparative purposes experiments were made with pure phenol and with the cresols similarly administered.

The results of this study are summarized in Tables 1 to 7.

The subcutaneous minimum lethal dose of phenol and of the several cresols for rabbits as given by Meili, (2) Tollens, (3) Wandel, (4) and in part confirmed by ourselves is about as follows:

Phenol	500 mg. per kilo
Ortho-cresol	450 mg. per kilo
Meta cresol	500 mg. per kilo
Para cresol	300 mg. per kilo

Administered orally the phenols are somewhat less toxic; thus Clarke and Brown (5) give the minimum lethal dose of phenol in rabbits on oral administration as 0.6 gm. per kilo.

When these figures are compared with the data given in Tables 1 to 7 it is at once obvious that tri-ortho cresyl phosphate stands out toxicologically apart, quantitatively as well as qualitatively, from either phenol, or the three isomeric cresols, or the phosphoric acid esters of phenol and para cresol. Thus from Table 5 it is evident that the minimum lethal dose of tri-ortho cresyl phosphate in the rabbit is 100 mg. per kilo, and as little as 50 mg. per kilo may result in definite symptoms which may occasionally prove fatal. The corresponding phosphoric acid ester of para cresol, on the other hand, failed to produce definite toxic effects in doses up to 700 mg. per kilo, as shown in Table 6. Furthermore, on repeated oral administration to rabbits, in daily sublethal doses, of some of the phenolic phosphoric acid esters it is shown that unlike tri-ortho cresyl phosphate they appear to be capable of detoxification at approximately the same rate as the corresponding phenols, while the action of the ortho ester is persistent and cumulative. Thus rabbits may survive as much as 1.4

to 1.5 grams per kilo of phenol, 1.4 grams per kilo of triphenyl phosphate, 1.2 to 1.7 grams per kilo of tricresol, 1.0 to 1.5 grams per kilo of ortho cresol, and 1.0 to 1.6 grams of tri-para cresyl phosphate, as shown in Tables 1, 2, 3, 4, and 7, respectively.

Summing up the results of the experiments on rabbits it may be concluded that tri-ortho cresyl phosphate differs from the corresponding cresol or the other cresols or phenol or their phosphoric acid esters, first in toxicity and second in the manner of action.[1] The difference in toxicity is sufficiently clear from the data in the tables. The difference in manner of action may be summarized as follows:

The systemic action of phenol or of the cresols is prompt. If a lethal dose is given, the symptoms appear within an hour or less, followed by coma and death in a very few hours. In case the dose is sublethal, recovery is equally prompt, so that by the following day the animal appears normal.

By contrast, the systemic action of tri-ortho cresyl phosphate is slow. The first effects following the administration of a lethal dose are none other than those of the alcohol in which it is administered. After an interval of from one to several days the animals develop a strikingly characteristic group of symptoms which are unmistakable. They peculiarly combine the manifestations of mild strychnine poisoning with some of the features of phenol poisoning. There is thus a moderate degree of hyperexcitability, but never convulsions; there is a spastic incoordinated gait developing into a generalized fine tremor of the entire musculature of the body; and along with this there is a certain degree of emprosthotonos. This may last for hours or days. With the hyperexcitability persisting, this state gradually passes into one of a flaccid muscular asthenia, involving more especially the muscles of the head and neck, so that the animal takes on a peculiar sitting posture with the head drooping, or perhaps more often and more characteristically an attitude of supporting the body in a state of more or less rigid emprosthotonos, with the aid of the chin as well as the fore and hind limbs. This finally passes into a general flaccid paralysis with impaired heart action, slow and shallow respirations, and death. Small sublethal doses produce in greater or less degree the early symptoms from which the animal eventually recovers. The effects of this poison when injected intramuscularly are essentially the same as when given orally. In no case has it been possible to elicit in the rabbit symptoms in any way comparable with the characteristic wrist drop and foot drop that the human ginger extract victims exhibited. The manifestations of the toxic action of tri-ortho cresyl phosphate in the rabbit are, however, exactly the same in every detail as those observed following the administration

[1] With the possible exception of tri-meta cresyl phosphate, which is not procurable at present and which we are having prepared for further work.

of about 6 c. c. to 8 c. c. per kilo of the adulterated gingers which were believed or known to have caused partial paralysis in man (1). On the basis of the rabbit experiments alone, therefore, there seems to be no question that the adulterated paralytic ginger extract contained tri-ortho cresyl phosphate in an approximate concentration of about 2 per cent. [2] [3]

THE ACTION OF TRI-ORTHO CRESYL PHOSPHATE IN THE MONKEY (*Macaccus rhesus*)

From the series of experiments on monkeys with the suspected gingers and fractions derived therefrom by saponification and distillation which constituted part of the first report, it was concluded that this species was refractory to the phenolic poison, but that when the poison was first hydrolyzed to split off the phenol part of the molecule it exhibited typical phenol poisoning in them. It was suggested, therefore, that the phenolic ester in its firm combination resisted hydrolysis in the monkey, and for that reason was harmless, while in the rabbit, by contrast, it was hydrolyzed with great ease. From the data given in the preceding section on the quantitative studies of the toxicity of tri-ortho cresyl phosphate in the rabbit it should be evident at once that this suggestion can be only partially correct, if at all. Assuming that a full lethal dose of this poison is at once hydrolyzed in the alimentary canal of the rabbit to yield quantitatively the corresponding ortho cresol, there would not be sufficient of the latter available to produce even mild symptoms of phenol poisoning. If the mechanism of action of the ortho ester in the rabbit or any other species is dependent upon hydrolysis and liberation of the ortho cresol it must be assumed that such liberation must take place in selected areas of the nervous system, for ortho cresol is no different pharmacologically from the related isomers. Indeed one may suppose that owing to the peculiar physical characteristics of the ortho isomer of the phosphoric acid esters of the cresols it has a special selective affinity for nervous tissues, and there it may exert its specific action as such, or on account of its firm chemical combination it is only slowly hydrolyzed, perhaps under the influence of specific enzymes, with the gradual liberation *in situ* of the corresponding ortho cresol. If some such mechanism as this is actually operative, the long latent period and its persistence of action become easily understandable, for under such special conditions it would

[2] The data on the toxicity of the paralytic ginger in rabbits reported in the first communication (1) were only approximately correct. The toxicity of the gingers was actually greater than that given in that paper, as subsequent work has shown. The error was due to the fact that the long latent period between the administration of the drug and the onset of symptoms was not realized and the gingers were administered in daily doses until definite symptoms developed, the dose in each case having been more than the minimum required one.

[3] Chemical analysis of this ginger for P_2O_5 confirms this conclusion, since 0.4082 gm. of P_2O_5 was found per 100 c. c. of the ginger. This corresponds to 2.1 per cent of tricresyl phosphate. This determination, however, gives no information as to which of the three isomeric cresols, if any, is in combination with the phosphoric acid.

probably not be subject to the same detoxification process that the body is capable of in its protective mechanism against the phenols, viz, conjugation in the liver to form water soluble glycuronates and sulphates, which are readily eliminated.

Whether such a suggestion will hold or not depends, of course, upon further work. The first requisite, however, would seem to be the production in the monkey, and indeed in all higher animals, of some symptoms referable to the central nervous system, if not the actual motor paralysis of the extremities as seen in man. The experiments on monkeys summarized in Table 8 show conclusively that a motor paralysis of the extremities can be produced uniformly in monkeys by the subcutaneous injection of the chemically pure as well as the technical tri-ortho cresyl phosphate. The failure to produce any symptoms whatever in the monkey with enormous doses of this poison given orally merely indicates that it is practically not absorbed from the alimentary canal in this species. Three monkeys (Nos. 3B, 23, and 7) received 3.0, 10.0, and 15.0 c. c. per kilo, respectively, of technical tri-ortho cresyl phosphate in 5 c. c. per kilo of alcohol by stomach tube, and in no case was there evidence of motor paralysis or indeed any untoward effects. Furthermore, one monkey (No. 21B) received 1.0 gm. per kilo of tri-para cresyl phosphate in alcohol *per os*, and it showed no effects other than of the alcohol Several days later the same monkey received a subcutaneous injection of 1.0 gm. per kilo of the para ester dissolved in olive oil. For several hours there was a slight suggestion of mild systemic phenol action with no further effects. On the other hand, monkeys receiving phenol or tri-cresol orally responded in the usual manner. Thus monkey No. 16A was given daily or every other day oral administrations of 5 c. c. per kilo of a 5 per cent solution of phenol in 80 per cent alcohol until a total of 10 doses had been given, or the equivalent of 2.5 gms. per kilo. Each administration was followed by the usual alcoholic intoxication and symptoms of moderate phenol poisoning, ending in prompt recovery. The same effects were noted in a monkey (2B) receiving 6 doses of 5 c. c. per kilo of 5 per cent tri-cresol in 80 per cent alcohol.[4]

It may thus be concluded from these experiments that the toxicity of phenol and the cresols in the monkey is of about the same order as in the rabbit, and that the free phenols are detoxified in the monkey probably by the same usual process of conjugation. Of the cresol phosphoric acid esters the para isomer is no more remarkable in its pharmacologic action in the monkey than in the rabbit, its toxicity in both species probably being determined by the concentration of

[4] From some experiments that need not be detailed here it appeared quite certain that the minimum lethal dose of phenol in alcohol in the monkey is about 0.5 gm. per kilo. That alcohol influences but little the toxicity of phenol was shown by Clarke and Brown (5) and Macht (6).

para cresol derived from the ester by hydrolysis. The ortho isomer, by contrast, can produce a partial motor paralysis of the upper and predominantly of the lower extremities after an interval usually of from 6 to 8 days. The essential differences in the reaction to this ester in the monkey as compared with man are as follows:

1. It does not appear to be readily absorbable from the gastro-intestinal canal.
2. The paralytic dose is not far from the minimum lethal dose.
3. The motor paralysis in the monkey is of relatively short duration as compared with that in the human cases.

With a better understanding of the action of this poison in the animal body some of these differences may at least in part become obliterated. Thus it is conceivable that by altering the mode of administration of this substance it may be possible to avert its lethal effects. And, lastly, the temporary character of the paralysis in the monkey is at least one hopeful outlook for the human victims.

THE ACTION OF TRI-ORTHO CRESYL PHOSPHATE IN DOGS

The experiments with this substance conducted upon dogs indicate that its behavior in this species is practically identical with that in the monkey. Oral administration of this substance to dogs appears to be without effect, corroborating the negative results with paralytic ginger in dogs previously reported (1). Subcutaneous or intramuscular injection of this ester direct or diluted with olive oil has resulted in a characteristic lameness, especially of the hind legs, with a typically ataxic gait after an interval of from 7 to 18 days. As in the monkey, however, the paralytic dose appears to be not far from the lethal dose, so that when a sufficiently large dose is given to produce pronounced paralysis, death generally follows in a few days. These findings are summarized in Table 9.

THE ACTION OF TRI-ORTHO CRESYL PHOSPHATE IN CALVES

In the preliminary communication on the cause of "ginger paralysis" (1) an experiment made on three calves was reported which served as the only piece of direct evidence to bring a phosphoric acid ester of one or more of the cresols into direct relationship with the recent epidemic of partial paralysis in man. At that time it appeared that the calf was the only animal to show a degree of susceptibility to this poison in every way comparable with that of man. Now that means have since been found of reproducing fairly accurately the human disease in more suitable laboratory animals the plan for conducting further experiments upon calves has been abandoned for obvious reasons. Two other experiments were made, however, which are of sufficient interest to record here, for they not only

confirm the first observations, but also corroborate in large measure all the other experiments reported herein.

The outcome of the experiment on the three calves reported previously (1) was as follows:

Calf No. 1, receiving the control ginger, remained normal for over 9 weeks.

Calf No. 2, receiving the same amount of paralytic ginger, and having developed typical mild paralysis of the posterior extremities within about 3 weeks, remained in apparently the same condition for about 5 weeks. It then began to improve rapidly and in a few days (by the 12th of August) it appeared practically normal.

Calf No. 3, receiving the adulterated U. S. P. fluid extract ginger containing 2.5 per cent technical tricresyl phosphate, resulting in paralysis like that of the preceding calf, remained paralyzed for about 3 weeks. The condition of this calf was getting worse, however, being complicated by a pronounced dyspnea, the cause of which has not yet been cleared up. It was then deemed advisable to sacrifice this animal and save the material for histopathologic studies.

The two surviving calves were subsequently used in another experiment with the following results:

Calf No. 1, was given, on August 6, by stomach tube, 200 mg. per kilo of C. P. tri-ortho cresyl phosphate dissolved in alcohol, the dose of the alcohol being 5 c. c. per kilo. A moderate degree of alcoholic intoxication followed from which the animal recovered the following day. For several days following, there was diarrhea, otherwise the animal appeared normal until the 24th. By the 31st it showed a well-developed "hoof drop" of the posterior extremities, with impaired gait and some ataxia. The animal tired easily and stumbled frequently on running. There was considerable dyspnea.

Calf No. 2, which had nearly, if not completely, recovered, was given, on August 12, an intramuscular injection of 200 mg. per kilo of technical tri-ortho cresyl phosphate. There were no symptoms of any description until the 24th, when there was just a barely detectable weakness of the hind legs. By the 31st there was definite and unmistakable weakness of the posterior extremities, with difficult and ataxic gait.

These two calf experiments, together with those previouslv reported, show that tri-ortho cresyl phosphate given orally or intramuscularly produces in the calf, after a long latent period, a paralysis of the extremities comparable in every detail with the human "ginger paralysis."

THE ACTION OF TRI-ORTHO CRESYL PHOSPHATE IN CHICKENS

Having become thoroughly convinced of the etiologic relationship of the ortho isomer of tricresyl phosphate to the recent epidemic of

so-called ginger paralysis it appeared desirable to extend the search further in the hope of finding a more suitable laboratory animal. The results of experiments upon albino rats have been indifferent so far. Observations upon chickens, however, have shown conclusively that in this species the symptoms as they appeared in man, as well as the entire course of the disease are reproducible with remarkable uniformity, as faithfully and as accurately as could be wished.[5]

Briefly stated, a series of 17 chickens (Plymouth Rocks) have been used in this work so far. The birds weighed from one to two kilograms, usually 1.5 kilos. Tri-ortho cresyl phosphate (technical) was administered by crop in suitable doses measured into number 0 gelatin capsules. The ester was diluted with alcohol when necessary. The following doses were given with the following results:

Group 1. Three birds. Each received 20 mg. of the ester per kilo. No definite leg lameness has become apparent so far (18 days).

Group 2. Three birds. Each received 50 mg. per kilo. Definite leg lameness in 8 to 15 days and partial leg paralysis in 18 days. Wings apparently unaffected.

Group 3. Three birds. Each received 200 mg. per kilo. Definite leg lameness in 11 days. Complete leg paralysis in 12 to 15 days.

Group 4. Three birds. Each received 400 mg. per kilo. Definite leg lameness in 6 to 9 days. Complete leg paralysis in 10 to 14 days. Pronounced wing disability.

Group 5. Three birds. Each received 1.0 gm. per kilo. Definite leg lameness in 8 days, complete leg and wing paralysis in 10 to 12 days.

Group 6. Two birds. One received 0.5 gm. and the other 1.0 gm. per kilo of tri-para cresyl phosphate. No effects whatever so far (40 days).

This experiment is in progress at the present writing, and it is not possible to discuss it in detail. The birds of group 4 have been under observation the longest. One of them was in a state of complete paralysis of the legs and partial paralysis of the wings, but otherwise in apparently good condition for 12 days, when it developed dyspnea and died shortly thereafter. The other two have been in a similar state of paralysis for 12 to 14 days. Both have considerable dyspnea.

It seems likely that the results will ultimately show that the lethal dose of tri-ortho cresyl phosphate in the chicken may be about one-half or one gram per kilo, while 50 mg. per kilo or even less may be sufficient to produce partial paralysis. What the lethal dose of the poison is

[5] While this work was in progress, a brief report appeared by Watkins (9) to the effect that leg lameness developed in chickens following the administration of ginger believed to have caused paralysis in man. Watkins also states briefly that ginger plus phenol has resulted in leg lameness in chickens. It is very doubtful that either ginger or phenol or the combination of the two could result in a type of leg lameness in chickens similar to the partial paralysis in man. All our work on chickens, which is still in progress, points to the phosphoric acid ester of ortho cresol as the specific etiologic factor, as in the other species investigated.

in man we do not know, for apparently there have not been any fatalities directly attributable to the drinking of adulterated ginger extract. From all the experiments reported herein it is not unlikely that the fatal dose in man may be in the neighborhood of one gram per kilo. Since on the basis of the present evidence this substance must have been contained in adulterated ginger in concentration of about 2 per cent, the average adult would have had to imbibe about 3,000 c. c. or more of some 80 per cent alcohol in a relatively short space of time to prove fatal. On the other hand, judging from the chicken experiments, as little as two grams of the poison and possibly less might have been sufficient to cause a moderate degree of paralysis in man. This would have required the consumption of 100 c. c. or less of the ginger extract. This fits in quite well with many apparently authentic histories of one 2-ounce bottle or even less of the ginger extract having caused paralysis in some of the cases (1).

DISCUSSION

The pharmacological evidence presented herein leaves no room for doubt that the phosphoric acid ester of ortho-cresol behaves differently from the manner in which the corresponding cresol or the other cresols behave, and that it is capable of producing specific paralysis of the motor nerves of the extremities in certain species of animals and under certain conditions more or less exactly the same as occurred recently in thousands of human victims, traceable to an adulterated fluid extract of ginger. Furthermore, evidence has been advanced to show that this remarkable specificity upon the motor nerves exhibited by the phosphoric acid ester of ortho-cresol is not shared by the similar esters of phenol or para cresol. It would be extremely hazardous to venture an opinion as to the behavior of the corresponding ester of meta-cresol which is being prepared for future investigation. The question also arises as to whether or not other phenolic esters might not also have their action modified or altered as to be essentially different from the phenols themselves. The problem of the action of phenol esters seems to have received but little attention in the past. Indeed the only piece of satisfactory pharmacological work on this problem is that of Greenwald (7), on meta-cresol acetate, and this shows quite conclusively that its behavior in the body is not different from that of the meta-cresol contained therein.

There are a number of questions that the unfortunate incident giving rise to this unusual find, fortuitous as it was, brings up for consideration. To the writer as pharmacologist, two points emphasize themselves especially: One is the relation of chemical constitution to pharmacologic action. Chapters have been written on this subject, but here is a new instance of a unique relationship of perhaps no known parallel, a relationship that probably no one could have

predicted, for it does not appear to follow any known rule or law. This incident therefore brings out forcibly the need of more new facts to enlarge our limited knowledge of the underlying principles that govern the relation of chemical structure to pharmacologic action.

Another matter that emphasizes itself especially is the desirability of a closer cooperation between the pharmacologist and the synthetic organic chemist. The present work, it is believed, is sufficiently convincing that the recent epidemic of partial paralysis, styled by one writer as "The 1930 Type of Multiple Neuritis" (8) was caused by the highly specific poison tri-ortho cresyl phosphate, the pharmacologic action of which was heretofore entirely unknown. The precise reason for including this remarkable substance as one of the ingredients of a substandard fluid extract of ginger made and sold for beverage purposes will probably never be known, unless a confession is wrung out of the guilty ones. It seems entirely reasonable, however, to suppose that it was included there on account of its physical or other properties which make it difficult to distinguish from the normal ginger constituents. Only a chemist of some ability could have thought of this; and had there been anything known about the pharmacologic action of this substance and the possible dire consequences, it is probable that it would never have been used and the disaster would never have happened. One may wonder whether there are not many other organic compounds of great pharmacologic interest, perhaps some with therapeutic possibilities, awaiting the attention of the pharmacologist.

FURTHER CHEMICAL STUDIES ON THE OCCURRENCE OF TRI-ORTHO CRESYL PHOSPHATE IN PARALYTIC GINGER

The chemical isolation of tri-ortho cresyl phosphate from an alcoholic ginger extract in a state of sufficient purity to enable one to identify it by its physical and chemical constants as distinguished from the other isomers has presented so far considerable technical difficulties. Some further chemical work has been done, however, the results of which fully harmonize with the pharmacological data already presented. Furthermore, pharmacological experiments with such material as has been isolated from the paralytic ginger leave no room for doubt that the fraction isolated is indeed to a large extent tri-ortho cresyl phosphate.

The chemical evidence concerned with the P_2O_5 determination in the paralytic ginger, indicating the equivalent of about two per cent of tri-cresyl phosphate has already been referred to.

The fraction behaving pharmacologically like tri-ortho cresyl phosphate was obtained from adulterated ginger (sample No. 1 of first report) by removal of the alcohol, separation of the supernatant liquid, and distillation of the residue at about 246° C. and 50 mm.

Hg. This fraction had the following properties as compared with those of C. P. tri-ortho cresyl phosphate, the latter as given by the manufacturers and partially confirmed by ourselves:

	Isolated fraction	C. P. Tri-ortho cresyl phosphate
Specific gravity	1.14	1.18.
Refraction index	1.545	1.555.
Boiling range	246° C.-275° C. (50 mm. Hg.).	263° C.-265° C. (20 mm. Hg.).
P_2O_5	13.8	19.0.

This fraction was found not to contain any free phenols and also that the phosphoric acid was apparently as firmly combined in this fraction as in tri-ortho cresyl phosphate. Although the Millon reagent applied to the distillate obtained upon saponfication with strong alkali, acidification, and steam distillation indicated the presence of at least one other phenol in this fraction besides ortho cresol, it gave Melzer's benzaldehyde test which, according to Autenrieth (10) is given only by ortho cresol and not by the other cresol isomers.

Pharmacologic examination of this fraction showed the following:

A. Rabbits.—Five rabbits receiving 0.125 c. c. per kilo, administered *per os* in the usual way in alcohol, survived. One, however, showed slight but definite symptoms of tri-ortho cresyl phosphate poisoning and another moderately severe symptoms.

Three rabbits receiving 0.200 c. c. per kilo exhibited typical symptoms of tri-ortho cresyl phosphate poisoning and died in from 2 to 5 days.

B. Monkeys.—Two monkeys receiving subcutaneously 1.0 and 2.0 c. c. per kilo, respectively, developed after an interval of 6 and 8 days typical motor paralysis of the lower extremities, the one mild, and the other moderately severe.

C. Chickens.—Three chickens each receiving orally 0.5 c. c. per kilo of this fraction all developed distinct leg lameness in from 8 to 9 days and pronounced paralysis of the legs in from 10 to 12 days.

These experiments prove conclusively that the fraction isolated from the suspected ginger contained some 50 per cent or over of tri-ortho cresyl phosphate. What else it may have contained can not be stated, nor is it pertinent.

SUMMARY

A pharmacologic study of the action of the phosphoric acid esters of phenol and some of the cresols has shown conclusively that tri-ortho cresyl phosphate, and in so far as the present evidence goes, it alone, can produce in experimental animals a specific type of motor

paralysis of the extremities in every sense comparable with that which occurred recently in human victims who drank of an adulterated fluid extract of Jamaica ginger.

Some of the differences in species susceptibility to tri-ortho cresyl phosphate previously reported appear now to be due to differences in its absorbability from the alimentary canal. Certain other differences in species susceptibility can not yet be accounted for on the basis of our present limited knowledge of the manner of action of this poison in the animal body.

Pharmacologic evidence has been presented to show conclusively that the adulterated fluid extract of Jamaica ginger used for beverage purposes, resulting in an epidemic of partial paralysis, contained tri-ortho cresyl phosphate to the extent of about 2 per cent. The chemical evidence we have secured confirms the pharmacologic evidence and fully harmonizes with it.

The etiologic relationship of tri-ortho cresyl phosphate to the recent epidemic of so-called ginger paralysis is thus definitely established.

It is a pleasure to acknowledge the cooperation of the Bureau of Animal Industry, Department of Agriculture, in making it possible to carry out the calf experiments.

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TABLE 1.—*Action of phenol in rabbits administered daily in 80 per cent alcohol per os*

No.	Weight, kilos	Dose				Result[1]
		C. c. per kilo	Solution, per cent	Number of doses	Total grams per kilo	
67	2.5	6	2	13	1.560	S
68	1.6	6	2	12	1.440	S
69	2.0	5	2	14	1.400	D (coccidiosis)
61	4.0	5	10	1	0.500	D (acute phenol poisoning)
62	2.9	5	10	4	2.000	D
63	2.1	5	10	2	1.000	D (acute phenol poisoning)

[1] S=survived 2 weeks or more after treatment had been discontinued; D=died

TABLE 2.—*Action of triphenyl phosphate (technical) in rabbits administered daily in 80 per cent alcohol per os*

No.	Weight, kilos	Dose				Result [1]
		C. c. per kilo	Solution, per cent	Number of doses	Total grams per kilo	
43	2.6	6	2	6	0.720	S
44	2.0	8	2	9	1.440	S
45	2.3	8	2	9	1.440	S
108	2.0	5	5	5	1.250	D
109	1.8	5	5	3	0.750	D
110	1.8	5	5	5	1.250	D

[1] S=survived 2 weeks or more after treatment had been administered; D=died.

TABLE 3.—*Action of tricresol in rabbits administered daily in 80 per cent alcohol per os*

No.	Weight, kilos	Dose				Result [1]
		C. c. per kilo	Solution, per cent	Number of doses	Total grams per kilo	
164	2.0	5	2.5	10	1.250	S
165	2.1	5	2.5	10	1.250	S
166	2.1	5	2.5	10	1.250	S
167	2.5	5	2.5	3	0.375	D
168	2.3	5	5.0	7	1.750	S
169	2.1	5	5.0	6	1.500	D
170	3.5	5	5.0	7	1.750	D
171	3.0	5	5.0	6	1.500	S

[1] S=Survived 2 weeks or more after treatment had been administered; D=died.

TABLE 4.—*Action of ortho cresol in rabbits administered daily in 80 per cent alcohol per os*

No.	Weight, kilos	Dose				Result [1]
		C. c. per kilo	Solution, per cent	Number of doses	Total grams per kilo	
156	2.3	5	2.5	8	1.000	S
157	2.0	5	2.5	4	0.500	D
158	2.7	5	2.5	8	1.000	D
159	2.3	5	2.5	3	0.375	D
176	1.9	5	2.5	9	1.125	S
160	2.3	5	5.0	6	1.500	D
161	2.2	5	5.0	3	0.750	D
162	2.5	5	5.0	2	0.500	D
163	2.2	5	5.0	6	1.500	S
177	2.3	5	5.0	6	1.500	S
178	1.6	5	5.0	6	1.500	S

[1] S=Survived 2 weeks or more after treatment had been administered; D=died.

TABLE 5.—*Toxicity of C. P. tri-ortho cresyl phosphate in rabbits, 2.5 per cent solution in 80 per cent alcohol per os*

No.	Weight, kilos	Dose per kilo		Result
		C. c.	Mg.	
185	2.9	6	150	Died within 24 hours.
188	2.5	6	150	Died in 2 days.
183	1.9	6	150	Died in 2 days.
189	2.5	6	150	Died in 4 days.
184	1.9	6	150	Died in 6 days.
192	1.9	4	100	Died in 2 days.
190	2.3	4	100	Died in 3 days.
193	2.3	4	100	Died in 3 days.
174	2.2	4	100	Moderately severe symptoms—survived.
175	2.1	4	100	Moderately severe symptoms—survived.
148	2.0	3	75	Moderately severe symptoms—survived.
149	1.5	3	75	Moderately severe symptoms—survived.
150	3.0	3	75	Moderately severe symptoms—survived.
195	2.1	3	75	Died in 4 days.
199	2.0	2	50	Died in 9 days.
197	2.2	2	50	Definite symptoms for 4 days followed by recovery.
198	2.1	2	50	No effects—survived.
200	2.6	2	50	Symptoms 4 days after administration lasting 10 days.

TABLE 6.—*Toxicity of C. P. tri-para cresyl phosphate in rabbits, 10 per cent solution in 95 per cent alcohol per os*

No.	Weight, kilos	Dose per kilo		Result [1]
		C. c.	Mg.	
201	2.6	5	500	S
202	2.1	5	500	S
203	2.3	5	500	S
204	2.5	5	500	S
205	2.3	7	700	S
206	2.0	7	700	S
207	2.0	7	700	D

[1] S=survived; D=died.

TABLE 7.—*Action of C. P. tri-para cresyl phosphate in rabbits administered daily in 95 per cent alcohol per os*

No.	Weight, kilos	Dose				Result [1]
		C. c. per kilo	Solution, per cent	Number of doses	Total grams per kilo	
141	2.0	4	2.5	11	1.100	D
142	1.9	4	2.5	14	1.400	D
144	2.0	4	2.5	13	1.300	D
151	2.6	4	2.5	10	1.000	S
152	2.2	4	5.0	8	1.600	S
153	1.6	4	5.0	5	1.000	D
154	1.9	4	5.0	8	1.600	S
179	1.8	5	5.0	4	1.000	D

[1] S=survived; D=died.

TABLE 8.—*Action of tri-ortho cresyl phosphate in monkeys (subcutaneous injections)*

No.	Weight, kilos	Dose injected, c. c. per kilo	Grade	Interval before onset of paralysis, days	Duration of motor paralysis, days	Result
11C	3.3	0.05	T.			No effects.
4D	3.5	.20	T.			Do.
22A	4.0	.50	T.			Do.
23A	2.7	.50	C. P.	4	3	Paralysis severe. Died.
4D1	3.5	1.00	T.	6	11	Do.
11C1	3.3	1.00	T.	6	2	Do.
17A	3.7	1.00	T.	8	2	Do.
22A1	4.0	1.00	T.	7	6	Paralysis severe—sick. Killed.
15B	3.3	1.00	T.	8	15	Paralysis moderate—improving.
16B	3.8	1.00	C. P.	4	16	Paralysis moderate—recovered.
12D	3.3	1.00	C. P.	8	8	Paralysis moderate—progressing.
2C	3.0	.50	C. P.			No effect.
1C	4.6	1.00	C. P.	7	40	Paralysis severe. Died.

TABLE 9.—*Action of tri-ortho cresyl phosphate in dogs*

No.	Weight, kilos	Dose c. c. per kilo	Grade	Route	Result
14	10.9	0.40	C. P.	Os	No effects in 40 days.
15	10.9	1.00	C. P.	Os	Do.
16	10.9	3.50	T.	Os	No effects in 30–50 days.
17	11.0	4.00	T.	Os	No effects in 30–50 days.
6	10.5	.20	T.	Subcutaneous.	No effect.
11	4.5	.20	C. P.	do	Do.
8	12.7	.40	T	do	Generalized spastic paralysis in 7 days, followed by death on the eighth day.
7	12.8	.50	T.	do	9 days after injection there was weakness of anterior and posterior extremities, lasting 13 days. Died.
5	10.5	.50	T.	do	Very slight weakness of posterior extremities 3 weeks after injection, lasting 16 days. Recovered.
9	7.3	.60	C. P.	do	No effect in 23 days.
12	7.3	1.40	T.	do	Flaccid paralysis of anterior and posterior extremities in 7 days. Paralyzed 8 days. Died.
13	9.1	1.60	T.	do	Paralysis as above in 8 days. Paralyzed 2 days. Died.
6	10.5	1.00	T.	do	Weakness of posterior extremities with some incoordination 18 days after injection. Paralyzed 30 days. Died.

UNDULANT FEVER

With Special Reference to a Study of Brucella Infection in Iowa

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(The first part of this article, dealing with the history, etiology, and epidemiology of undulant fever, was published in PUBLIC HEALTH REPORTS for October 10, 1930 [1])

VI. CLINICAL INFORMATION

GENERAL

Marston (2), in his essay on fevers, pointed to the outstanding characteristic of this infection in these words: "There is no fever so irregular as this in its course and symptoms." Hughes (1), who has given us an excellent clinical description of undulant fever, introduces his chapter on symptomatology as follows: "So variable are the symptoms and so uncertain is the duration and course of this fever, that it is impossible to give a description to which all cases can be referred." Craig (10) reiterated the same thought when he said: "It is extremely difficult to describe accurately all the forms which this truly protean disease may assume." A simple clinical description of the commonest type of case would, therefore, be misleading. We shall here present descriptions of the four accepted clinical types of this disease, followed by a clinical analysis of the cases studied in Iowa, supplemented by data taken from the studies of Simpson (63), Kern (80), and others. The description here given will be restricted to that of undulant fever due to *Br. melitensis* var. *abortus* and *suis;* but, in conclusion, the characteristics of this infection will be compared with that of undulant fever of caprine origin.

CLINICAL TYPES

Undulant fever was first classified by Hughes (1). On the basis of differences in temperature curves, he described three types—the malignant, the undulatory, and the intermittent. He also recognized an ambulatory form and mentioned the irregular and mixed varieties. These same types have been observed in cases of infection with the more recently discovered varieties of *Br. melitensis,* although not well defined. Still the acceptance of this classification does facilitate an adequate clinical description.

[1] The complete article will be issued later as National Institute of Health Bulletin, No. 158.

Intermittent type.—Most of the Iowa cases of human infection with *Brucella* were of this type. The onset was insidious, a sense of progressing afternoon weariness first oppressing the patient. General aching, some headache, a distaste for food, spells of chilliness in the early evening, and moderate insomnia followed in turn, and sometimes a suspicion of fever. Backache, stiffness or pain in the neck and joints, constipation, and loss of weight were added to the accumulating signs and symptoms. There was, in some cases, a hacking cough which was occasionally persistent. Later, night sweats occurred, frequently drenching in character. Repeated rigors were sometimes distressing. It was usually a matter of weeks before these patients sought medical advice, most often in an office consultation. These patients often found difficulty in defining their ailments; or, perhaps, one of the above mentioned symptoms was the chief complaint. Physical examination usually revealed no abnormalities except the signs of anemia, weakness, and loss of weight, although sometimes the spleen was palpable or the abdomen tender. The patients usually felt much better when confined to bed either by the physician's advice, or their own disabilities. With mild infections they might be up in the morning, but glad to rest in the afternoon. The most persistent symptoms were anorexia and weakness, or weakness alone. These symptoms, plus the fever, were in some cases the only manifestations of disease. The severity of these cases varied, so that while some were confined for only a few days, others suffered a prolonged infection which terminated fatally. Most of the infections lasted between six weeks and four months, with about one-third of this period spent in bed. Morning temperatures were found between normal and 100° and evening temperatures between 101° and 104°. A few complete records revealed superimposed undulatory waves. The fever terminated by a slow lysis, but early in convalescence it readily recurred following overexertion (fig. 22). Cases 1B and 2B, reported in Appendix B, illustrate this type.

The ambulatory type.—In our series an average of 25 per cent of the cases were ambulatory. Simpson reported that one-fourth of his cases experienced a relatively short and mild illness, 12 per cent remaining at work throughout. The onset in these cases was quite insidious, the one constant symptom and occasionally the only one, being weakness or lack of endurance. All the symptoms already noted in the intermittent form occurred in some cases, though mild in degree. Physical examination usually revealed no abnormality. The spleen was palpable in a few patients. The temperature, normal in the forenoon, rarely reached 101° in the evening. The duration varied from two weeks to several months, but often it was more than one month and less than four. We have here a gradation from

the mild intermittent form to subclinical infection. Illustrative cases are No. 3B and 4B, Appendix B.

Undulatory type.—The distinguishing characteristic of these cases was the occurrence of relapses. When intervening short periods of apyrexia occurred, the temperature records had a wave-like appearance. This feature was a frequent occurrence in the Mediterranean cases, but has only occasionally been met with in cases of infection with the *abortus* or *suis* variety. Fifteen per cent of our cases, and also of Simpson's Ohio cases, suffered relapses; but even in these, typical undulations were rarely observed. Because the onset of these cases was accompanied by complaints of weakness, general aching, headache, and anorexia, it often suggested to the patient and his physician the presence of influenza or the so-called "intestinal flu" or "summer flu." Scarcely recovered from the first attack, a second supervened in which the early symptoms were aggravated and to which were added headache, constipation, and insomnia. Night sweats sometimes occurred from the first, but often were not noted until later. Characteristically, the temperature increased day by day, in a step-like manner, until the maximum was reached. Morning remissions were not marked, and after a variable period the temperature decreased by a gradual lysis. Occasionally such a train of events was repeated several times in the same patient. In other cases, however, the disease began as the usual intermittent type and was followed, at variable intervals, by one, two, or more relapses. These usually decreased progressively in intensity and duration. We have observed that our cases of the undulatory type equalled in severity the milder and *moderately severe* intermittent forms. Without carefully following these cases, one can not state with certainty their actual duration, but we have not observed an undue prolongation of symptoms. Temperature curves are shown in Figure 22 and selected cases reported in Appendix B (cases 5 B and 6 B).

The malignant type.—Infections of this nature due to *abortus* or *suis* varieties of *Br. melitensis* were rare, comprising less than 1 per cent of the Iowa cases. They were characterized by sudden onset, an acute course, and usually a fatal termination. The temperature was high and sustained, with an extreme hyperpyrexia occurring before death. There were great prostration, severe headache and backache, marked anorexia, and usually true rigors, and constipation. Sooner or later delirium and coma appeared. Profuse perspiration seemed to be lacking. The spleen was much enlarged. The duration of both of our cases of this type was about three weeks. These cases are described in the Appendix (Nos. 7 B and 6 D).

ATYPICAL CASES

In the diagnosis of undulant fever, due consideration must be given to the occurrence of atypical forms. These infections may closely simulate other diseases, and an accurate diagnosis is then dependent upon laboratory findings. Atypical cases, selected from our series, are presented in Appendix C. A study of these case histories will reveal that undulant fever may present the clinical manifestations of typhoid fever, tuberculosis, broncho-pneumonia, meningitis, cystitis, "rheumatism," and various surgical conditions. This infection may simulate other disease entities, especially during the period of onset, as is illustrated by case 7 C, in which the complicating "orchitis" of undulant fever was first considered to be a gonorrheal epididymitis. Another case was particularly well disguised. A farmer had injured his right foot, but the wound healed after local treatment. Twelve days later, however, he returned to his physician complaining of a "stiffness" of his limbs, chiefly the right leg. Because of difficulty in accounting for these symptoms on any other basis, they were regarded as the earliest indication of tetanus. Antitoxin was given, but undulant fever developed, and during the course manifested the usual symptoms and signs.

In cases such as these, errors in diagnosis are excusable, perhaps unavoidable. It may, however, be observed that these errors often resulted from considering only the immediate complaint and some local condition. A full history and a complete physical examination made by an examiner having an accurate knowledge of undulant fever in all its forms, and aided by the available laboratory tests, would, we believe, give early and accurate diagnoses in a large majority of the cases of this disease.

FATAL CASES

At the present time data are inadequate for any description of the morbid anatomy of infections with the *abortus* or *suis* varieties. This can be determined only from an accumulation of the information contained in the reports of fatal cases, and of necropsies. For these reasons, we detail, in Appendix D, the symptoms, signs, and course of the 10 fatal cases in Iowa, and the necropsy findings in two cases. The following observations on this group are presented:

In five cases death occurred without clinical evidence, in one case without pathological evidence, of any complication or localized infection; there was involvement of the cardiovascular system in three instances, revealing evidence of malignant endocarditis in two of these; in one a lung abscess occurred; in another the gastrointestinal system was mainly involved. The etiological relationship of *Brucella* to the production of these fatal complications was uncertain. It is

to be noted that some cases which began as the intermittent or ambulatory type terminated fatally as well as those which, from the first, were malignant in nature.

In addition, two other fatalities occurred which may possibly be attributed to *Brucella* infection. One patient, with a past rheumatic history and a well compensated mitral lesion, developed an auricular fibrillation early in his attack of undulant fever. Throughout his illness cardiac symptoms were prominent, and following subsidence of fever he failed to gain and died a few months later. The second case was that of a farmer who had prolonged clinical manifestations of undulant fever, but whose serum agglutination was not above 1:40 dilution. Cultures were not taken. At his death the attending physician performed a necropsy, but failed to find gross lesions which could account for the death. The tissues were not saved for section.

In the literature there are few reports of fatal cases of *Br. melitensis* infection, variety *abortus*. Baastrup (81) describes a 6-month illness in a gardener of 48 years. The immediate cause of death was uremia, and this was attributed to an acute nephritis caused by *Brucella*. Such a complication is most unusual and the possibility of the nephritis having an unrelated etiology must be recognized. Scott and Saphir (82) recently report the isolation of *Br. melitensis* var. *abortus* from the blood stream of a patient whose illness of nine months was terminated by endocarditis with embolic phenomena. *Brucella* was also isolated from blood obtained at necropsy, and at no time was any other organism cultured. The clinical history was that of a prolonged undulant fever, with at least one afebrile period of undetermined length. A leucocytosis was found, but only one count was reported. Clinically there was a mitral stenosis which was accounted for by a clear history of acute rheumatic fever. On both the mitral and aortic valves friable, grayish, or yellowish gray vegetations were found. The spleen was markedly enlarged. The authors are very guarded in their conclusions. Still we believe that the terminal illness in this case may be largely explained as a *Brucella* infection. Kristensen (60) mentions 7 fatalities among 216 patients, but only 2 of these had been healthy immediately preceding the onset of undulant fever. Two other fatal cases have been reported by Duffie (83), but the epidemiological, clinical, and laboratory data do not justify their acceptance as *Brucella* infections.

A CLINICAL ANALYSIS

Undulant fever is a generalized infection. Occasionally definite evidence of localization appears, though variable in location, so that all symptoms and signs must be included in a complete consideration of the disease. Three hundred of the Iowa cases provide adequate data for a detailed study. An analysis of these cases in toto has served

to verify in a large measure the findings on 125 cases previously reported (99). Any significant differences in the two groups will be noted here. A composite presentation of the information contained in Simpson's (63) report on 90 cases, the observations of Kern (80), the data in numerous case reports in the literature, and our own study, ought to provide an adequate conception of infection due to the *abortus* and *suis* varieties of *Br. melitensis*.

INCUBATION PERIOD

An accurate determination of the incubation period was difficult in the majority of cases, because exposures were usually multiple and the exact day of onset could rarely be named. In human infections, experimentally produced with *Br. melitensis* var. *suis*, Otero (66) reported that the incubation period of one was at least 17 days, and may have been 34 (two exposures were given 17 days apart). The second infection arose after five daily exposures, with suggestive symptoms at 7 days, and definite symptoms at 10 days after the last dose. The incubation period in these cases was apparently between 10 and 15 days. In a recent personal communication from this author he stated that in six cases, infected through the abraded skin, the periods of incubation were from 10 to 16 days. In one of our cases, a packing-house employee, the disease followed a severe laceration of the arm. The wound was produced by the knife used in freeing the rectum in a freshly killed hog. Eighteen days later his first symptoms appeared, although it was four weeks before he consulted his physician. In such cases a heavier inoculation than usually occurs may be assumed. The data obtained through the experimental infection of monkeys is scarcely comparable, though Huddleson has shown, by the demonstration of agglutinins, that infection was well established in from 10 to 15 days following exposure. Our impression is that in human beings, naturally infected, the incubation period varies from 10 days to 3 weeks.

ONSET

The onset of undulant fever may be sudden or insidious. The physician may be called a few hours after appearance of acute symptoms (see case 70), or as in suspected tuberculosis cases, medical consultation may be sought after weeks of mild disability (case 2 C). The intervals from the appearance of first symptoms to the medical consultation, or to the time when the patient became bedfast, we have designated in our case records as the period of onset. Among 230 cases the onset duration in 27 cases (12 per cent) was less than one week, and one-half of these were ushered in abruptly. In 38 cases (17 per cent) it was one week; in 55 cases (24 per cent) ten days to two weeks; in 61 cases (26 per cent) three weeks to two months; in

19 cases (8 per cent) six weeks; in 30 cases (13 per cent) two months or longer.

During this period the symptomatology was highly varied. In some cases clinical symptoms of an acute respiratory infection, including sinusitis, preceded the prolonged illness, and in some cystitis or pyelitis apparently first gave concern. Whether or not these local infections during the invasion were specific has not been determined. An acute onset following operative procedure has been noted by Kern (80). He further mentioned the case of Warren, Smith, and Linder, in which a sudden onset of illness followed a dose of typhoid vaccine. One of our cases was similar in nature. The patient suffered from chronic appendicitis and came into the hospital for operation with no immediate complaint. A slight elevation of temperature was manifest on the evening of admission, and after her operation the following day an acute febrile condition developed, which proved to be a *Brucella* infection. With these cases it seemed probable that a very mild subclinical or a dormant infection had been provoked into acuteness by conditions which lowered the resistance.

With the exception of two cases which began abruptly with rigors, all cases with rapid or with insidious onset were initiated by similar symptoms though differing markedly in intensity. Varying degrees of lassitude, weakness, lack of energy, or easy tiring were the initial symptoms in slightly more than one-half of our patients. Headaches gave the first indication of illness in 10 per cent while in others spells of chilliness, anorexia, and general aching were noticed. Hence the patient sometimes stated that his illness began with an attack of "la grippe," "flu," or "intestinal flu." Infrequently the first symptoms were night sweats, backache, stiffness of the neck or joints, arthralgia, abdominal pain, drowsiness, and dizziness.

On visiting a physician, the patients' complaints included the above symptoms, but most frequently they sought medical advice because of suspicion of the occurrence of fever. Less frequently, general or localized aches, abdominal pain, loss of weight, painful micturition, insomnia, cough, constipation, or dizziness, either singly or variously combined, were the complaints emphasized. Others, though rare, were those related to the complications of undulant fever.

SYMPTOMS

The common symptoms and signs and their relative frequence are shown in Figure 21. The occurrence of severe symptoms and prominent physical findings are also indicated. These tabulations were prepared from our own clinical record forms where not only the fact of their presence or absence, but the degree of severity, the time of occurrence, and other characteristics of the symptoms were briefly

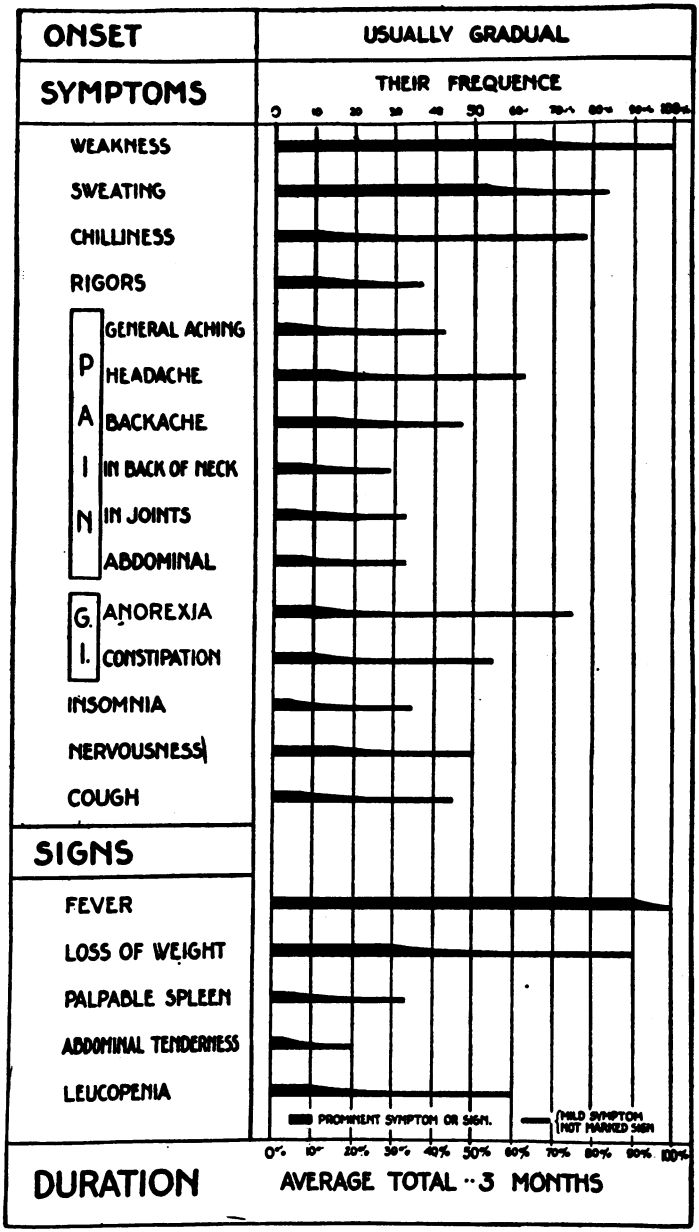

FIGURE 21.—Common clinical characteristics of *Br. melitensis* var. *abortus* and *suis* infections. (Data from which this chart was prepared were obtained by an analysis of the Iowa cases.)

noted at the time of taking the history. The following observations have been made concerning these symptoms:

Weakness.—This was the one symptom assuredly present in all cases, although in mild infections it was often experienced only in the afternoon. Occasionally it constituted the only subjective manifestation of the disease. During the period of onset it was the most common symptom; during the fastigium in two-thirds of the cases, the most prominent or severe; and during convalescence, the most persistent.

Sweating.—The most distinctive feature of the disease was the sweating, which occurred in 84 per cent of our cases. Such patients experienced marked remissions of temperature and included most of those whose temperature curves were of the intermittent form. The very mild cases with a low-grade fever, and the malignant ones with a high and sustained temperature, were those which experienced no sweating.

In 53 per cent of our cases, as in Simpson's, the sweating was profuse or moderately so. It usually occurred soon after midnight, and was of short duration. The patient ordinarily awakened bathed in perspiration, but again rested comfortably after a change of linen. The diaphoresis was sometimes, however, quite prolonged, necessitating several changes during a single night, or it occurred irregularly whenever the patient slept, even in the forenoon. This symptom still appeared at night in two ambulatory cases, who worked during night hours.

When true rigors did occur, sweating followed; but the rigor was not constantly related to any other symptom. Occasionally the nurse, attendant, or patient reported a very disagreeable odor associated with the perspiration. Sometimes a regional sweating was reported, but usually the diaphoresis was general.

Chills.—Chilliness was a symptom of the period of invasion and usually occurred in association with the daily rise of temperature. Although experienced by 77 per cent of the cases, it usually gave little discomfort. A farmer, for instance, counteracted it by wearing a heavy sweater, even though others were complaining of the summer heat. An afflicted physician wore his topcoat, even in the warm operating room while giving an anesthetic. Relief was thus sought and ordinarily obtained by additional covers or external heat. Once the patients became bedfast the symptom usually disappeared.

True rigors were a feature of more than one-third of our cases, though in but 12 per cent did more than two occur. When these appeared early they frequently led to a diagnosis of pneumonia and when they developed during the course, if regularly recurring, they suggested malaria. In an occasional case there was more than one in the 24 hours, one patient reporting two a day for several days in

succession, and another stating that on one day he had five. In the mild infections rigors were not noted; in the severe cases they were common.

Only in patients who also experienced spells of chilliness did the true rigors occur, and occasionally from history alone it was not clear whether the patients had only severe chilliness or a true rigor.

Pain.—In many cases the physician was much impressed by his patient's almost complete freedom from pain. In the morning there was usually no complaint, and, if bedfast, the patient was generally ready to talk and joke. Yet in the case of an infection so disseminated, one can not be surprised that aches and pains do occur in association with almost every system.

General aching was complained of in less than one-half of the cases, but was a prominent symptom in but 5 per cent. In ambulatory patients it often persisted throughout the disease and was aggravated by exercise, but in patients who became bedfast it usually disappeared rapidly. Some individuals described this aching as resembling the muscular soreness which follows overexercise; some likened it to the effects of a generalized trauma, while others said it was "just like the flu."

Headache, a common initial symptom, was ordinarily confined to the early stages of the disease. It was associated with the fever, hence appeared in the afternoon and was most severe in the evening; at times it was accompanied by pain in the eyes. Usually it was bilateral and frontal, rarely occipital.

Early in the disease a mild pain in the lumbar region was often induced or aggravated by exercise. Sometimes it became quite persistent and difficult to control and in 15 per cent of our cases it became a prominent symptom. In 29 per cent, pain in the back of the neck occurred, and was severe in one-quarter of these cases. A "stiff neck" (a mucular soreness with the pain aggravated by motion) was occasionally the first symptom of the disease. Rarely this was so intense as to lead to a suspicion of meningitis (case 4 C).

In both Simpson's and our own series, arthralgia, frequently described as "stiffness," occurred in one-third of the patients, either during the height of the disease process or in convalescence. This was usually very mild—sometimes almost indistinguishable from the general aching. Several of the large joints were usually involved and the associated pain has been "shifting" in character. A hydrarthrosis occurred in only one case.

Abdominal pain was the chief complaint in 15 per cent of Simpson's series. In 7 per cent of the Iowa series it was definite and severe, sometimes continuous and sometimes "cramplike." When mild, it has blended with the general aching, particularly since the localization was inconstant, appearing in some in the epigastrium, in some in

the right lower quadrant, or in almost any region. This symptom must be particularly borne in mind as it has led to erroneous diagnoses and needless, even harmful, surgical procedures.

Gastrointestinal symptoms.—Profound anorexia occurred in severe cases, but this symptom was entirely absent in the mild cases. It was found in three-fourths of our patients and in one-half of Simpson's. It has varied with the degree of fever, so that patients have enjoyed a good breakfast and luncheon yet had no appetite for an evening meal. When normal appetite returned, even though the fever still continued, one could prophesy an early recovery.

Nausea and vomiting occurred in some of the moderately severe infections, but even in such cases were not persistent. Nausea alone was present in 8 per cent and present in association with vomiting in 13 per cent.

Constipation was manifest in one-half to two-thirds of the cases and its degree paralleled the gravity of the infection. A specific diarrhea rarely, if ever, occurred.

Respiratory symptoms.—We have stated above that acute upper respiratory symptoms occurred at the onset of the disease. There was little to suggest that these symptoms were due to specific infection, although possibly pulmonary involvement was. We have gradually become aware of the frequency of a hacking, nonproductive cough. Rarely was it particularly troublesome, and in our earlier cases was attributed to unrelated pharyngeal irritation. More careful records in our later series of 175 cases indicate that more than one-third of the patients had a cough, some with mucoid or muco-purulent sputum. Here we may make mention of two cases diagnosed by consultants as broncho-pneumonia, of one case diagnosed as miliary tuberculosis, and of another in which a pulmonary abscess developed at the end of an infection in which the respiratory symptoms had been prominent throughout. (See Appendix, cases 2 C, 3 C, and 4 D.) In infected guinea pigs definite areas of broncho-pneumonia often occur, and we believe a similar pathological process may be found in man. *Br. melitensis* var. *abortus* has been cultured from tonsils (84) and *Br. melitensis* var. *melitensis* from the sputum (85)—findings which demand careful study of all respiratory symptoms and lesions in these infections.

Neurological symptoms.—Insomnia of varying degree was experienced by 50 per cent of our patients during the height of the disease. A hypersensitive state of the nervous system was manifest by marked restlessness, irritability, or by undue apprehension. Delirium and coma supervened only in very grave infections. Pains, which may be dependent on actual damage to the nervous system, have already been mentioned.

Genitourinary symptoms.—A few patients in our series were first treated as cases of cystitis or pyelitis. Mild symptoms of a localized infection, such as burning, pain on micturition, or frequency, though transient in nature, have occurred in 11 per cent. Difficulty in urination or retention rarely occurred. There was in some cases a definite decrease in urinary output, due presumably to the excessive perspiration.

Cardiovascular symptoms.—Palpitation and the symptoms of an irritable heart have occurred during the course of the disease. These same symptoms, through their long continuance, were in a few instances notable sequelae. Dizziness was at times a complaint early in the course or during the height of the disease. Other cardiovascular symptoms were related to the complications.

Loss of weight.—A progressive loss of weight usually occurred. Emaciation was marked in the severe infections and in those of a prolonged, though mild, nature. Farmers, for instance, who continued to work throughout a two to four months' period of illness, became very much wasted. Bed rest and adequate diet, both in severe and mild infections, largely prevented this. Among our patients who lost 20 pounds or more, one-third were ambulatory and one-third spent more than four weeks in bed. Among those who lost none or less than 10 pounds, one-third were ambulatory and one-third spent four weeks or more in bed. In 10 per cent of the cases there was no apparent loss of weight.

PHYSICAL OBSERVATIONS

Our study of the signs of undulant fever has been somewhat unsatisfactory. Usually we saw the patients once only, and often this was during convalescence. Although they recalled vividly their own symptoms, they knew little or nothing of the associated signs. Practitioners have generously placed at our disposal their observations, but owing to the many and urgent calls of practice, these were often made hurriedly and seldom recorded. Our later data have, however, confirmed the observations already reported, and the findings in Simpson's series and Kern's collected cases are in general agreement. We believe, therefore, that we have a fairly accurate knowledge of the physical findings of infection due to the *abortus* and *suis* varieties.

Signs detected by physical examination.—There was a great variation in the general appearance of those ill with undulant fever. A majority of the patients seen in bed did not appear sick. They were fairly comfortable, mentally alert, and ready to talk. Pallor was frequently noted, and the patients often appeared quite tired. In contrast to these usual cases, however, some patients were obviously

extremely ill, but even these were usually mentally clear and lacked the dullness so characteristic of typhoid fever.

The examination of the head rarely revealed anything significant. The tongue was usually somewhat coated, and a moderate congestion of the throat was not uncommon. About 10 per cent of our cases had the moist and dry rales indicative of bronchitis, while in two of the severe infections with recovery the findings justified the tentative diagnosis of broncho-pneumonia. In uncomplicated infections any abnormality in the cardiovascular system was unusual. A low blood pressure, rarely of marked degree, has been found late in the disease.

Abdominal tenderness was commonly encountered (20 per cent of our cases) and was usually associated with abdominal pain. Occasionally the tenderness was diffuse, but frequently localized in the right upper or lower quadrant, less frequently in the left upper quadrant. The spleen was palpable in one-third of the cases; marked enlargment was rare. It was quite firm and sometimes seemed tender. Occasionally the liver was definitely enlarged.

A skin eruption occurred in 11 per cent of Simpson's cases, and in the same proportion of Kern's series. A general eruption has been noted in only one of the Iowa cases, but physicians have frequently mentioned the observation of scattered maculae which somewhat simulated rose spots.

A localized hyperesthesia has been reported by a few patients which has been found on examination. The lumbar and calf muscles were occasionally quite tender.

The other physical findings which have been noted were those associated with the complications. These, and the findings related to them, will be described later. There has not been found, therefore, any characteristic physical sign of undulant fever. Indeed, an outstanding feature of the disease has been the absence of physical abnormalities. Our question concerning the findings on physical examination usually called forth from the attending physician the answer "I found absolutely nothing." Probably no one thing should so influence a physician to consider *Brucella* infection in differential diagnosis as a fever unexplained by positive physical signs.

Temperature.—Representative curves illustrating the types of temperature in the different varieties of undulant fever are shown in Figure 22. For comparison a curve regarded by Hughes (1) as typical of undulant fever of the Mediterranean region is also included. In infection due to *abortus* and *suis* varieties of *Br. melitensis* such a fever must be very unusual, since as yet we have not encountered a single chart which conformed closely to the type so frequently described. A few of our cases have shown definite undulations with periods of apyrexia, though all have had a rather low-grade fever. Complete temperature records have been available on only a small number of

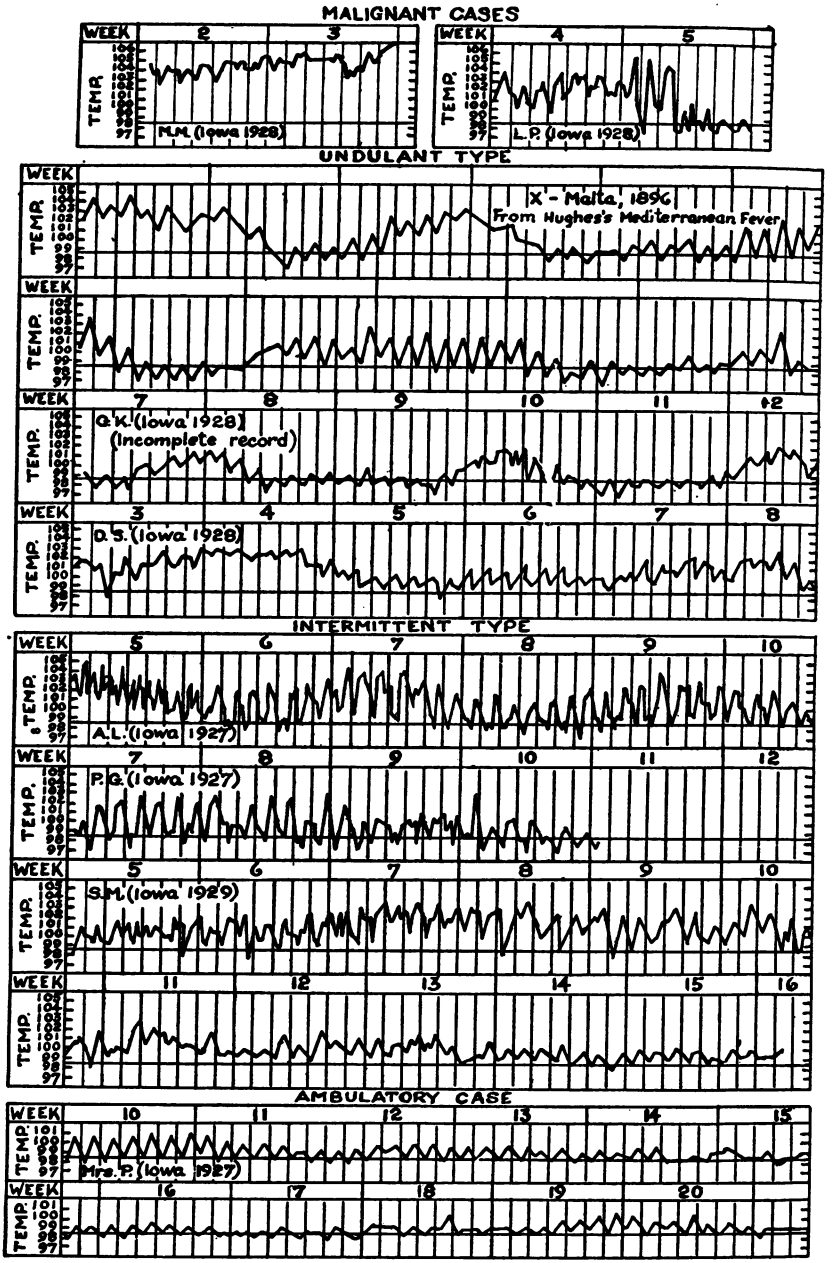

FIGURE 22.—Temperature curves in the different types of undulant fever in Iowa cases, with one chart from Hughes

our cases, but judging from the clinical histories and the available records, we found evidence of suggestive "undulatory pyrexial relapses" in less than 15 per cent, and in these this feature was rarely outstanding. Simpson found undulations of fever in 12 per cent, while in Kern's collected cases they were described in 9 of the 21 cases in which the nature of the temperature curve was stated. The higher proportion in Kern's series is possibly accounted for by the fact that the occurrence of this unusual type of temperature led to the reporting of some of these earlier cases of *Brucella* infections. Very few temperature curves of the malignant type have been observed; only a small percentage had definite undulations. Not uncommonly an intermittent type was followed by one or two relapses, usually of short duration, which came after a few days or even after a period of months of apyrexia. The usual chart showed an intermittent fever, the temperature gradually increasing during the period of invasion and disappearing by a slow lysis. In ambulatory cases, the height of the temperature was variable and was readily increased by overexertion. It was also noted that there were often peaks of fever in convalescence brought on by undue exercise.

There was frequently a wide discrepancy between the degree of fever as registered by the thermometer and the patient's sense of feverishness. It was not uncommon for a patient to apply to his physician without complaint of fever, yet to his own and the physician's surprise he would actually have a temperature of 102°, 103°, or even 104°. Obviously fever occurring without, or with little subjective feverishness might lead to clinical error. The following advice is as appropriate in America to-day as it was in the Mediterranean countries when this statement was made by Hughes more than 35 years ago: "It is always well to take the temperature of a case reporting sick with symptoms of dyspepsia, debility, etc., as a preliminary measure, and if there is any doubt, take it during the afternoon or evening. Fever is often overlooked for want of such precautions, and cases are treated for slight symptoms for some time before the real condition is discovered, to the detriment of the patient's health and the doctor's reputation."

Pulse.—Usually the pulse varied directly with the temperature, but there was no constant relation between these curves. Occasionally an unduly rapid pulse was observed; other cases showed a slow pulse similar to that of typhoid. Frequently the heart rate remained within normal limits.

Urine examination.—The urinalysis frequently revealed the trace of albumin commonly found in febrile diseases. In some, the presence of numerous pus cells indicated either secondary infection or localization of the specific infection in the genito-urinary tract.

Blood examinations.—A secondary anemia usually occurred with the hemoglobin and red blood cells both decreased, the amount of decrease depending directly on the severity and duration of the disease. Variations from normal in the total white blood cell and differential counts were commonly noted, and all observers are in general agreement concerning these variations. A leucopenia was the rule, though rarely of marked degree; a white blood cell count within normal limits was not uncommon; a leucocytosis was very unusual. The differential count usually revealed a decreased neutrophile percentage. The cells accounting for the lymphocytosis were large mononuclears, of which some were pathological forms. The eosinophils and basophils did not show any essential change from the normal. Whether the total and differential counts vary in different periods of the disease has not yet been ascertained.

Duration.—Most patients have found it difficult to tell at just what date recovery from the infection took place. The onset also was insidious; hence one could not accurately determine the total duration of the disease process. However, we have been able to measure the period on 212 of our patients from the time the patient found difficulty in continuing his regular work until he was free from symptoms and able to resume it. The percentage by periods are as follows: One month or less, 19 per cent; 1 month to 10 weeks, 27 per cent; 3 to 4 months, 34 per cent; 5 to 6 months, 11 per cent; more than 6 months, 9 per cent. The average total duration was therefore about 3 months.

Only the few patients who were acutely ill were ever strictly bedfast. Early in the disease and in convalescence patients were up and dressed, resting on a couch perhaps during the afternoon. Even during the fastigium most individuals got up for toilet purposes and in the morning many insisted on sitting in a chair or walking about for a short time. Records of "time in bed" therefore simply estimate the period during which the patient spent most of the time in bed. Those who got up, dressed, and went about, though being forced frequently to lie down to rest, are now regarded as ambulatory. Making determinations in this way, we have found that 9 per cent spent more than 10 weeks in bed; 24 per cent, 1 month to 10 weeks; 33 per cent, 2 weeks to 1 month; 8 per cent, 1 to 2 weeks; and 26 per cent were in bed less than 1 week, or were entirely ambulatory.

COMPLICATIONS

These have been seen so rarely that detailed descriptions are not possible at this time. The following, however, have been observed in *Brucella* infections:

Endocarditis.—The occurrence of endocarditis associated with a *Brucella* bacteraemia, which brought about a fatal termination of a

clinical case of undulant fever, has already been indicated. The failure, after repeated attempts to isolate organisms other than *Brucella* from the blood stream and the absence of corresponding findings of other infections, strongly indicates that endocarditis was due specifically to *Brucella*. This complication, associated in one case with pericarditis, occurred in 1 per cent of our cases.

Arthritis.—We have already pointed out that any detectable hydrarthrosis or swelling of the joints was unusual in our cases, occurring in less than 2 per cent. Tenderness in the region of the joints was not unusual and pain on active motion was a rather frequent complaint. In one reported case (86), the specific nature of the hydrarthrosis has been established by the isolation of *Br. melitensis* var. *abortus*, from the joint fluid.

Bursitis has been described as a complication of *Br. melitensis* var. *melitensis* infection and may, therefore, be expected in undulant fever due to the other varieties of *Brucella*.

Orchitis.—This has been noted in our series in 5 per cent of the males but in one-third of these the symptoms were not severe or the findings marked. Orchitis has appeared during the invasion, in the fastigium, and during convalescence. Its usual duration was two weeks, after which time it completely subsided. Simpson (63) mentioned the occurrence of this complication in a much higher percentage of his cases, though he does not give details. Whether the infection involved only the testis, or the epididymis as well, we have not been able to determine from reported observations.

Mastitis.—In two of our cases (3 per cent of the adult females) a bilateral mastitis occurred as a symptom and sign of onset. Both patients were nonlactating, and the infections, which were of a mild degree, subsided spontaneously after 10 days to 2 weeks.

Abortion.—We have observed but one infection during pregnancy. The condition proceded normally. However, there are reports in the literature of abortion associated with *Brucella* infection. Kristensen (60) isolated *Br. melitensis* var. *abortus* from the placenta in one case. Simpson (63) reported agglutination of *Brucella* antigen by the serum of five women who had no signs of syphilis, but who had repeatedly aborted. Four of these patients gave histories suggesting previous attacks of undulant fever. Prolonged observation will be necessary to determine the frequency of abortion as a complication or sequel of undulant fever.

Miscellaneous.—Associated with our cases, or described in the literature, are the following additional complications: Pulmonary abscess, oophoritis, pyelitis, cystitis, nephritis, seminal-vesiculitis, prostatitis, and broncho-pneumonia. The significance and etiology of these can be determined only by further study.

SEQUELÆ

We have attempted by means of a questionnaire to follow the Iowa cases in order to obtain data in regard to persisting symptoms and sequelæ. Eighty replies have been received. Since this follow-up letter was not sent at a regular period after apparent convalescence, the replies in different cases are scarcely comparable. The striking feature, however, was the prolonged period of disability after the subsidence of fever. This was much more than one would expect following an illness in which there were few acute symptoms. Its occurrence renders the incidence of the infection more serious. In one-half of the cases weakness or easy tiring was the longest persisting symptom. Other continuing symptoms which were mentioned more than once were fever, stiffness or pain of muscles or joints, headache, backache, general aching, anorexia, palpitation, and sweating.

Seventy patients replied that there had been no fever or other illness following the original infection. In one patient there was a persistent and possibly unrelated pyelitis. Two patients with the undulatory type, whom we considered well, had had further recurrences of fever. This was true of seven others who had previously had but one attack of fever. The following answers are taken from this latter group: "For about 12 months following my illness I had recurring spells of fever. These would last from one to three days, and following them I would be tired for several days. The spells were not serious and did not take me from my work." A packing-house employee, after apparent recovery, stated: "I had another illness which resembled in every way undulant fever. There were chills, fever, sweats, aching in joints and muscles, and general prostration." This last period of illness continued for eight days as compared with three months' duration of the first.

Fourteen patients reported that in convalescence there had occurred mild or moderately severe joint pains. This involved, in the order of their frequence, knees, shoulders, ankles, hips, and wrists. In half of the cases one joint only was affected. Swelling or redness was not mentioned in any instance. In some the discomfort was described as a stiffness. This symptom is known to have persisted for more than two months in only three instances. Symptoms which suggested a neuritis were reported by three patients. We have also had reports of a few cases in which mental depression or nervous irritability was a serious and prolonged sequel.

DIAGNOSIS

Undulant fever is an infection having neither a pathognomonic symptom nor sign. Moreover, patients rarely appear dangerously ill; hence the taking of a detailed history and the performance of a

complete physical examination are readily neglected. In addition, practitioners have not been familiar with the nature of this infection, nor are they accustomed to consider it in differential diagnosis. It is for these reasons, we believe, that admittedly erroneous diagnoses have been made in so many of the cases. It has been gratifying, however, to see the accuracy with which undulant fever was diagnosed when once the physician had seen cases or had familiarized himself with the clinical characteristics of the disease.

The importance of laboratory tests in diagnosis has been stated repeatedly. Stitt, for example, says: "Once there is a suspicion of Malta fever, one should try to confirm it by the more accurate method of agglutination tests or blood cultures, rather than from clinical observations." The laboratory procedures which are of value in diagnosis are the agglutination tests, cultural studies, and white blood cell and differential counts. Of these three the one of greatest value in differential diagnosis is the agglutination test. It is usually readily available, often without cost to the patient or physician. It may be urged, therefore, that this be more frequently used in the investigation of febrile illnesses. In those infections with an insidious onset, agglutinins can usually be demonstrated when the patient first applies for medical advice; in those with sudden onset, they may not appear until the end of the second week, or, according to Simpson, (63) occasionally not until the fourth week. Apparently it is true of undulant fever as of typhoid, that infrequently the serum of an infected individual may persistently fail to show any agglutinins. It must always be remembered also that a positive agglutination test may be related to past or subclinical infection, and not to the present ailment of the patient. Blood, urine, and stool cultures are all valuable in the study of suspected cases of undulant fever, but these are only practical in diagnosis when the patient is within easy reach of a laboratory. In this infection any cultural study will consume at least one week, and a negative report on a blood culture can not be made reliable until the end of the third or fourth week. Moreover, negative cultural findings on one examination can scarcely be given any weight. Cultural studies are, therefore, limited in their applicability as a diagnostic test.

The white blood cell findings in undulant fever are in no way peculiar to this infection; still a leucopenia with a lymphocytosis will serve to rule out all but a few conditions with which this disease may be confused.

The skin test is advocated as a valuable diagnostic procedure by Giordano (87), Simpson (63), and others. We have had little experience with it, and have encountered only a few cases in which it might have given additional evidence of value.

DIFFERENTIAL DIAGNOSIS

We have noted in our case records the erroneous diagnosis, provisional diagnosis, or impressions of attending physicians. We found the three most frequent erroneous diagnoses to be typhoid fever, influenza, and tuberculosis. There were also included malaria, pyogenic septicemia, and various respiratory infections (bronchitis, sinusitis, and pneumonia). Appendicitis and cholecystitis accounted for 7 per cent of the erroneous impressions, the former being seriously considered twice as often as the latter. Disease of the cardiovascular system has been diagnosed, including subacute bacterial endocarditis, pericarditis, and hypotension. Infections of the genitourinary system were also in the list, including cystitis, pyelitis, pyonephrosis, orchitis, and epididymitis. Other infrequent impressions were liver abscess, infantile paralysis, spastic colitis, carbon-monoxide poisoning (chronic), tetanus, and such conditions as "nervous breakdown," "liver trouble," and "eye trouble." In none of our cases was acute rheumatic fever or tularemia suspected, but these also may be considered in differential diagnosis.

When the nature of undulant fever is not known and when it is not considered in differential diagnosis, or when immediate complaints or local conditions only are considered, one can readily understand how the above-mentioned clinical impressions may be formed. When the physician, however, is armed with the facts, most of these possible diagnoses can immediately be dismissed. Some, however, may often present difficulty. The differential features of these may be briefly discussed.

Typhoid and paratyphoid fever.—The more rapid onset, the dull, toxic appearance of the patient, the diarrhea and tympanites, the sustained temperature, and absence of sweats usually lead to a correct opinion, while a positive Widal, or the isolation of *B. typhosus* or *B. paratyphosus* establishes the diagnosis. It may here be urged that sporadic cases of typhoid fever are becoming more and more rare, and prolonged fevers, without localizing signs, which occur sporadically, demand a consideration of undulant fever.

Influenza.—About 20 per cent of the cases erroneously diagnosed were called influenza. This is not because undulant fever has any similarity to the acute respiratory infection which occurs in pandemics or epidemics, but because the name is used as an accepted label for all indefinite fevers. We can but advocate a more careful and general consideration of undulant fever and less misuse of the name "influenza" as such, or its corrupted forms "flu," "intestinal flu," and "summer flu."

Tuberculosis.—There may often be a real difficulty in the differential diagnosis of *Brucella* infection and pulmonary tuberculosis. The

insidious onset, weakness, night sweats, anorexia, and loss of weight are common to both, and cough also may be a prominent feature of undulant fever. The chilliness or rigors, the general aching, headache, backache, or arthralgia, the constipation and nervous irritability all point to undulant fever. Laboratory tests usually readily settle the diagnosis.

Malaria.—The regularly repeated rigors which sometimes occur in undulant fever may suggest malaria. A careful history and a resort to the available laboratory tests will establish the diagnosis in either disease.

Pyogenic septicemia.—A leucopenia, or a normal white blood cell count associated with a lymphocytosis, which is ordinarily observed in undulant fever, usually accurately differentiates this disease from pyogenic infections. Cultural studies and agglutination tests may be necessary.

Subacute bacterial endocarditis.—The course of this disease may simulate closely that of undulant fever. The weakness, remitting fever, loss of weight, and anæmia are characteristics common to both. Sweating may occur in cases of subacute bacterial endocarditis. Moreover, in undulant fever there may also be an endocarditis, presumably caused by *Brucella*. When this does occur, the diagnosis may depend wholly upon laboratory studies, blood counts, cultures, and agglutination tests.

Acute rheumatic fever.—We have encountered no case in which this diagnosis had been considered. The striking absence in our cases of any physical abnormality of the joints probably explains this. The arthralgia of undulant fever was often shifting in nature, but the definite swelling or hydrarthrosis, when it did occur, remained localized in the joint or joints attacked. The acute onset and course of rheumatic fever is in striking contrast to the insidious onset and the subacute course of *Brucella* infection.

Tularæmia.—The clinical characteristics of the ulceroglandular, glandular, and oculoglandular types of tularæmia are so striking that a clinical diagnosis of this infection is usually made with ease. However, in the typhoid type, which is of rare occurrence, there may be confusion with undulant fever. Moreover, in differentiating these infections the agglutination test may be misleading, owing to the phenomenon of cross agglutination. *Brucella* antigens may be agglutinated in diagnostic titers by the serum of tularæmia patients. Hence if in that infection a test is performed for undulant fever only, agglutination of *Brucella* may lead to an erroneous diagnosis. If there has been any history of a possible exposure to *B. tularense*, agglutination tests for this as well as undulant fever should be requested. The agglutination results are usually conclusive.

Appendicitis and cholecystitis.—Fever, abdominal pain, and localized tenderness are the misleading features. When generalized infection is not considered these clinical findings may seem to be best explained by a chronic or subacute appendicitis or cholecystitis. Simpson (63) has a record of 12 appendectomies and 2 cholecystectomies which were performed on cases of undulant fever. The pathological examination revealed no evidence of inflammatory process in the organs removed. In the cases of this nature which we have observed, we have felt that appendectomy was too readily advised, and that a careful history with a complete physical examination, supplemented by blood counts, would have left no reason for surgical intervention. We have, however, seen one case with a retrocecal appendix, in which an appendicitis continued to perforation and peritonitis through hesitation, occasioned by a weekly positive agglutination reported for undulant fever. In two of our cases in which the diagnosis of cholecystitis was seriously considered, undulant fever, when called to mind, was almost at once accepted as a provisional diagnosis.

Infections of the genito-urinary tract.—Frequency, painful micturition, and pus in the urine are not uncommon features of *Brucella* infection. These may or may not be specifically related to the disease. Since these conditions have occurred late in the invasive period or during the fastigium, a careful history will usually lead to suspicion of a generalized infection with local manifestations. This is also true of orchitis when it is the major complaint.

PROGNOSIS

There has been a case fatality of 3 per cent in our cases. Deaths have occurred in infections beginning as the ambulatory type, as well as among those of the malignant variety. The duration of the infection has been variable and can not be predicted. It is apparent, therefore, that prognosis must be somewhat guarded. Particularly is this so in infections known or believed to be caused by the *suis* or porcine variety of *Br. melitensis*. On the other hand, we have found it safe to give a fair prognosis in cases which could be attributed to the *abortus* (or bovine) variety of *Br. melitensis*.

THERAPY

We can find no record of a properly controlled systematic investigation of therapy. One may find different specifics recommended, usually because of the uneventful recovery of only a few treated cases. The natural course of the infection due to the more recently discovered varieties of *Br. melitensis* is only now becoming known. Physicians too generally have assumed that it was identical with the prolonged and distressing infections frequently seen in the Mediterranean region,

and commonly described in current texts and systems. Having learned the natural course of infection with the *abortus* and *suis* varieties we are now prepared to better evaluate the different therapeutic procedures recommended. Conclusive data will only be obtained, however, by the observation of an adequate series of treated cases, and their comparison with a corresponding number of untreated ones. We feel that a study of the various specifics which have been recommended is highly important.

Available therapeutic procedures of proven value and of first importance are rest, liberal diet, adequate fluids, and appropriate measures for the alleviation of prominent symptoms. Exercise in convalescence should be followed closely by temperature records, and so modified that elevations of fever above 100° F. are prevented.

Among the therapeutic procedures of unproved value, the one most commonly recommended is the use of specific vaccine. Attention was first called to this means of treatment of undulant fever by Angle (88), who reported its successful use in 10 cases. Simpson reported the results in a larger series, as follows: "In 46 of our cases we have utilized the vaccine made of heat-killed *Br. abortus*, standardized to two billion per cubic centimeter, with such an apparently favorable result that we are now employing it as a routine treatment. The vaccine is given by deep subcutaneous injections. The usual dosage has been one-fourth cubic centimeter for three injections, followed by one-half cubic centimeter for three injections, followed by 1-cubic centimeter doses, all at 3-day intervals. The first one or two injections have been followed by a mild or moderately severe general reaction in two-thirds of our cases, following which the reaction has diminished in intensity after each succeeding vaccination. In several instances the site of injection remains indurated for many days. No necrosis or abscesses developed. Following the first two or three injections the fever usually approaches the normal level and the symptoms abate. As a general rule those patients who experience the most marked general reaction had a most rapid favorable response to the vaccine. It is hoped that other workers will give the abortus vaccine a thorough clinical trial, and report their observations. Due caution must be exercised in the evaluation of any therapeutic measure in a disease characterized by natural remissions." While this measure has not been used in a large number of the Iowa cases, we have observed rapid recovery following administration of the vaccine, but have also seen other cases whose infections continued unmodified by the same treatment.

More extensive studies have been carried out in the therapeutic value of vaccine in *Br. melitensis* var. *melitensis* infection. The opinions differ as to its value. Favorable reports are made by Bassett-Smith (89), Owen and Newham (90), Guiffre (91), DeFinis (92),

and others. Arloing (93) observed an Arthus phenomenon following its use. Alfred Coury (94) considers it dangerous while other observers regard it as of unknown value.

In three cases of *Brucella* infection Awe and Palmer (95) report recovery following the injection of nonspecific protein. Simpson (63), however, has found this to be of no appreciable value. Mercurochrome has its advocates, though Ross and Martin (96) consider that in *Br. melitensis* var. *melitensis* infection its value has not been demonstrated. Acriflavine, trypaflavine, colloidal metals, and neoarsphenamine have all been recommended, but the very length of the list shows clearly that the value of any drug, thus far advocated, has not been satisfactorily demonstrated.

UNDULANT FEVER OF BOVINE OR PORCINE ORIGIN COMPARED WITH THAT OF CAPRINE ORIGIN

The following descriptions of undulant fever of caprine origin have been taken from current medical texts: "A specific fever caused by the *Micrococcus melitensis* characterized by undulatory pyrexial relapses, profuse sweats, arthritis and an enlarged spleen" (Osler and McCrae, 1925). "A specific infectious disease * * * having a fever of indefinite duration running an irregular course and made up of a series of waves of pyrexia * * * giving rise to symptoms of toxic septicemia, with enlargement of the spleen, sweats, constipation, effusion about the joints, and pains; sometimes with endocarditis and orchitis; later associated with great anemia, multiple neuritis and hectic fever. Convalescence is tedious and mortality low" (Bassett-Smith in The Practice of Medicine in the Tropics; Byam and Archibald, 1922). One finds the following description by Hughes quoted repeatedly: "Clinically the fever has a peculiar irregular temperature curve, consisting of intermittent waves or undulations of pyrexia, of a distinctly intermittent character. These pyrexial waves or undulations last, as a rule, from one to three weeks, with an apyrexial interval lasting for two or more days. In rare cases the remission may become so marked as to give an almost intermittent character to the febrile curve * * *. Its course is often irregular and even erratic in nature. The pyrexia is usually accompanied by obstinate constipation, progressive anemia, and debility. It is often complicated with and followed by neuralgic symptoms referred to the peripheral or central nervous system, arthritic effusion * * * or swelling of the testes."

Although one finds mentioned here no features which are not observed in infection with the *abortus* or *suis* varieties of *Br. melitensis*, still there are not accurate descriptions of the latter disease. The one feature which overshadows all others in the description of undulant fever of caprine origin is the undulatory type of temperature, a

rare finding in that of bovine or porcine origin. Moreover, the effusion into the joints must have been much more common than we have observed, and the neuritis more severe. On the other hand, in our cases rigors were noteworthy but these are rarely mentioned in the descriptions of the *melitensis* variety of infections. In other respects, however, the symptoms and signs differ not at all, or only in degree. In the disease due to the *melitensis*, as well as the *abortus* and *suis* varieties of *Brucella*, premature births and abortions have been noted. The picture of undulant fever, therefore, in order to include the infections with the *abortus* and *suis* varieties, demands the placing in the background of the "undulatory pyrexial relapses," the effusion into the joints and the neuritis, and also a softening of all symptoms save possibly the rigors which should stand out more prominently. Viewing this another way, we see that in undulant fever of caprine origin the undulatory type predominates; in that of bovine or porcine it is the intermittent type that is the most common. A true description of undulant fever may only be written when we picture equal numbers of these two types, with a few of the malignant and a few more of the ambulatory varieties included.

VII. PREVENTION

In considering the prevention of *Brucella* infections in human beings and animals, due consideration must be given to the possibility of immunization. Is the injection of an adequate number of killed organisms as effective in this disease as it appears to be in typhoid fever? Experimental evidence justifies little hope in such procedure. We, as well as others, have tested a fairly large series of guinea pigs as described. Those injected with three doses of heat-killed *Br. melitensis* var. *abortus*, and which developed agglutinins in a titer of 1:80 or higher, seemed to become infected as readily as did untreated animals, when exposed by the skin route. Moreover, neither the course of the disease nor the pathologic lesions were significantly different in the treated guinea pigs and the controls. The injection of killed organisms, therefore, does not promise to be an effective prophylactic measure.

A second question of fundamental importance is the thermal death point of *Br. melitensis* var. *abortus* and var. *suis*. The recent report by Arnold (97), who found living *Brucella* after exposure to the temperature of commercial pasteurization, has demanded a reconsideration of this subject. These findings are in contrast to those of Carpenter and Boak (98), who reported all organisms to be killed after 20 minutes at 140° F. In our own tests we have not found living organisms following exposure to temperatures of 144° to 145° F. for 30 minutes, followed by rapid cooling in the ice box. Organisms were, however, cultured in one test in which the temperature was

slightly inconstant, fluctuating between 139° and 142° F. These laboratory data and our epidemiological findings demonstrate that controlled pasteurization is effective against organisms of the *Brucella* group.

At present effective control measures are those designed to prevent human contact with virulent organisms which leads to the infection of susceptible individuals. It would appear that work along this line might be best carried out by bearing in mind the chief modes of transmission of the disease, namely, ingestion of raw dairy products and contact with infected animals; also to remember that the portal of entry into the human body may be either the mouth or the skin.

MEASURES DEALING WITH THE PROBLEM AS A WHOLE

Eradication of contagious abortion in cows and hogs.—The significance of contagious abortion in its relationship to disease in man equals, if it does not exceed, bovine tuberculosis, and is in like manner primarily a problem of the veterinary medical profession. In the prevention of undulant fever, as in no other disease, members of the human and veterinary medical professions are called upon to unite forces in a common attack.

Agglutination tests.—Those familiar with the epidemiology of this disease are impressed with the advisability (preferably required by law) of routine agglutination tests to determine the presence or absence of infection in animals.

Bacteriological studies needed.—It is apparent that further intensive studies need to be conducted to determine the presence, variety, and pathogenicity of strains of *Brucella* in cow's milk, cream, and raw dairy butter. Further work is likewise indicated to determine the distribution of porcine organisms in the various organs, tissues, and discharges of actively infected hogs. More accurate knowledge of these might throw much light on the means of avoiding exposure and hence of preventing infection.

Health education.—Health education should be conservative and guarded, but it is essential that accurate information be conveyed so that the groups concerned may more intelligently practice preventive measures. The public should be so taught that people will demand safe dairy products, without diminishing the consumption of the same; the stock breeders so that they will work for healthier and more productive animals.

MEASURES DESIGNED TO PREVENT TRANSMISSION THROUGH RAW DAIRY PRODUCTS

On the farm.—It should be a relatively simple matter, particularly when families are supplied with the milk of but one or a few cows, to have these tested serologically. It is apparent—considering dosage,

repeated exposure and absence of the dilution factor—how potent are the possibilities of infection if the disease occurs in a small herd. It is encouraging that, by means of agglutination tests associated with bacterial studies of milk where practicable, and with the isolation or elimination of known infected animals, the transmission of this infection may be controlled without prohibitive expense.

In cities and towns under 5,000 population.—It would seem advisable to require that all dairy cows pass satisfactorily an agglutination test before such milk be distributed in raw form. The wider use of properly pasteurized dairy products ought to be encouraged in every way possible.

In cities over 5,000 population.—Measures applying to smaller cities are indicated also in the larger centers. Adoption and enforcement of a standard milk ordinance, including the requirement that the contagious abortion test be applied to all dairy cows and that dairy products be pasteurized, is a consummation much to be desired. Undulant fever is only one more condition added to the already formidable list of diseases transmitted from time to time through the use of raw milk and cream.

Municipal and county health departments.—These are, wherever established, an effective means of carrying out the above-mentioned measures.

MEASURES DESIGNED TO PREVENT INFECTION THROUGH CONTACT

On the farm.—Precautions here are largely of a prophylactic nature and consist in the avoidance, to as great a degree as possible, of what may be termed special types of contact. These occur in such procedures as vaccinating, ringing, or castrating hogs, in the handling of new-born pigs and in loading or unloading hogs for market. It is felt that such direct forms of hog contact are significant, and that the chances of infection might be materially reduced through appropriate measures, such as wearing of heavy gloves whenever such contact is necessary. Many of these special types of contact might be avoided if farmers were taught to appreciate their significance. Farmers will do well also to avoid the direct contact with infected bovine tissues incident to manual removal of placentae.

Others having direct contact with livestock.—Livestock dealers represent another group exposed in special ways through direct contact. A growing knowledge, on their part, of the intimate relationship existing between contagious abortion in animals and undulant fever in man should reduce to a minimum all but casual or relatively insignificant contacts with hogs. Veterinarians as a group have the most direct types of contact with cows. Their remarkable freedom from active undulant fever infection probably indicates that cattle contact is relatively less significant than hog contact and the practice of

aseptic precautions is no doubt a factor in lessening exposure to infection in members of the veterinary medical profession. The possibility of an immunity being acquired by this group can not be set aside.

Packing-house workers.—No other group is exposed to this infection through direct contact in any way comparable to packing-house workers, who handle the naked tissues of large numbers of infected animals brought together from wide geographical areas. It is felt that contact infection in employees on the killing floor may be reduced by giving more prompt attention to the care of minor knife wounds or cuts. Such workers might possibly be assigned less hazardous employment during the healing of wounds. Protection with gloves might be of some value. Effective measures need to be adopted in this group of persons, to materially decrease the hazard of undulant fever infection.

VIII. APPENDIX

A. EPIDEMIOLOGICAL CASE RECORDS

Case 1 A.—E. B., female, aged 45, housewife, living in a city of 50,000 population. Had no significant travel, and no direct or indirect contact with livestock. No pets were kept. Raw milk and cream, purchased at a small grocery store, were used by the family. The patient used milk freely with cereal and fruits, but drank not more than one glass daily. Creamery butter only was used. Of the 20 cows in the herd supplying the milk used by this family, 4 were negative, 6 gave reactions in titers of 1:40 and 1:80 only, and the remaining animals were definitely positive.

Case 2 A.—W. B., male, aged 26, single, stone-quarry worker, living in the country. He had taken local trips only. The family kept one cow—the only livestock on the place—but the patient rarely had direct contact with this animal. He drank little milk, but used cream and home-churned butter very freely. The patient's mother, during the previous year, had had tularaemia, and primarily to determine if agglutinins for *B. tularense* still persisted, a specimen of her blood was taken. She made no complaint, but remarked that for a few days during her son's illness she had noticed some lassitude and weakness, but did not consider herself ill. Her serum titer for *Br. melitensis* var. *abortus* was 1:1,280; for *B. tularense* 1:160.

Here was a mild infection, accidentally detected. The one cow was examined and showed a serum agglutination of *Br. melitensis* var. *abortus* in a titer of 1:80. Milk was also obtained, and the whey agglutinated in a dilution of 1:20, a titer so low as to be ordinarily disregarded. The cream was injected into guinea pigs, and *Br. melitensis* var. *abortus* was isolated.

Case 3 A.—G. F. C., male, aged 46, physician. Recently had taken short local trips only. Had no direct contact with livestock. Used raw milk very freely, from 1 to 3 quarts daily, supplied by a distributor who purchased from several different farmers. An examination of the herds involved was not attempted.

Case 4 A.—H. P., male, aged 50, farmer and hog raiser. Had not traveled recently. On his farm he had assumed the responsibility for all work around the hogs. He used raw milk and cream freely, obtained from his own cows. There was no history of contagious abortion among the cattle, and he "always had good luck with the hogs." Blood specimens were obtained first from the cows, but no reactors were found. The source of the patient's infection remained undetermined. Three months later, during convalescence, his son, aged 20, became ill

and was also found to have undulant fever. A second visit was made and it was learned that during the father's illness this son had cared for the hogs. Careful questioning then elicited the history that one sow had been known to lose her young, but this was considered accidental. There had also been some trouble with sterility. The hogs were examined, and of the 19 tested, all sera, except 3, agglutinated *Brucella* in diagnostic dilutions.

Case 5 A.—H. L., male, aged 40, farmer. Used raw milk, cream, and homemade butter freely. One of his 10 cows had aborted during the preceding year and remained sterile, and another was also considered as sterile. Among the hogs there was no history in any way suggestive of *Brucella* infection. Serological findings revealed no reactors among the cattle, but of the hogs examined three were positive, four doubtful, and eight negative. The farmer had had the usual contacts with his stock. From his blood stream a *Br. melitensis* var. *suis* organism was isolated.

Case 6 A.—G. W., male, aged 52, farmer. Except for short business trips to a near-by town, the patient had been at home. He drank milk very freely, and used cream and homemade butter in liberal quantities. One of his four cows had aborted and the milk from this animal was used with that from the others. His sows had all raised good litters and his ewes had given birth to a normal number of young. The serological findings were as follows: Cows, 1 positive, 3 negative; hogs, 18 negative; sheep, 11 negative, 1 doubtful.

Case 7 A.—A. W., female, farmer's wife. She had frequent and direct contact with the cows, but only indirect contact with the hogs. Used milk and a small amount of cream with coffee and cereal. Creamery butter was puchased. Among the six cows there had been two abortions and one of these had been followed by sterility. There had been no known abortion among the hogs and litters of good size had been raised. Laboratory examinations revealed as follows: Cattle, 1 positive, 7 negative; hogs, 11 positive, 5 doubtful, 1 negative.

Case 8 A.—A. F., male, aged 22, packing-house employee. He had always lived in a city of 30,000 population, and had no direct contact with livestock. He used no fresh dairy products, but supplied himself with condensed milk and oleomargarine. In the packing plant he worked in the hog division, and his duty consisted in trimming the fat from off the sigmoid near the rectum. Fecal contamination was frequent. Cuts or scratches on his hands were infrequent. *Br. melitensis* var. *suis* was isolated from his blood.

Case 9 A.—P. M., male, aged 16, farmer's son and high-school pupil. Had not traveled. Never drank milk and rarely used cream. Creamery butter was purchased. There were four cows on the farm; all were apparently healthy and were serologically negative. Of 17 sows, only 5 raised pigs; the others had aborted. Prior to the onset of illness the patient was attending school and had no direct contact with the hogs. There was on the farm a bitch that had aborted four months previously, but later gave birth to a litter of normal pups. This animal was the boy's particular pet, and in play contact was intimate. We obtained the animal for study. Repeated serological tests were positive, although bacteriological findings were negative. The farmer volunteered the information that the sows after abortion had a persistent vaginal discharge, and the bitch was often seen following the animals and licking the discharge.

Case 10 A.—The R. family. In July, 1929, Mr. R. contracted undulant fever, from which he was recovering when seen in October. He used all dairy products very freely. Among his cattle and hogs the only history suggestive of *Brucella* infection was the case of one cow which had had a retained placenta and was later sold because of sterility. Another cow, still on the farm, was also possibly sterile. Serological examination revealed: Cows, 1 positive, 2 doubtful, 3 negative; hogs, 3 positive, 2 doubtful, 9 negative.

During August this patient's son, aged 10, became ill with symptoms very characteristic of undulant fever. No blood specimen was sent at that time. After five weeks he rapidly convalesced. A blood specimen, taken in October, failed to show agglutinins, but clinically the case had been undulant fever. This lad had had some contact with both the cattle and hogs and used milk and cream freely. Cautioned regarding the possible danger in the use of raw milk, the family ceased drinking it unless it was scalded. However, they continued to use raw cream, and from it made butter for their own use. In December, Mrs. R. contracted the disease. From her blood *Br. melitensis* var. *suis* was isolated. She used very little cream or butter and had had no contact with the livestock, but handled and churned the cream. The test of milk from the one reacting cow failed to reveal *Brucella*.

Case 11 A.—The H. family. One positive blood specimen had been received from a physician serving a rural clientele. An investigation was made later, at a convenient time. That there had been an exceptional occurrence of the infection was apparent, when the physician said that he thought there had been two or three other cases in the farm household. The family consisted of intelligent parents with six children, ranging in age from 2 to 8 years. During the previous five months the husband had had typical undulant fever; two children had had febrile illnesses of six and four weeks' duration, with the symptoms and findings of the same infection; one had had a prolonged atypical attack, while two other children and the mother had had an ailment, with fever, of short duration only. The oldest child had apparently been well throughout. Blood was obtained from all; from four of the children only a dried blood specimen could be collected. The four microscopic tests showed titers of 1:160, 1:320, 1:320, and 1:640. Two of the microscopic tests showed small clumps in the 1:40 and 1:80 dilutions—a reaction which we regard as suspicious. The findings justified the opinion that at least six, probably all, of the family had been infected.

There was no history even suggesting contagious abortion among the cattle, though 2 of the 11 cows were serologically positive. Three years previously several of the sows had aborted, but none since that time. Of the 10 examined serologically only 1 showed a titer as high as 1:40. Milk shipped to us from the two reacting cows failed to reveal *Brucella* by guinea-pig injection. The true explanation of the occurrence of infection in this family is therefore quite uncertain.

B. CLINICAL TYPES

INTERMITTENT TYPE

Case 1 B.—E. B., male, aged 40, farmer and hog raiser. Late in December, 1926, he noted that he was unusually tired in the evening and that his appetite was somewhat impaired. During January, 1927, the weakness increased. There were sleeplessness, more marked anorexia, occasional feverishness, and irregular night sweats. He was also troubled at times by backache, and complained of a stiffness of the neck. Symptoms progressively became worse. Early in February he consulted his physician and an elevation of temperature was found. Pyorrhea was noted, but otherwise the physical examination was negative. Dental examination, with X ray, was advised. Several apical abscesses were revealed. The teeth concerned were extracted. One week later the physician was called to the patient's home. He found an obviously ill man, with a moderately high fever, and distressing joint pains. A pyogenic septicemia was considered and a blood culture was taken. Blood for a Widal test was sent to the State hygienic laboratory, to rule out typhoid, and this, examined routinely for undulant fever, was found to agglutinate *Br. melitensis* var. *abortus* in a 1:320 dilution. Two later tests were also positive. The blood culture was dis-

carded after 72 hours' incubation, at which time the subcultures showed no growth. Throughout February and March the patient was bedfast. So profuse were the night sweats that quite regularly the bed linen would have to be changed between 1 and 2 a. m. His wife, who nursed him, reported that through his illness he was restless and quite irritable. There was a gradual loss of weight. The fever was somewhat irregular, varying from normal to 101° in the morning and 101° to 103° in the evening. An unusual feature of this case was a definite arthritis, with effusion into the knee joints. There was an uneventful convalescence which covered a period of two months, following which the patient gradually returned to work. Twenty months later he reported that he had been enjoying good health.

Case 2 B.—A. F., male, aged 38, farmer. About the middle of August, 1929, patient noted that in the evening he would be unusually tired, lacking appetite, and frequently complaining of headache. These symptoms persisted, and one month later he consulted an oculist. Lenses were prescribed, but the frontal headache persisted. He then consulted his physician, who accurately diagnosed his ailment. Two blood serum tests showed agglutination for *Br. melitensis* var. *abortus* in 1:2560 dilution. Early in November, when we saw him, he was still ambulatory. His chief complaints at that time were profuse night sweats, rigors, of which he had had 10, and diffuse lower abdominal pain. He had moderate backache, some joint discomfort described as stiffness, and constipation. At noon we found his temperature to be 102.5° (unusually high for that hour, he explained, since he had had to do quite strenuous work that morning). Ordinarily, the temperature was normal until noon, reaching a maximum of 102° to 105° in the early evening.

The patient's own disability and our advice were at first not sufficient wholly to restrict his activity. He became strictly bedfast, only when a unilateral orchitis and epididymitis developed a few days later. Throughout October the patient continued to be quite ill. During November he improved rapidly, but during this month there developed a condition which was diagnosed as teno-synovitis of the right hand. At the end of the month there was still a low-grade fever and moderate weakness. The appetite was very good, and weight was being regained. By January the patient considered that he had fully recovered.

AMBULATORY TYPE

Case 3 B.—B. N., male, aged 38, farmer. There was an insidious onset during April, 1929. During the first six weeks of illness the patient thought he had "chronic flu". Because of the persistence of the symptoms, he applied to his physician at the end of this period. He reported that he had a moderate weakness, though in the morning he ordinarily felt fairly strong, but in the afternoon he was able to do little. He complained of general aching, some headache, chiefly behind the eyes, but also in the lower occipital region, and back of the neck. He had no definite joint pains, but complained of muscular soreness and stiffness. "I could scarcely move," was his own description. Night sweats had occurred, but these were not profuse. These symptoms varied somewhat in severity and persisted throughout the summer. His appetite was never good, and he was troubled with constipation. More than 20 pounds of weight were lost. A nonproductive cough persisted throughout the illness. The fever occurred only in the afternoon and evening and was rarely above 101°, but reached a maximum of 103°. This patient also noted that the more active his exercise, the higher the temperature rose. Except for the coarse râles, and a moderate tenderness in the upper abdomen, the physician reported no abnormal physical findings. Laboratory tests showed a blood serum agglutination for *Br. melitensis* var. *abortus* in 1:320 dilution in June, and in 1:80 in November.

Throughout his illness of seven months' duration, the patient continued to do the necessary work on his farm. He obtained extra help only during the more strenuous season of harvest and threshing.

Case 4 B.—T. T., male, aged 13, doctor's son. This illness began insidiously during October, 1928. The lad's parents first noted that he was less eager to play in the afternoon and was not interested in the evening meal. The boy complained of some headache, which he described as pain in the eyes and back of the neck. An evening temperature of 99.5° to 100° was found. There were no other abnormal physical findings. The father restricted the boy's activity for one week, but throughout the remainder of the mild illness, which lasted one month, he lived normally. His blood serum gave an agglutination for *Br. melitensis* var. *abortus* in 1:160 dilution.

UNDULATORY TYPE

Case 5 B.—G. K., male, aged 43, farmer. Patient's illness began about December 15, 1928. He noted marked weakness, a moderate anorexia, general aching, particularly in the lumbar and cervical regions, and some fever. By January 1 he felt that he had recovered from an attack of "la grippe." Shortly after the new year the same symptoms reappeared, this time more severe, and the physician was consulted for the first time. During January and February he had at least four attacks of fever, with apyrexial intervals, in which he did not feel ill. His case was diagnosed as typhoid and typhoid flu, but on analysis of a blood specimen sent to the laboratory, by the third physician consulted, the diagnosis of undulant fever was established. The titer found was 1:1280. On March 1 the patient was admitted to the university hospital during an apyrexial period. Five days after admission the fever reappeared and increased daily for four days, reaching a maximum of 101.8°. Following this it gradually subsided, reaching normal four days later. During this febrile period patient's only complaint was constipation. He noted some feverishness, did not enjoy reading, and was less interested in his food. He did not, however, appear ill and no notable physical abnormality was detected throughout the illness. On March 15 the patient was discharged, and from that time his progress was followed by correspondence. Letters of March 24 and April 25 reported recurrences of fever. The note of the latter date, after describing the early symptoms of these attacks, read as follows: "I had almost forgotten how I did feel when I was sick, but it all came back, the ache and pain in my limbs, the headache and backache, the soreness across my bowels, and constipation. I didn't have any chill this time, but my fever broke about 2 a. m. to-day, and I sure did sweat." The patient considered that his symptoms were most severe during what proved to be his last febrile period. A later letter, on June 1, stated that he had no fever, but was still weak, even though doing light work.

Case 6 B.—M. C., male, aged 38, farmer. This patient was unable to give any date of onset, but stated that during the spring months of 1927 he noted that he tired easily and had headache which gradually became more frequent and severe. Early in June he first consulted his physician, making an office call when he had a temperature of 103.2°. His symptoms at that time were marked weakness, profuse night sweats, rigors, anorexia, and constipation. Shortly after this the patient came to the hospital. His temperature was found to be remitting, normal or about normal in the morning, and 102° to 103° in the evening. The physical examination was negative except for slight abdominal tenderness. The agglutination test was positive for undulant fever. The patient did not consider himself sufficiently ill to remain in hospital, and after 10 days insisted on being discharged. The fever gradually subsided and with this his symptoms disappeared. Two relapses occurred, the first after six weeks, with a duration of two

weeks, the second after four months lasting one week only. During the relapses his symptoms were mild in degree though similar in nature to those in the original attack.

MALIGNANT TYPE

Case 7 B.—L. P., male, aged 26, a packing-house employee and laborer. Serum agglutinated *Br. melitensis* var. *abortus*, in diagnostic dilution. The onset of his acute illness was preceded by a definite complaint for a period of almost one month, of lassitude, headaches, and drowsiness. During this time he continued at work. For three days before a physician was called the patient was quite ill. During that period he had marked prostration, complained of some headache and backache, and an acute pain in the back of the neck. The physician reported that the temperature at first was regularly remitting, but observation showed an increase by daily additions of one degree until 104° was reached. It was then sustained for 10 days at a high level. During this period the patient was acutely ill. Early in his acute illness he had one rigor. Throughout he had marked constipation. Delirium and coma rapidly developed. A fatal outcome seemed certain. Unusual in the course of this illness was the rapid enlargement of the spleen. At the time of the first consultation the physician reported that the organ was not palpable. Four days later it could just be felt, and one week after this its lower margin had reached the umbilicus. After the 10 days with high fever the temperature dropped almost by crisis. The spleen decreased in size as rapidly as it had increased, and convalescence proceeded uneventfully.

C. ATYPICAL CASES

Case 1 C.—B. H., male, aged 15, a packing-house employee. During the period of invasion patient felt weak and able to work only part time. His physician was called after one week, at which time the history revealed weakness, gradually increasing fever, epistaxis on four occasions, spells of chilliness, two rigors, considerable abdominal pain, anorexia, and headache. The temperature was found to be 104°, the pulse 90, and respiration 25. The patient appeared dull and drowsy. During the following days the abdominal discomfort persisted. There was moderate tympanites, with intermittent diarrhea and constipation. There was some cough with slight evidence of bronchitis and sinusitis. Sweating did not occur. During the third week of observation the patient was very toxic and delirious. The temperature throughout ranged from 104° to 105°, with only slight morning remissions. The pulse was not rapid. Four white blood counts read as follows: 5,000, 5,600, 4,250, and 3,800. The temperature had returned to normal one month from the date that the physician was first called. The serological studies gave the following results: Widal tests were persistently negative and on the four different occasions when *Br. melitensis* var. *abortus* was agglutinated, the titers were 1: 1,280 and 1: 2,560. A blood culture yielded *Br. melitensis* var. *suis*. After a period of 10 days without fever, the patient suffered a relapse and, though the temperature reached 103°, he was much less dull and toxic, presenting the usual picture of undulant fever.

The attending physician and others examining this case agreed that without laboratory findings this case would certainly have been diagnosed as typhoid fever.

Case 2 C.—F. B., male, aged 26, farmer. There was no history of tuberculosis in the family. The onset was very insidious, the patient stating that he "had not been feeling fit all fall." In January, 1930, he first consulted his physician, at which time his major complaints were progressively increasing weakness, fever, cough, and night sweats. Additional inquiry revealed that he had also spells of chilliness, two rigors, severe pain in the back of the neck, anorexia, moderate

irritability, sleeplessness, and very profuse sweats. He had also lost weight. During the following months, the condition which was not definitely diagnosed did not improve and the family requested a consultation. A diagnosis of tuberculosis in its worst form was made; the patient, his wife, and parents were acutely distressed by the outcome, but acting on the advice of the consultant, the young man prepared to sell his farm, stock, and equipment. The family physician had, however, after long delay sent us a blood specimen and this we found to agglutinate *Br. melitensis* var. *abortus* in a serum dilution of 1:640. This young man came to the university hospital for further study. His cough persisted, and he had mucoid or mucopurulent sputum. Moist râles, diffusely scattered, were heard chiefly at the bases, posteriorly. The spleen was easily palpable. X ray of the chest was entirely negative. The patient looked well, and after a short rest in bed felt so well that he could not be persuaded to remain in the hospital.

The prominent features of this case are the symptoms and findings of pulmonary tuberculosis, but the rigors, pain in the back of the neck, and the palpable spleen make such a clinical diagnosis questionable. The course of the infection supports the diagnosis indicated by the laboratory findings.

Case 3 C.—M. F., male, aged 40, preacher. The patient became suddenly ill, feeling weak and somewhat feverish. His temperature was taken and found to be elevated and a physician was called. Early in the infection a rigor occurred, and from the first he had a cough. Only later did general aching appear. Sweating was never marked. Physical examination at first was essentially negative. A few days later, auscultation revealed moist râles over the bases, posteriorly. His physician detected slight dullness on percussion, and a suggestion of bronchial breathing. Pneumonia was suspected. Considering undulant fever also, blood was sent to the laboratory and the first specimen agglutinated *Br. melitensis* var. *abortus* in a 1:640 dilution; the second, four weeks later, in 1:5120 dilution. Two blood cultures remained sterile, but by guinea pig inoculation *Br. melitensis* var. *suis* was isolated from the feces.

Case 4 C.—O. I., male, aged 35, farmer. The onset was sudden, with headache, followed by a rapid rise in temperature. The prominent symptoms at first were occipital headache and severe pain in the back of the neck, particularly on the right side and associated with some local muscular twitching. Because of the acute pain, patient was unable to sleep. He had almost complete anorexia. His early temperature was high and sustained; later it was remittent. Meningitis was suspected, and he was admitted to hospital. Spinal fluid showed eight cells per cubic millimeter. The white blood count was 13,000, with an increase in the polymorphonuclears. The liver was found to be enlarged, and the spleen easily palpable. Less than a week later very profuse night sweats appeared, and the white blood cell count returned to normal. The total duration of illness was less than four weeks. During the course of the disease two agglutination tests showed titers for *Br. melitensis* var. *abortus* of 1:320 and 1:640, while early in convalescence the titer had decreased to 1:160.

Case 5 C.—M. P., male, aged 53, farmer. This patient applied to his physician, complaining of frequency of urination and pain in urinating. During the 10 days in which this condition persisted he was treated as a case of cystitis. Later inquiry revealed that for more than three months he had been ailing, having noted progressively increasing weakness, aching particularly in the legs and loss of weight. His later course was that of an intermittent type of undulant fever, but he did have an orchitis and epididymitis as a complication. The diagnosis of *Brucella* infection was supported by laboratory findings, since on two occasions his blood serum showed specific agglutination in 1:320 dilution.

Case 6 C.—T. D. T., male, aged 39, farmer. This patient applied for medical treatment, complaining of pain in the back, which was most severe on first motion

after a period of rest. He complained of pain in one ankle. He was treated for "rheumatism." Later inquiry obtained a history of moderate weakness, irregular but profuse night sweats, and some headache. During the patient's illness his daughter developed fever, and only after her case had been diagnosed as undulant fever was the father suspected of having the same ailment. This suspicion was confirmed by laboratory tests, his blood serum agglutinating *Br. melitensis* var. *abortus* in 1:1280 dilution.

Case 7 C.—B. L., male, aged 36, farmer. Patient first presented himself having as his only complaint swollen and painful testes. In spite of a negative history, a gonorrheal epididymitis was diagnosed. A detailed history later revealed that for four months prior to the onset of this symptom he had had an increasing lassitude and weakness. Subsequently the sweating characteristic of undulant fever developed, and this led to the correct diagnosis. Two tests on the blood serum were made showing agglutination for *Br. melitensis* var. *abortus* in titers of 1:640 and 1:1280, respectively. In addition, an organism of the *suis* variety was isolated from the blood stream.

Case 8 C.—P. T., male, aged 12, schoolboy. One evening this lad came home from school with headache and no appetite. The parents noticed on this same evening that he had a slight, right-sided limp. On the two following days he went to school, but the same symptoms appeared in the afternoon. On the fourth day he had a rigor, then felt chilly all through the night, and the next day he complained of pain and tenderness in the right loin. He was taken to an osteopath who found a temperature of 103.8°, and an apparently weak right leg. These symptoms in a patient who had headache, and felt generally ill, led to a diagnosis of anterior poliomyelitis, and the patient was quarantined. The fever subsided after about one week; following a few days of normal temperature, it gradually returned, and with it the same type of pain in the right hip. The boy was then taken to another hospital for surgical consultation, where osteomyelitis of the pelvis was considered in view of the localized pain and tenderness. An operation was performed. A small amount of material, supposedly pus, was withdrawn by aspiration. A bacteriological report on this later revealed a contaminant only. An incision was made along the line of the needle puncture, but no abscess or abnormality of any kind was found. Previous and subsequent X-ray analyses of the pelvis were negative.

The material which drained from the wound consisted only of blood. For five days following the operation the boy had daily peaks of high fever. Then for five days his temperature was almost normal, and he went home feeling much better. Three days later, however, a physican was called because of suspected fever, and found the boy's temperature was 101°. The fever persisted, and there continued to be a gradual loss of weight. The pain over the hip also returned, so the family physician reopened the wound, and a drachm of pus was removed. One month after his discharge from hospital he was again readmitted for X-ray and blood tests. At this time a positive agglutination of *Brucella* antigen in a serum dilution of 1:5120 was found. Blood culture also yielded *Br. melitensis* var. *suis*.

Case 9 C.—N. U., male, aged 27, farmer. A three months' illness before admission to hospital was fairly typical of undulant fever, and had been so diagnosed with confirmation from the laboratory. The serum was positive in dilution of 1:5120 and *Br. melitensis* var. *suis* was isolated by blood cultures. From the beginning, however, the patient experienced a dull ache in the abdomen, more prominent in the afternoon or evening when the fever was high. This pain was at the left of the mid line and in the lower part of the epigastrium. On one occasion during the stay in hospital the patient complained of a severe stabbing pain in the left lower quadrant. The area was tender to pressure, and the pain was increased

by movements of the left leg. A little later the pain appeared again, sharp, intermittent, and radiating toward the umbilicus. He was nauseated at the time and was unable to eat the following meal. Examination revealed a diffuse tenderness throughout the abdomen, but without localization or rigidity. There was no surgical intervention and the patient made an uneventful recovery.

Case 10 C.—E. A., male, aged 37, farmer. An early and accurate diagnosis of undulant fever was made in this case. The agglutination titer was 1:640. Here, also, an early and prominent complaint was abdominal pain. This was to the right and was severe, the patient stating that his "stomach was as tender as a boil." The patient felt hungry at times but was afraid to eat on account of the pain. Another time he suffered nausea and vomiting. The examination revealed a tense abdomen, with definite rigidity and marked tenderness, especially in the right upper quadrant. With convalescence from undulant fever this symptom disappeared.

D. FATAL CASES

Case 1 D.—H. G., male 21, packing-house employee. Our first contact with this patient was during a survey conducted in a packing plant for evidence of *Brucella* infection of the employees. At that time the patient considered himself well, but his serum agglutinated *Br. melitensis* var. *abortus* in the 1:2560 dilution. One month later (November 26, 1928) he stopped work and consulted his physician because of profound weakness. During December and January he passed through a moderately severe course of undulant fever, with the usual night sweats, anorexia, and restlessness, and in addition two attacks of anginal pain in the left chest, side, and arm (January 14, 1929, and January 24, 1929). In February, evidence of myocardial failure, but without constant signs of valvular lesions, appeared, and soon the patient died (February 21, 1929). Blood received on December 10, 1928, agglutinated *Br. melitensis* var. *abortus* in a serum dilution 1:2560. Culture medium, inoculated with blood and incubated four days, was sent to us, and from this *Br. melitensis* var. *suis* was isolated.

Three hours after death a necropsy was performed by Doctor Woodward of Mason City, and from him the following notes were obtained.

Height 5 feet 8 inches; weight 140 pounds.

Below the knees there was oedema.

The serous cavities contained clear fluid as follows: Abdominal, 2 liters; pleural, 1 liter on each side; pericardial, 300 cubic centimeters.

The lower lobe of right lung showed fibrous adhesions to the chest wall. There was marked anthracosis in lungs and bronchial lymph glands. The trachea and bronchi contained muco-purulent material.

The heart was hypertrophied to twice its usual size, and weighed 597 grams. When removing the heart, an abscess in the anterior mediastinum was opened. It was the size of a hen's egg and contained a bloody pus. The aorta had an erosion 1 centimeter in diameter and the anterior cusps were entirely destroyed. There was a mass 3 centimeters in diameter occupying the sinus behind the valve and connecting with the abscess in the mediastinum.

The liver was markedly enlarged and of the nutmeg type.

The spleen was enlarged, but on section no unusual pathological changes were noted. Other gross abnormalities were not noted.

From a culture of heart blood which was sent us, the *Br. melitensis* var. *suis* was isolated. The content of the abscess cavity was not examined culturally.

Portions of the various organs were preserved and also sent for examination. Sections were prepared and stained. One set was sent to the Hygienic Laboratory (now National Institute of Health), Washington, D. C., and the detailed report, made by Passed Assistant Surgeon R. D. Lillie, is presented here in full:

A. Pancreas.—Islets numerous and some quite large. No focal lesions.

B. Peribronchial lymph gland.—Moderate amount of coal pigment, marked reticuloendothelial hyperplasia, with relatively few free macrophages, some of which contain phagocytosed red corpuscles. Germinal centers are inconspicuous

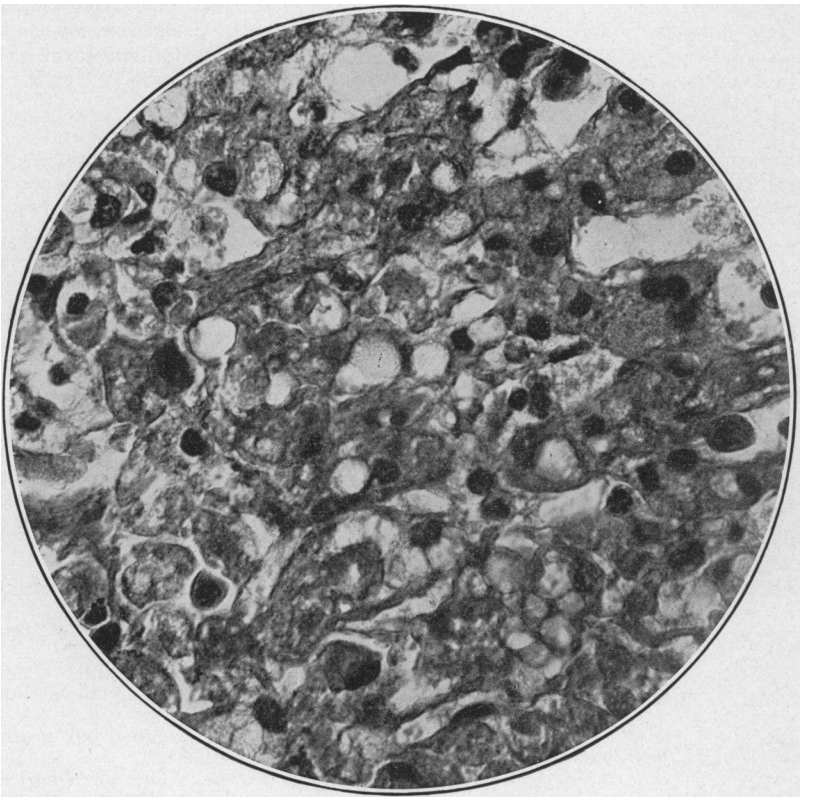

FIGURE 23.—Lesions in the liver of a fatal case of undulant fever (H. G.) due to *Br. melitensis* var. *suis*

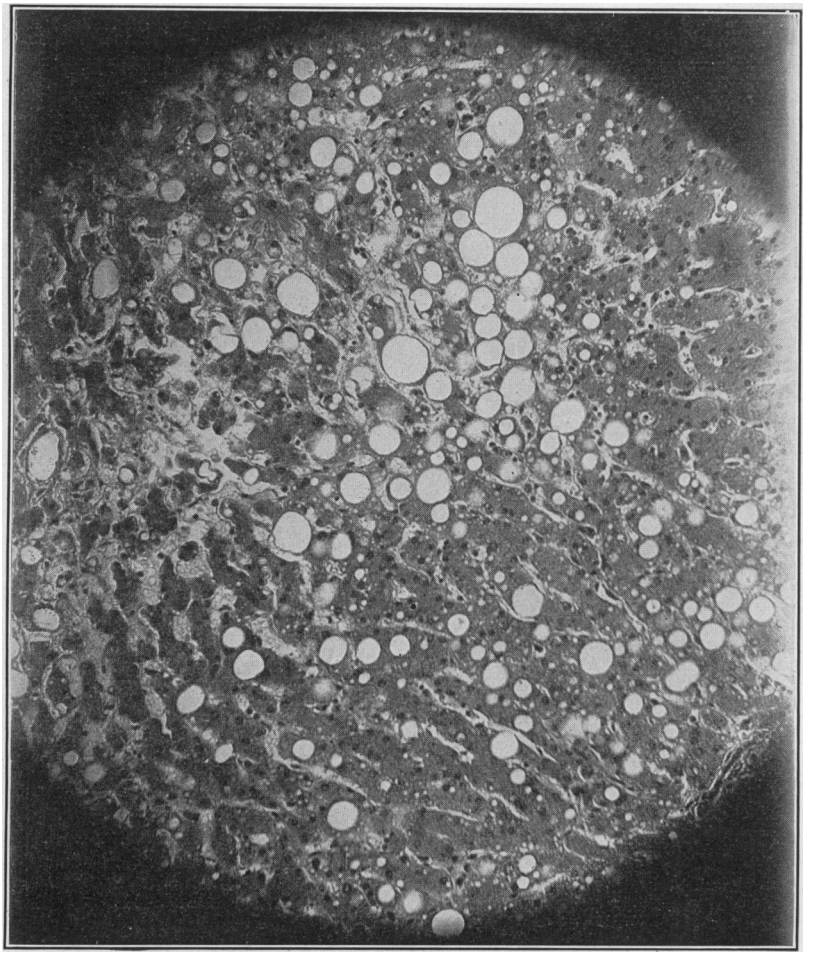

FIGURE 24.—Lesions in the liver of a fatal case of undulant fever (Mrs. H. B.)

and made up largely of small lymphoid cells. Moderate numbers of leucocytes seen among the reticulum cells.

C. Lymph gland.—Reticuloendothelial hyperplasia is even more marked, macrophages are more numerous and more of them contain red corpuscles. The swelling and vacuolation, close packing, and necrosis of these cells seen in typhoid are not noted here, and fixed reticuloendothelial cells greatly predominate.

D. Lymph gland.—Again even more marked hyperplasia of the reticuloendothelium along the course of the sinuses, with moderate numbers of lymphocytes, polymorphonuclears, and macrophages in the meshes of the fixed tissue cells. This gland lies in fatty tissue resembling mesentery.

E. Liver.—The portal areas are surrounded by zones of well-preserved liver cells. Around these, occupying about the middle third of the lobules are zones of swollen liver cells with more eosinophilic vacuolated cytoplasm, the vacuoles being fine to medium in size. The centers of the lobules are occupied by a more or less confused mass of vacuolated oxyphil liver cells with karyolytic nuclei, or no nuclear staining whatever, between which are surviving endothelial nuclei, endothelial cells containing yellowish-brown granular pigment and not infrequently dilated blood-filled capillaries. The periportal connective tissue shows moderate lymphocyte infiltration.

H. Spleen.—The follicles are of moderate size. A few of these show centers of large swollen reticulum cells with cloudy-appearing oxyphil cytoplasm which appears very finely granular with a wide aperture immersion lens on oblique illumination. The pulp contains a considerable amount of blood, a few leucocytes and macrophages, and moderate numbers of lymphoid cells.

I. Kidney.—The glomeruli present *occasional* patches of swollen granular parietal capsular epithelium. The cortical convoluted tubules show granular oxyphil or finely reticular cytoplasm, often with distinct rodded border toward the lumen. Their lumina contain granular débris, irregular granular oxyphil masses, and some more compact hyaline castlike masses. The coarse limbs of Henle's loops show probably a little more cellular swelling and more débris in the lumen. The collecting tubules of the cortex and the medulla present relatively normal epithelium and contain frequent hyaline and less often granular, elongated, rather compact masses.

A small area in the pyramid shows centrally more or less broken down polymorphonuclear leucocytes, about this a zone of mixed polymorphonuclear and large rounded or stellate cells with large vesicular leptochromatic nuclei and rather broad lightly eosinophil cytoplasm. The last grade over into fibro blasts toward the periphery. Here considerable numbers of lymphocytes are seen and the whole lesion is surrounded by a zone of intense congestion and interstitial hemorrhage.

Anatomical findings: Reticuloendothelial hyperplasia of lymph glands. Centro-lobular necrosis and degeneration of liver. Nephrosis, acute toxic.

Case 2 D.—Mrs. H. B., female, aged 57, housewife. There were in the past, present, or family history no significant data. When seen June 20, 1928, the following history was obtained: Patient last felt well in December, 1927, but from that date she noted weakness which progressively became more severe. In January, 1928, she was in bed for one week with a febrile illness considered by her to be "flu." Recovery from this was slow and incomplete. Through February and March she continued with her housework, but complained of general aching and weakness. From April 1 the symptoms were moderately severe, though she did not become bedfast until June 1. Her symptoms were weakness, progressively increasing, and spells of chilliness, particularly in the afternoon, so severe that she would go to bed with a hot soapstone and would still be cold. Several rigors occurred. She sweat profusely, usually after midnight, the bed linen becoming "wet clear to the mattress." General aching, varying in severity, mild headache, backache, marked anorexia, with distressing and persistent nausea and occasional vomiting, occurred. The latter was the prominent symptom throughout the last part of her illness. There was a hacking cough, with glairy mucoid sputum and loss of weight, estimated at 40 pounds in a woman weighing normally 180 pounds. The temperature during June was remitting in character, rarely above 102° F.

Physical examination.—The striking feature was the weakness of the patient, who readily became exhausted by talking. A few fine râles were heard scattered over the lung bases posteriorly. The heart had a rate of 80, and the sounds were of good quality. The spleen or liver could not be palpated. Superficial glands showed no enlargement.

The later course of this illness showed no new features. The nausea and vomiting could not be controlled; the patient progressively became worse and died August 15, 1928.

During June and July three blood specimens were received and *Br. melitensis* var. *abortus* was agglutinated twice in 1:640 dilution and once in 1:1,280. No hemo-cultures were taken.

The urine was found to contain a small number of pus cells and some albumen.

A partial necropsy was allowed, and this was performed by Doctor Nyquist of Eldora. He reported that the striking feature was the complete absence of any notable gross pathological changes in the organs of the abdominal and chest cavities. This we confirmed by an examination in the gross of the organs sent for study.

Sections were prepared and, for confirmation of our observations, were also sent to the Hygienic Laboratory (now National Institute of Health) at Washington, D. C. Their report in part follows (findings by Passed Assistant Surgeon R. D. Lillie):

A. Small intestine.—Moderate postmortem autolysis of mucosa.

B. Pancreas.—Patchy interstitial fibrosis with areas of infiltration chiefly with lymphocytes, and a few plasma cells and macrophages; chronic interstitial pancreatitis.

C. Spleen.—Pulp spaces with contents largely laked blood. Moderate numbers of free large mononuclear cells in sinuses. Follicles small, made up largely of small lymphoid cells. No focal lesions.

D. Muscle.—Plainly striated, much interstitial fat.

E. Gall bladder.—Mucosa autolyzed; stroma shows patches of infiltration with lymphocytes and in places plasma cells. The serosal layer appears thickened and fibrous.

F. Duodenum.—No focal lesions nor ulceration. Mucosa shows autolysis of villus epithelium and many lymphoid cells in the stroma.

G. Lung.—New small patches of scarring and coal pigmentation.

H. Heart.—Considerable epicardial fat. Fibers show moderate lipochrome pigmentation at the poles of the nuclei, clear-cut cross striation, well defined fibrillae, and a moderate amount of transverse fragmentation.

I. Fat tissue.—

J. Liver.—The centers of the lobules show dilated capillaries filled with laked blood, between which are compressed liver cells distended by large vacuoles. This zone is often surrounded by a zone of much distended, coarsely vacuolated liver cells. This vacuolation is probably due to fatty infiltration. There are no focal necroses. The periportal connective tissue contains a moderate number of lymphocytes.

K. Colon.—No ulcers nor focal lesions. Moderate autolysis of mucosa.

L. Kidney.—Considering the degree of autolysis in other tissue and the hemolysis of the blood in the kidney capillaries, the convoluted tubules appear very well preserved. Lesions of the glomeruli or vessels are not noted.

Anatomical findings.—Chronic interstitial pancreatitis; chronic cholecystitis; fatty infiltration of liver; passive congestion of liver; fragmentary myocardial degeneration.

"Without knowledge of the bacteriological, clinical, serological and gross anatomical findings we hesitate to make an interpretation of the findings in these two cases. The presumed fatty infiltration or degeneration in the centers of the liver lobules in the two cases rests purely on morphology, the ordinary microchemical methods not being applicable, as the material is already stained and mounted in balsam."

Case 3 D.—H. L., male, aged 40, farmer. Prior to May, 1928, the patient had enjoyed good health. During that month he had a mild febrile illness regarded as "flu" with general aching and chilliness. During June he improved,

but dating from July 4 he noted an increasing weakness, which caused inability to do his usual work, spells of chilliness in the evening, and night sweats. During July and August he was ambulatory, worked intermittently even though he suffered as a result, and felt fairly well during the days of inactivity. His physician was first consulted about September 1, and at that time his complaint was "my kidneys are not working right." For only two days he had a scant flow with pain on micturition, but because of these symptoms he desired medical advice. During most of September he was in bed; from October 10 to 19 he was in the hospital for observation and diagnosis, and on November 29 he died suddenly. During this period the same symptoms prevailed. There was a moderate to marked weakness. The spells of chilliness did not occur while the patient was in bed, but he did have four or five rigors. The night sweats continued, though they became less profuse late in the disease. The only pain of which he complained was in the chest, bilateral, with radiation to left shoulder and arm. This was not severe and disappeared with rest in bed. While ambulatory he had moderate anorexia, but the appetite returned in September and continued to be good. Constipation was also severe for the same period, but later was less marked. There was a hacking cough. Insomnia occurred, though other nervous disturbances were not noted. A loss of 40 pounds in weight occurred chiefly during the ambulatory period. Doctor Kriebs reported that during the last month of patient's illness, when asked how he was feeling he invariably replied: "Fine; I feel like getting up and going to work."

The hospital record contained the following notes concerning physical findings: Patient looks and feels well. There is a rough systolic murmur over the whole precordium; there are also mitral and pulmonary diastolic murmurs. Röntgenogram showed only a slight enlargement of the heart and no evidence of pericardial effusion. "Clinically, he has plastic pericarditis with slight effusion and dilatation." Two blood counts showed 5,900 and 6,800 white blood cells, respectively, with 65 per cent polymorphonuclears on one count only. On a later examination in addition to the above signs it was found that the spleen was easily palpated. During October, at two different times blood was drawn for the agglutination test and for culture. *Br. melitensis* var. *abortus* was agglutinated in titers of 1:1,280 and 1:2,560, respectively, and from both samples *Br. melitensis* var. *suis* was isolated.

While in the hospital the temperature was irregular and remitting, never as high as 102° F., and rarely ever above 101° F. The pulse was rapid, usually between 100 and 120. During the following month both temperature and pulse definitely decreased.

The added features of the last two weeks of illness were three attacks of dyspnea, the last with a marked increase in pulse rate. For the following five days the pulse remained high, and the cardiac dullness increased. Suddenly, on November 29, when drinking a glass of milk, he collapsed and died.

Though necropsy was not allowed, the clinical evidence of diffuse cardiac disease, involving the pericardium, myocardium, and endocardium, was very definite.

The clinical, serological, and bacteriological findings allow the acceptance of *Br. melitensis* var. *suis* as the etiological agent in this infection.

An examination of the livestock on the farm revealed no infected cattle but showed four serologically positive sows.

Case 4 D.—W. B., male, aged 27, farmer. Prior to present illness the patient had enjoyed good health. Onset began insidiously about the end of March, 1929. He first noted that he readily became tired and drowsy and with difficulty continued his work. He remained ambulatory until early in June, but for most of the period could undertake no work. He was seen June 19, 1929,

at which time his symptoms were as follows: Marked weakness; rigors, nightly for two weeks; profuse night sweats; severe general aching; intermittent headache, worse during ambulatory period; mild lumbar backache, stiff neck—"required rubbing every night"; anorexia and constipation, with severe spells of nausea and vomiting; restlessness and irritability; for two weeks a severe cough, which persisted and became the prominent feature of the latter part of the disease.

We have no record of the physical findings.

Two blood specimens collected during June both agglutinated *Br. melitensis* var. *abortus* in a 1:320 dilution.

Br. melitensis var. *abortus* and *suis*, was isolated from the blood cultures. The fact that both porcine and bovine varieties were isolated was of particular interest.

Toward the end of July the symptoms and signs of lung abscess developed. Early in August an operation for drainage was performed. The patient gradually grew weaker and shortly afterwards died.

Whether one or both of the varieties of *Br. melitensis* found by culture were primarily involved in the production of this abscess, or whether the infection reduced the general and local resistance to other organisms which brought about the tissue destruction, can not be determined.

Case 5 D.—R. B., male, aged 29. Stock buyer, purchasing and handling only hogs. From about January 24, 1929, he complained frequently of chilliness. From February 6 he had an attack of "flu" which confined him to bed for two days. Though still ill he returned to work after four days but on February 13 he came home, admitting he was sick, went to bed, and died one month later.

His symptoms during this period were as follows: Marked prostration, severe and persisting frontal headache and backache, anorexia, constipation, restlessness, rapid loss of weight, and later delirium. He had two rigors. Night sweats were never profuse and were confined to the early period of his disease. He had three convulsions shortly before death.

The physical examination was reported as essentially negative.

The temperature was high, with slight morning remissions. The pulse at first was relatively slow (80 to 100), but during the last week it was rapid and slightly irregular.

Albuminuria was noted late in the disease.

Three white blood counts between March 3, 1929, and March 12, 1929, were 4,150, 4,250, and 2,950. One differential showed polymorphonuclears 32 per cent, small lymphocytes 19 per cent, and large mononuclears 50 per cent.

On February 27, 1929, the blood serum agglutinated *Br. melitensis* var. *abortus* in the 1:1,280 dilution.

This case, therefore, was a malignant type of infection without signs of localization.

Case 6 D.—Mrs. C. H., female, aged 27, housewife. This patient had enjoyed exceptionally good health prior to the onset of the present illness. Her husband had suffered from a fever during January and February, 1927, which clinically had been diagnosed typhoid fever, but the characteristics were found to be those of undulant fever. Blood serum obtained during April agglutinated *Br. melitensis* var. *abortus* in the 1:100 dilution, but caused no agglutination of *B. typhosus* or *paratyphosus*. Throughout his illness he had been nursed by his wife. During his convalescence, on March 12, she suddenly became acutely ill. The evening meal had been enjoyed with friends, but shortly afterwards she was taken home to bed with severe headache, prostration, and fever. Before midnight the physician was called and a temperature between 103° and 104° was found. For 10 days the patient was cared for at home. Her complaints were

extreme weakness, marked general aching, spells of chilliness, complete anorexia with frequent nausea and occasional vomiting. On March 21, the 10th day of illness, the patient was admitted to the hospital. The physicians admission note was as follows: "The high temperature is not characteristic of influenza, but the absence of all symptoms and findings of typhoid make it the only diagnosis available at this time." There was no diarrhea, tympanites, or notable constipation, and the patient lacked the dull toxic appearance so characteristic of typhoid. During the subsequent course the patient was remarkably free from discomfort, complaining only of extreme exhaustion and feverishness. The temperature was sustained and continued to rise. The pulse became very rapid, but the respiration was never embarrassed.

Physical examination throughout was essentially negative. Late in the disease râles appeared. The spleen was not palpated.

The illness progressed, delirium and coma appeared, and there were involuntary passages of urine and feces. On the 21st day of the disease the patient died, with death attributed to a myocardial failure.

Blood smears were examined by us. There was obviously a marked leucopenia and the differential count showed polymorphonuclears, 21 per cent; small lymphocytes, 10 per cent; large mononuclears, 69 per cent.

Three blood specimens were sent for Widal tests, which were collected on the 11th, 15th, and 19th days of the disease. The first two were dried specimens, the last whole wet blood. The first specimen showed no agglutination of *Br. melitensis* var. *abortus*, the second showed microscopically some clumping in the 1:40 and 1:80 dilutions, while the third showed complete agglutination in the 1:80 serum dilution. This evidence of increasing agglutinins gives strong evidence of the specific nature of the infection, even though the final titer is not high.

The temperature chart of this case is shown as the first of the two malignant types illustrated (fig. 22).

No postmortem examination was allowed.

There is here clinical evidence of an overwhelming septicemia, with no evidence of any localization.

The possible source of this infection gives added interest to this case. The family used pasteurized milk. The husband was frequently away and acquired his infection from some undetermined source; while the wife, as the nurse, may have acquired her infection from the excreta of her husband.

Case 7 D.—E. B., male, aged 22, farmer. The patient first consulted his physician August 15, 1927, and stated that he had not been well for one month. He was weak, had no appetite, and had had spells of chilliness and fever. He also complained of abdominal pain in the right lower quadrant. From that time the patient was acutely ill. He was observed and treated in the hospital for the first two weeks of September, but died at home on September 23. He was dull and early in the disease had delirium. There was profuse perspiration and constipation. The patient's chief complaint was arthralgia involving chiefly the toes, ankles, knees, and elbows. There was local tenderness but no swelling. During the last week he complained of epigastric pain, and tenderness was noted. Throughout, an unusual and prominent symptom was deafness.

The spleen was not palpable.

The temperature was irregularly remitting, having a daily maximum of 103° F., but rarely reaching 104° F. or higher late in the disease.

There was a marked leukopenia, the white blood count on two occasions being 2,150.

Widal tests were repeatedly negative. The first blood specimen received by us was dried, and agglutination of *Br. melitensis* var. *abortus* was found. Blood for a microscopic test was later received, and we reported agglutination

positive in the 1:320 dilution. As this was one of our earlier cases, the blood was also sent to the Hygienic Laboratory (now National Institute of Health), Washington, D. C., and there a titer of 1:640 was found.

Case 8 D.—L. J. H., male, aged 52, farmer. The past, present, and family history is irrelevant. There was a gradual onset about September 20, 1928, spells of chilliness being his first complaint. On October 10 he was admitted to the hospital at which time the prominent features of his disease were stated to be "chills" and sweats. These had persisted for three weeks and the chills were true rigors. He perspired after his chills, and "in the evening after going to sleep, wakened, drenched with sweat." Slight general aching, mild headache, listlessness, and drowsiness are mentioned.

Physical examination notes are confined chiefly to the cardio-vascular system. On October 18, 1928, an aortic diastolic murmur was first heard. This persisted and became more marked. Blood pressure on admission October 10, 1928, was 104/64, and on October 21, 1928, it was 88/30. On October 31, 1928, there was numbness and weakness of the entire right leg, which was attributed to an embolism. Condition progressively became worse and patient died November 11, 1928.

The temperature was of a septic type, 99° to 100° F. in the morning and in the evening 104° or higher. The day before death it reached 106.4°.

There was a moderate tachychardia.

Two blood cultures, incubated for only 72 hours, showed no growth.

Two agglutination tests gave titers for *Br. melitensis* var. *abortus*, as follows: October 15, 1928, 1:20; October 25, 1928, 1:80.

It is our opinion that this patient died of a malignant endocarditis due to *Brucella* infection.

Case 9 D.—A., male, aged 29, laborer. The patient, a very poor Mexican, spoke no English, lived alone, and was practically unattended throughout his illness. The physician, moreover, was called late and infrequently. Data are therefore meager. As far as is known the duration of illness was three weeks. The nature of onset could not be determined. He had true rigors and excessive sweating. There was headache, backache, and pain in the back of the neck. Patient was very restless, had marked insomnia and "was much worse at night." Until late in the illness he could not be kept in bed. He had a moderate cough, and some sputum in which tubercle bacilli could not be found. Just before death there was delirium, some cyanosis, and rapid respiration.

The attending physician and a consultant found, late in the disease, "evidence of capillary bronchitis" and an enlarged spleen. The temperature was taken only occasionally, but there was clearly a high fever. One serum specimen, sent to us from the local laboratory, showed agglutination of *Br. melitensis* var. *abortus* in 1:2,560 dilution.

Miliary tuberculosis was carefully considered as a diagnosis, but the illness and death was attributed to undulant fever. This opinion seems justified.

Case 10 D.—Mrs. D. B., female, aged 55, farmer's wife. Illness began the last week of November, 1928. She first noted burning and smarting on urination. This continued until December 10, at which time the patient first complained of feverishness. She was also "nervous and uneasy" and distressed by sleeplessness. At about this date diarrhea commenced and nausea and vomiting became prominent. Weakness became more and more marked. There was also severe aching, worse in the joints, causing the patient to state she "had rheumatism." On December 24 she was admitted to the hospital. In the nurses' daily record it is stated that "she is continually soaked in perspiration." This symptom is not mentioned after January 1. She continued to be distressed by diarrhea, nausea, and vomiting. There was severe headache which persisted.

The weakness rapidly increased. The patient became irrational and died February 9. Emaciation at the last was extreme, the total loss of weight estimated at 75 pounds. The pulse became rapid and weak, and death was attributed to exhaustion.

The temperature chart during the period of hospitalization showed only a low grade fever, higher during the first two weeks, but even then it rarely reached 102°.

The physical examination was reported essentially negative, a fiery red, dry tongue alone being emphasized.

The white blood counts were high. Three examinations while in the hospital (date unrecorded) were 15,000; 11,000, and 7,000, with corresponding polymorphonuclears of 93 per cent, 78 per cent, and 55 per cent.

The urine examination was stated to be negative. There were no notes concerning the character of the stools.

Blood serum agglutinated *Br. melitensis* var. *abortus* as follows: December 29, 1:640; January 10, 1:320.

Blood cultures were not obtained and necropsy was not allowed.

Whether *Brucella* infection of an unusual type caused the patient's death or whether it was an incidental intercurrent infection remains undetermined.

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DEATH RATES IN A GROUP OF INSURED PERSONS

Rates for Principal Causes of Death for August, 1930

The accompanying table, taken from the Statistical Bulletin for September, 1930, issued by the Metropolitan Life Insurance Co., presents the mortality record of the industrial insurance department of the company for August, 1930, as compared with that for the preceding month and for the corresponding month of last year. It also gives the cumulative rates for the period January–August for the years 1930 and 1929. These rates are based on a strength of approximately 19,000,000 insured persons in the United States and Canada.

Thus far in 1930 only one month (July) has registered a higher death rate among this group than did the corresponding month last year. The mortality rate in August was 7.5 per 1,000, as compared with 7.7 last year, and the cumulative rate for the eight elapsed months was only 8.9 per 1,000, as against 9.9 in 1929—a reduction of 10 per cent. Among the policyholders living west of the Rocky Mountains, the August death rate was 5.9 per 1,000, as compared with 6.1 last year; in the remainder of the United States this year's figure was 7.6, as compared with 7.8; while among Canadian policyholders, the corresponding figures were 8 and 8.1.[1]

Tuberculosis, diphtheria, and puerperal diseases continue to be the outstandingly favorable items in what bids fair to be the best health year of all time. The tuberculosis death rate for the eight elapsed months was more than 9 per cent below that for the like period of last year, the previous minimum. The diph-

[1] These rates apply to a more or less selected group of persons. In recent years the general annual death rate in this group has been approximately 73 per cent of the rate for the registration area of the United States.—*Ed.*

theria rate has declined 28 per cent and that for maternal diseases over 11 per cent in a single year.

With two-thirds of 1930 already past, the cancer death rate is 76.1 per 100,000, as compared with 77.5 for the like part of 1929. There is now a very good prospect that the almost continuous rise in the mortality from malignant growths will be broken this year. While this is an encouraging development, too much significance must not be attached to it. If, however, 1931 shows a further drop, there will be good grounds for the hope that the rise in cancer mortality is destined to be checked. Pneumonia, heart disease, Bright's disease, and, more particularly, influenza, are other important diseases which have recorded much lower mortality rates than in 1929. Slight improvement is also in evidence for diabetes and diarrheal complaints.

Death rates (annual basis) per 100,000 for principal causes of death, August, 1930

[Industrial department, Metropolitan Life Insurance Co.]

Cause of death	Rate per 100,000 lives exposed [1]				
	August, 1930	July, 1930	August, 1929	Cumulative, January–August	
				1930	1929
Total, all causes	751.3	843.7	767.7	892.1	987.8
Typhoid fever	3.2	2.6	3.3	1.7	2.1
Measles	.6	2.3	1.6	3.8	4.0
Scarlet fever	1.1	1.8	1.1	2.9	3.0
Whooping cough	5.4	4.9	6.4	4.7	6.4
Diphtheria	3.0	4.3	4.0	6.2	8.6
Influenza	3.3	4.2	3.9	16.7	55.8
Tuberculosis (all forms)	71.6	85.0	78.6	83.7	92.1
Tuberculosis of respiratory system	62.3	74.4	70.1	72.7	81.6
Cancer	73.6	79.3	72.6	76.1	77.5
Diabetes mellitus	16.1	16.7	14.4	18.8	19.5
Cerebral hemorrhage	53.7	60.2	[2] 46.4	60.8	[2] 59.3
Organic diseases of heart	112.7	133.7	117.0	148.0	154.7
Pneumonia (all forms)	29.4	39.2	34.4	84.4	103.5
Other respiratory diseases	8.3	10.2	9.2	11.7	13.1
Diarrhea and enteritis	32.2	22.9	31.5	16.3	17.4
Bright's disease (chronic nephritis)	58.6	66.9	59.9	69.0	72.0
Puerperal state	10.8	11.4	11.7	12.5	14.1
Suicides	9.3	9.3	7.1	9.5	8.6
Homicides	6.3	7.8	6.6	6.4	6.4
Other external causes (excluding suicides and homicides)	75.6	80.1	71.6	62.5	64.0
Traumatism by automobiles	22.4	22.2	22.5	19.2	18.6
All other causes	176.7	200.9	186.6	196.1	205.4

[1] All figures in this table include insured infants under 1 year of age. The rates for 1930 are subject to slight correction, as they are based on provisional estimates of lives exposed to risk.
[2] Rate not comparable with that for 1930.

DEATHS DURING WEEK ENDED SEPTEMBER 27, 1930

Summary of information received by telegraph from industrial insurance companies for the week ended September 27, 1930, and corresponding week of 1929. (From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce)

	Week ended Sept. 27, 1930	Corresponding week, 1929
Policies in force	75,495,053	74,762,355
Number of death claims	12,170	12,587
Death claims per 1,000 policies in force, annual rate	8.4	8.8

Deaths [1] from all causes in certain large cities of the United States during the week ended September 27, 1930, infant mortality, annual death rate, and comparison with corresponding week of 1929. (From the Weekly Health Index issued by the Bureau of the Census, Department of Commerce)

[The rates published in this summary are based upon mid-year population estimates derived from the 1930 census. The rates are not exactly comparable with similar rates published in the Public Health Reports earlier than the issue of August 22, 1930, which were based upon estimates made before the 1930 census was taken]

City	Week ended Sept. 27, 1930				Corresponding week 1929		Death rate [2] for first 39 weeks	
	Total deaths	Death rate [2]	Deaths under 1 year	Infant mortality rate [3]	Death rate [2]	Deaths under 1 year	1930	1929
Total (77 cities)	6,879	10.5	762	[4] 61	11.1	769	12.1	12.9
Akron	40	8.2	4	37	9.3	4	7.9	9.5
Albany [5]	40	16.3	2	41	19.0	4	15.0	16.5
Atlanta	63	12.3	6	61	16.3	12	16.0	16.2
White	31		6	95		6		
Colored	32	(6)	0	0	(6)	6	(6)	(6)
Baltimore [5]	178	11.6	28	97	13.2	24	14.1	14.9
White	126		21	93		17		
Colored	52	(6)	7	112	(6)	7	(6)	(6)
Birmingham	62	12.5	7	67	12.0	7	13.9	16.4
White	26		3	48		3		
Colored	36	(6)	4	98	(6)	4	(6)	(6)
Boston	180	12.0	23	67	13.2	26	14.2	15.3
Bridgeport	24	8.5	4	68	9.9	4	11.1	12.3
Buffalo	122	11.1	14	62	14.7	15	13.1	14.2
Cambridge	20	9.2	3	60	12.0	4	11.7	12.7
Camden	30	13.3	1	18	14.2	6	13.9	14.7
Canton	18	8.9	4	107	8.5	5	10.0	11.4
Chicago [5]	666	10.2	64	57	10.0	64	10.5	11.4
Cincinnati	148	17.1	12	71	16.0	21	15.8	17.3
Cleveland	164	9.5	23	69	10.0	13	11.2	12.7
Columbus	77	13.8	9	89	11.1	7	15.8	15.1
Dallas	45	8.9	10		10.1	7	11.6	11.7
White	33		9			5		
Colored	12	(6)	1		(6)	2	(6)	(6)
Dayton	50	12.9	6	90	13.2	7	10.7	11.6
Denver	77	13.9	13	142	10.6	2	14.9	15.0
Des Moines	24	8.3	6	111	12.2	2	11.8	11.8
Detroit	218	7.2	33	51	10.2	44	9.4	11.4
Duluth	26	13.4	2	54	11.9	3	11.3	11.7
El Paso	27	13.7	6		13.5	10	17.7	20.2
Erie	17	7.6	3	66	9.5	1	11.3	12.6
Fall River [5] [7]	27	12.3	8	185	8.6	1	12.2	14.1
Flint	35	11.6	11	130	12.0	11	9.3	10.9
Fort Worth	29	9.4	1		8.9	5	11.3	12.7
White	24		1			3		
Colored	5	(6)	0		(6)	2	(6)	(6)
Grand Rapids	37	11.4	6	90	12.2	3	10.5	10.3
Houston	67	11.9	12		11.7	4	12.4	12.8
White	44		7			2		
Colored	23	(6)	5		(6)	2	(6)	(6)
Indianapolis	118	16.8	16	120	14.5	13	14.8	14.9
White	97		13	112		9		
Colored	21	(6)	3	175	(6)	4	(6)	(6)
Jersey City	45	7.4	4	35	15.1	8	11.4	12.8
Kansas City, Kans	34	14.5	2	47	11.1	1	11.6	13.4
White	25		1	28		1		
Colored	9	(6)	1	152	(6)	0	(6)	(6)
Kansas City, Mo	97	12.8	9	75	12.6	5	13.6	14.2
Knoxville	15	7.3	4	94	12.1	1	13.8	14.0
White	13		4	104		0		
Colored	2	(6)	0	0	(6)	1	(6)	(6)
Los Angeles	242	10.1	23	70	9.2	11	11.1	11.5
Lowell [7]	21	10.9	2	53	13.4	3	13.5	14.4
Lynn	9	4.6	0	0	12.3	0	10.5	11.5
Memphis	50	10.3	8	94	14.8	3	17.5	19.4
White	28		4	72		1		
Colored	22	(6)	4	135	(6)	2	(6)	(6)
Milwaukee	101	9.2	11	48	10.5	17	9.8	11.2
Minneapolis	87	9.8	5	33	10.5	4	10.7	11.0
Nashville	50	17.7	6	94	15.6	4	17.5	19.1
White	33		4	84		4		
Colored	17	(6)	2	124	(6)	0	(6)	(6)
New Bedford [7]	27	12.5	5	128	10.1	3	10.9	12.5

See footnotes at end of table.

Deaths [1] from all causes in certain large cities of the United States during the week ended September 27, 1930, infant mortality, annual death rate, and comparison with corresponding week of 1929. (From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce)—Continued

City	Week ended Sept. 27, 1930				Corresponding week 1929		Death rate [2] for first 39 weeks	
	Total deaths	Death rate [2]	Deaths under 1 year	Infant mortality rate [3]	Death rate [2]	Deaths under 1 year	1930	1929
New Haven	10	3.2	0	0	12.5	2	12.8	13.4
New Orleans	144	16.4	16	89	16.8	17	17.6	17.8
White	87		10	85		8		
Colored	57	(6)	6	97	(6)	9	(6)	(6)
New York	1,192	8.9	110	46	9.3	137	10.9	11.5
Bronx Borough	172	7.0	11	32	7.6	13	8.0	8.3
Brooklyn Borough	389	7.8	32	34	8.1	45	9.8	10.4
Manhattan Borough	472	13.3	51	65	13.8	67	16.3	16.7
Queens Borough	126	6.0	14	56	5.5	8	7.1	7.7
Richmond Borough	33	10.9	2	39	15.2	4	14.5	16.2
Newark, N. J	86	10.1	11	58	10.5	16	12.1	13.0
Oakland	54	9.8	1	12	9.1	4	11.0	11.5
Oklahoma City	12	3.4	4	72	8.3	7	10.7	10.8
Omaha	34	8.3	5	61	13.7	1	13.6	13.9
Paterson	26	9.8	2	35	10.2	4	12.3	13.6
Philadelphia	426	11.3	61	91	10.8	50	12.7	13.3
Pittsburgh	177	13.8	18	64	15.2	29	13.9	15.0
Porland, Oreg	49	8.5	5	62	12.3	0	12.3	12.9
Providence	46	9.5	5	46	13.1	5	13.2	14.8
Richmond	39	11.1	4	58	17.2	5	15.0	16.5
White	20		2	44		1		
Colored	19	(6)	2	85	(6)	4	(6)	(6)
Rochester	68	10.9	4	36	10.3	8	11.7	12.6
St. Louis	204	12.9	25	87	12.4	7	14.3	14.9
St. Paul	40	7.7	4	40	8.6	4	10.1	10.6
Salt Lake City [4]	15	5.6	0	0	10.2	4	12.3	13.1
San Antonio	47	9.5	8		11.2	7	15.4	14.7
San Diego	40	14.0	1	21	13.1	4	14.5	15.3
San Francisco	165	13.7	8	54	12.3	2	13.3	13.3
Schenectady	20	10.9	1	31	10.4	2	11.4	12.5
Seattle	58	8.3	4	40	9.7	4	11.0	11.1
Somerville	15	7.5	1	32	11.7	0	9.8	9.4
Spokane	24	10.8	1	26	10.4	0	12.3	13.0
Springfield, Mass	33	11.4	1	17	12.7	5	12.2	13.0
Syracuse	36	9.0	5	62	10.4	5	11.7	13.4
Tacoma	19	9.3	0	0	10.3	1	12.5	11.8
Toledo	74	13.2	2	18	13.2	13	12.7	13.8
Trenton	29	12.3	5	96	15.3	1	16.8	17.3
Utica	24	12.2	3	83	17.3	8	14.8	15.7
Washington, D. C	127	13.6	16	94	12.7	10	15.2	15.5
White	76		3	26		6		
Colored	51	(6)	13	232	(6)	4	(6)	(6)
Waterbury	14	7.2	1	24	6.2	4	9.7	9.5
Wilmington, Del. [7]	39	19.4	5	121	11.4	5	14.8	14.1
Worcester	35	9.3	2	28	8.8	1	12.8	12.8
Yonkers	16	6.1	0	0	9.4	2	8.1	9.4
Youngstown	35	10.7	7	100	10.5	6	10.3	12.3

[1] Deaths of nonresidents are included. Stillbirths are excluded.

[2] These rates represent annual rates per 1,000 population, as estimated for 1930 and 1929 by the arithmetical method.

[3] Deaths under 1 year of age per 1,000 live births. Cities left blank are not in the registration area for births.

[4] Data for 72 cities.

[5] Deaths for week ended Friday.

[6] For the cities for which deaths are shown by color the colored population in 1920 constituted the following percentages of the total population: Atlanta, 31; Baltimore, 15; Birmingham, 39; Dallas, 15; Fort Worth, 14; Houston, 25; Indianapolis, 11; Kansas City, Kans., 14; Knoxville, 15; Memphis, 38; Nashville, 30; New Orleans, 26; Richmond, 32; and Washington, D. C., 25.

[7] Population Apr. 1, 1930; decreased 1920 to 1930; no estimate made.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

Reports for Weeks Ended October 4, 1930, and October 5, 1929

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended October 4, 1930, and October 5, 1929

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929
New England States:								
Maine	2	3	1		8	1	0	0
New Hampshire	1	4		3		8	0	0
Vermont		4			1		0	0
Massachusetts	38	75	1	2	54	29	5	2
Rhode Island	4	5				2	1	0
Connecticut	7	14	8	2	2	4	2	0
Middle Atlantic States:								
New York	60	113	[1] 3	[1] 1	41	81	8	12
New Jersey	79	79	1	4	25	8	2	2
Pennsylvania	121	167			49	75	5	11
East North Central States:								
Ohio	48	68	2	11	12	59	3	2
Indiana	63	44	16		5	10	5	0
Illinois	118	165	18	10	34	62	9	7
Michigan	43	77		3	11	83	0	20
Wisconsin	1	23	10	23	36	89	3	4
West North Central States:								
Minnesota	17	17	1	3	1	25	3	0
Iowa	3	7				2	1	2
Missouri	30	39	1	1	34	20	3	8
North Dakota	3	9			18	2	0	2
South Dakota	5	7			1	2	0	1
Nebraska	10	19				11	2	1
Kansas	9	28		2	3	10	2	0
South Atlantic States:								
Delaware	1	3			4	1	0	0
Maryland [2]	11	16	1	3	4	1	0	2
District of Columbia	9	12		2	3	4	0	0
Virginia								
West Virginia	21	38	5	1	17	12	0	0
North Carolina	129	229	5		5	4	1	2
South Carolina	38	63	187	316			0	0
Georgia	22	22	20	42	23	3	1	0
Florida	4	9		2	2	3	0	0
East South Central States:								
Kentucky	28	24					7	1
Tennessee	36	39	2	16	7	1	0	1
Alabama	43	44	22	7	22		1	1
Mississippi	40	54					1	2

[1] New York City only.

[2] Week ended Friday.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended October 4, 1930, and October 5, 1929—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929
West South Central States:								
Arkansas	3	25	5	29	1	2	0	0
Louisiana	24	22	4	5	3	1	0	2
Oklahoma [3]	38	75	6	33	7	29	0	1
Texas	41	57	11	28	2	2	0	0
Mountain States:								
Montana	4					81	0	0
Idaho	1				7	4	0	0
Wyoming	1	2					0	1
Colorado	5	8			65	3	2	2
New Mexico	5	10					2	0
Arizona	6	4	2		12	1	1	0
Utah [3]		2	5	6	1	3	4	2
Pacific States:								
Washington	12	11		2	11	3	1	5
Oregon	2	8	15	12	45	9	1	0
California	39	40	31	24	67	43	1	7

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929
New England States:								
Maine	9	1	12	12	0	0	8	1
New Hampshire	1	0	0	12	0	0	2	0
Vermont	1	0	0	1	0	0	0	0
Massachusetts	38	4	67	108	0	0	8	5
Rhode Island	2	1	4	5	0	0	1	3
Connecticut	10	1	16	14	0	0	4	10
Middle Atlantic States:								
New York	50	34	100	81	0	5	86	44
New Jersey	3	4	47	44	0	0	12	5
Pennsylvania	15	14	151	166	0	0	43	94
East North Central States:								
Ohio	75	12	162	125	36	27	95	41
Indiana	17	0	72	59	18	21	20	13
Illinois	23	2	108	229	7	46	38	31
Michigan	20	11	90	144	2	18	27	15
Wisconsin	14	0	54	61	4	2	7	17
West North Central States:								
Minnesota	17	0	28	55	19	1	0	0
Iowa	25	6	36	35	12	10	4	4
Missouri	18	0	28	42	0	12	25	13
North Dakota	3	2	7	8	6	3	6	4
South Dakota	14	0	3	7	6	19	2	2
Nebraska	60	0	13	15	5	6	3	0
Kansas	87	1	38	18	2	13	11	5
S uth Atlantic States:								
Delaware	0	0	0	2	0	0	3	3
Maryland [3]	2	2	24	35	0	0	35	30
District of Columbia	0	1	4	10	0	0	4	1
Virginia		21						
West Virginia	1	1	48	44	0	8	70	42
North Carolina	1	8	86	122	0	2	21	29
South Carolina	2	3	19	18	0	0	41	38
Georgia	3	1	27	38	0	0	32	18
Florida	2	1	2	6	1	0	1	3
East South Central States:								
Kentucky	2	0	51	28	0	0	40	22
Tennessee	1	2	49	43	0	1	55	32
Alabama	4	1	39	48	0	6	31	13
Mississippi	0	2	18	29	1	1	19	26
West South Central States:								
Arkansas	11	0	10	20	0	0	21	23
Louisiana	7	0	15	12	1	0	28	16
Oklahoma [3]	4	2	41	56	3	4	35	49
Texas	8	0	24	38	17	4	20	21

[2] Week ended Friday.

[3] Figures for 1930 are exclusive of Oklahoma City and Tulsa.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended October 4, 1930, and October 5, 1929—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929	Week ended Oct. 4, 1930	Week ended Oct. 5, 1929
Mountain States:								
Montana	2	0	13	8	0	7	8	53
Idaho	0	0	1	5	0	4	3	1
Wyoming	12	0	6	2	0	3	0	0
Colorado	5	1	16	12	3	15	8	10
New Mexico	2	0	6	6	0	1	14	16
Arizona	3	1	10	2	0	0	1	3
Utah [1]	0	0	11	14	0	----	7	2
Pacific States:								
Washington	3	1	33	35	22	19	11	8
Oregon	2	0	16	8	0	7	9	1
California	68	2	73	73	10	12	14	8

[1] Week ended Friday.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Cerebrospinal meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
August, 1930										
Massachusetts	10	183	4	4	227	3	104	192	0	63
Montana	3	3	5	----	14	1	1	32	11	13
Texas	6	99	18	1,187	----	10	18	74	----	138
September, 1930										
Nebraska	4	14	2	----	18	----	72	47	45	17

August, 1930

	Cases
Actinomycosis:	
Massachusetts	1
Anthrax:	
Massachusetts	1
Chicken pox:	
Massachusetts	70
Montana	11
Dysentery:	
Massachusetts	5
German measles:	
Massachusetts	22
Mumps:	
Massachusetts	144
Montana	26
Ophthalmia neonatorum:	
Massachusetts	73
Septic sore throat:	
Massachusetts	9
Tetanus:	
Massachusetts	2
Trachoma:	
Massachusetts	1
Trichinosis:	
Massachusetts	2
Undulant fever:	
Montana	1
Whoping cough:	
Massachusetts	551
Montana	87

September, 1930

	Cases
Nebraska:	
Anthrax	1
Chicken pox	32
Mumps	12
Undulant fever	1
Whooping cough	55

Cases of Certain Communicable Diseases Reported for the Month of June, 1930, by State Health Officers

State	Chicken pox	Diphtheria	Measles	Mumps	Scarlet fever	Smallpox	Tuberculosis	Typhoid and paratyphoid fever	Whooping cough
Maine	125	35	197	217	40	0	60	8	77
New Hampshire	----	3	----	----	18	0	----	0	----
Vermont	96	1	196	1	25	0	28	0	62
Massachusetts	876	203	4,227	454	603	0	523	17	773
Rhode Island	87	17	100	2	46	0	39	1	32
Connecticut	281	47	146	112	161	0	142	4	172
New York	1,894	478	8,618	1,498	1,069	30	1,877	71	1,445
New Jersey	613	341	4,268	----	467	0	481	23	316
Pennsylvania	1,323	345	3,984	924	973	0	583	65	857
Ohio	1,016	127	1,808	353	627	328	703	43	698
Indiana	179	43	530	11	260	440	261	28	148
Illinois	839	553	1,777	726	1,120	327	1,052	55	783
Michigan	742	259	3,514	565	918	230	311	21	1,004
Wisconsin	894	50	1,735	579	329	88	164	9	672
Minnesota	355	60	437	----	207	24	292	7	110
Iowa	74	18	360	44	111	427	35	10	58
Missouri	196	97	315	118	352	196	161	41	124
North Dakota	15	15	56	48	53	87	20	3	89
South Dakota	44	27	415	11	21	129	10	1	27
Nebraska	113	20	279	40	90	140	12	7	33
Kansas	178	43	839	211	123	298	136	29	338
Delaware	18	3	18	3	26	0	10	1	17
Maryland	312	57	138	60	188	0	248	28	195
District of Columbia	92	25	260	----	34	0	109	4	19
Virginia	305	58	1,259	----	72	6	167	137	751
West Virginia	60	23	199	----	72	38	40	36	154
North Carolina	228	42	268	----	63	44	----	134	1,201
South Carolina	209	67	149	87	14	7	151	244	400
Georgia	25	13	416	67	41	8	54	100	149
Florida	16	24	216	71	4	2	26	15	16
Kentucky [1]	----	----	----	----	----	----	----	----	----
Tennessee	83	23	349	32	100	49	[2] 143	119	122
Alabama	57	32	404	65	32	19	356	64	197
Mississippi	337	27	231	406	13	12	278	189	1,432
Arkansas	17	7	84	26	9	12	[2] 14	45	108
Louisiana	26	59	41	5	63	8	[2] 201	119	27
Oklahoma [3]	4	34	233	13	53	287	47	48	107
Texas	----	51	----	----	56	----	----	61	----
Montana	21	2	81	46	67	17	11	6	50
Idaho	75	5	46	22	13	24	10	2	73
Wyoming	7	5	184	11	9	48	[2] 1	1	9
Colorado	98	19	1,616	329	53	38	92	6	294
New Mexico	33	37	156	30	20	21	91	6	13
Arizona	27	4	228	45	13	11	148	24	54
Utah [1]	----	----	----	----	----	----	----	----	----
Nevada	----	----	14	12	----	16	[2] 2	----	----
Washington	179	15	1,876	292	63	139	142	17	255
Oregon	91	15	386	85	39	69	54	17	160
California	941	212	5,919	1,646	393	178	921	87	863

[1] Reports received weekly. [2] Pulmonary. [3] Exclusive of Oklahoma City and Tulsa.

Case Rates per 1,000 Population (Annual Basis) for the Month of June, 1930

[The rates here given and those for previous months of 1930 have been calculated by use of approximated populations and may not be strictly comparable with similar rates for subsequent months of the year, which will be based on results of the 1930 census]

State	Chicken pox	Diphtheria	Measles	Mumps	Scarlet fever	Smallpox	Tuberculosis	Typhoid and paratyphoid fever	Whooping cough
Maine	1.90	0.53	3.00	3.30	0.61	.00	0.91	0.12	1.17
New Hampshire	------	.08	------	------	.48	.00	------	.00	------
Vermont	3.31	.03	6.77	.03	.86	.00	.97	.00	2.14
Massachusetts	2.43	.56	11.73	1.26	1.67	.00	1.45	.05	2.14
Rhode Island	1.43	.28	1.64	.08	.76	.00	.64	.02	.53
Connecticut	1.98	.33	1.03	.79	1.13	.00	1.00	.03	1.21
New York	1.95	.49	8.89	1.54	1.10	.03	1.94	.07	1.49
New Jersey	1.88	1.05	13.10	------	1.43	.00	1.48	.07	.97
Pennsylvania	1.59	.42	4.80	1.11	1.17	.00	.70	.08	1.03
Ohio	1.75	.22	3.12	.61	1.08	.57	1.21	.07	1.20
Indiana	.67	.16	2.00	.04	.98	1.66	.98	.11	.56
Illinois	1.34	.89	2.85	1.16	1.79	.52	1.69	.09	1.25
Michigan	1.88	.66	8.92	1.43	2.33	.58	.79	.05	2.55
Wisconsin	3.60	.20	6.98	2.33	1.32	.35	.66	.04	2.70
Minnesota	1.55	.26	1.90	------	.90	.10	1.27	.03	.48
Iowa	.37	.09	1.80	.22	.55	2.13	.17	.05	.29
Missouri	.67	.33	1.08	.40	1.21	.67	.55	.14	.43
North Dakota	.28	.28	1.06	.91	1.01	1.65	.38	.06	1.69
South Dakota	.74	.46	7.01	.19	.35	2.18	.17	.02	.46
Nebraska	.96	.17	2.37	.34	.76	1.19	.10	.06	.28
Kansas	1.17	.28	5.52	1.39	.81	1.96	.89	.19	2.22
Delaware	.89	.15	.89	.15	1.29	.00	.49	.05	.84
Maryland	2.30	.42	1.02	.44	1.38	.00	1.82	.21	1.43
District of Columbia	1.92	.52	5.44	------	.71	.00	2.28	.08	.40
Virginia	1.41	.27	5.82	------	.33	.03	.77	.63	3.47
West Virginia	.41	.16	1.36	------	.49	.26	.27	.25	1.05
North Carolina	.92	.17	1.08	------	.25	.18	------	.54	4.84
South Carolina	1.34	.43	.95	.56	.09	.04	.97	1.56	2.56
Georgia	.09	.05	1.55	.25	.15	.03	.20	.37	.55
Florida	.13	.19	1.74	.57	.03	.02	.21	.12	.13
Kentucky [1]	------	------	------	------	------	------	------	------	------
Tennessee	.40	.11	1.67	.15	.48	.24	[2].69	.57	.59
Alabama	.26	.15	1.88	.30	.15	.09	1.65	.30	.91
Mississippi	2.29	.18	1.57	2.76	.09	.08	1.89	1.28	9.73
Arkansas	.10	.04	.51	.16	.06	.07	[2].09	.28	.66
Louisiana	.16	.36	.25	.03	.39	.05	[2]1.23	.73	.17
Oklahoma [3]	.02	.19	1.29	.07	.29	1.58	.26	.26	.59
Texas	------	.11	------	------	.12	------	------	.13	------
Montana	.47	.04	1.80	1.02	1.49	.38	.24	.13	1.11
Idaho	1.60	.11	.98	.47	.28	.51	.21	.04	1.56
Wyoming	.33	.23	8.64	.52	.42	2.25	[2].05	.05	.42
Colorado	1.06	.21	17.52	3.57	.57	.41	1.00	.07	3.19
New Mexico	.99	1.11	4.70	.90	.60	.63	2.74	.18	.39
Arizona	.65	.10	5.50	1.09	.31	.27	3.57	.58	1.30
Utah [1]	------	------	------	------	------	------	------	------	------
Nevada	------	------	2.20	1.89	------	2.51	[2].31	------	------
Washington	1.33	.11	13.94	2.17	.47	1.03	1.06	.13	1.90
Oregon	1.20	.20	5.07	1.12	.51	.91	.71	.22	2.10
California	2.38	.54	15.00	4.17	1.00	.45	2.33	.22	2.19

[1] Reports received weekly. [2] Pulmonary. [3] Exclusive of Oklahoma City and Tulsa.

GENERAL CURRENT SUMMARY AND WEEKLY REPORTS FROM CITIES

The 97 cities reporting cases used in the following table are situated in all parts of the country and have an estimated aggregate population of more than 31,760,000. The estimated population of the 90 cities reporting deaths is more than 30,165,000. The estimated expectancy is based on the experience of the last nine years, excluding epidemics.

Weeks ended September 27, 1930, and September 28, 1929

	1930	1929	Estimated expectancy
Cases reported			
Diphtheria:			
46 States	1,058	1,564	----------
97 cities	350	499	682
Measles:			
45 States	445	745	----------
97 cities	113	77	----------
Meningococcus meningitis:			
46 States	66	111	----------
97 cities	24	55	----------
Poliomyelitis:			
46 States	595	143	----------
Scarlet fever:			
46 States	1,511	1,696	----------
97 cities	441	571	482
Smallpox:			
46 States	140	212	----------
97 cities	20	24	6
Typhoid fever:			
46 States	976	743	----------
97 cities	108	117	152
Deaths reported			
Influenza and pneumonia:			
90 cities	353	410	----------
Smallpox:			
90 cities	0	0	----------

City reports for week ended September 27, 1930

The "estimated expectancy" given for diphtheria, poliomyelitis, scarlet fever, smallpox, and typhoid fever is the result of an attempt to ascertain from previous occurrence the number of cases of the disease under consideration that may be expected to occur during a certain week in the absence of epidemics. It is based on reports to the Public Health Service during the past nine years. It is in most instances the median number of cases reported in the corresponding weeks of the preceding years. When the reports include several epidemics, or when for other reasons the median is unsatisfactory, the epidemic periods are excluded, and the estimated expectancy is the mean number of cases reported for the week during nonepidemic years.

If the reports have not been received for the full nine years, data are used for as many years as possible, but no year earlier than 1921 is included. In obtaining the estimated expectancy, the figures are smoothed when necessary to avoid abrupt deviation from the usual trend. For some of the diseases given in the table the available data were not sufficient to make it practicable to compute the estimated expectancy.

Division, State, and city	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
		Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
NEW ENGLAND								
Maine:								
Portland	0	0	0	----------	1	0	3	3
New Hampshire:								
Concord	0	0	0	----------	0	0	0	0
Vermont:								
Barre	0	0	0	----------	0	0	0	0
Burlington	0	0	0	----------	0	1	0	0
Massachusetts:								
Boston	8	19	14	----------	0	15	2	9
Fall River	3	3	2	----------	0	0	0	0
Springfield	4	3	1	----------	0	2	1	1
Worcester	3	4	2	----------	0	2	0	1
Rhode Island:								
Pawtucket	0	1	2	----------	0	0	0	0
Providence	0	4	1	----------	0	0	0	0
Connecticut:								
Bridgeport	0	4	0	----------	0	0	0	1
Hartford	2	2	1	----------	0	0	2	1
New Haven	1	0	0	1	0	0	0	0
MIDDLE ATLANTIC								
New York:								
Buffalo	7	11	6	----------	0	2	4	7
New York	20	97	28	2	3	11	12	86
Rochester	3	3	1	----------	0	0	0	3
Syracuse	4	2	0	----------	0	0	1	1
New Jersey:								
Camden	1	4	1	----------	0	3	0	0
Newark	2	11	11	----------	0	3	1	6
Trenton	0	2	1	----------	0	0	1	0
Pennsylvania:								
Philadelphia	10	39	9	1	0	6	5	31
Pittsburgh	5	16	12	----------	1	3	2	22
Reading	0	1	0	----------	0	0	2	2
EAST NORTH CENTRAL								
Ohio:								
Cincinnati	0	8	3	----------	0	0	0	6
Cleveland	7	35	5	5	0	2	5	6
Columbus	5	4	5	1	0	0	1	3
Toledo	0	7	4	----------	0	3	6	1
Indiana:								
Fort Wayne	0	2	0	----------	0	0	0	2
Indianapolis	1	11	2	----------	1	0	1	5
South Bend	1	1	0	----------	0	1	0	3
Terre Haute	0	0	0	----------	0	0	0	0
Illinois:								
Chicago	15	65	78	1	2	7	11	27
Springfield	0	0	0	----------	0	0	1	0
Michigan:								
Detroit	14	46	22	1	0	8	4	17
Flint	2	2	0	----------	0	1	3	2
Grand Rapids	4	2	0	----------	0	0	0	0
Wisconsin:								
Kenosha	15	0	0	----------	0	0	1	0
Madison	1	2	0	1	----------	0	1	----------
Milwaukee	16	9	4	----------	0	2	6	4
Racine	3	0	0	----------	0	0	0	0
Superior	0	0	0	----------	0	0	0	1

City reports for week ended September 27, 1930—Continued

Division, State, and city	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
		Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
WEST NORTH CENTRAL								
Minnesota:								
Duluth	5	1	0	----	0	0	0	1
Minneapolis	9	23	4	----	0	1	8	2
St. Paul	4	12	1	----	0	0	0	3
Iowa:								
Davenport	0	0	0	----	----	0	0	----
Des Moines	1	2	0	----	----	0	0	----
Sioux City	1	2	0	----	----	0	0	----
Waterloo	4	1	0	----	----	0	0	----
Missouri:								
Kansas City	----	6	----	----	----	----	----	----
St. Joseph	0	1	0	----	0	0	0	0
St. Louis	2	27	16	----	----	13	4	----
North Dakota:								
Fargo	0	1	0	----	0	0	15	1
Grand Forks	0	0	0	----	----	0	1	----
South Dakota:								
Sioux Falls	0	1	0	----	----	0	0	----
Nebraska:								
Omaha	0	9	1	----	0	1	0	0
Kansas:								
Topeka	1	1	1	----	0	0	0	0
Wichita	0	2	2	----	0	0	0	1
SOUTH ATLANTIC								
Delaware:								
Wilmington	0	1	0	----	0	0	0	0
Maryland:								
Baltimore	9	17	8	1	0	2	0	11
Cumberland	0	1	0	1	0	0	0	1
Frederick	0	1	0	----	0	0	0	0
District of Columbia:								
Washington	1	10	13	----	0	3	0	8
Virginia:								
Lynchburg	0	3	2	----	0	0	0	0
Norfolk	1	3	1	----	0	0	1	1
Richmond	0	18	1	----	0	0	0	2
Roanoke	0	5	4	----	0	0	0	0
West Virginia:								
Charleston	0	1	1	----	0	0	8	0
Wheeling	0	1	0	----	0	0	0	3
North Carolina:								
Raleigh	0	4	0	----	0	0	0	0
Wilmington	0	1	10	----	0	0	0	1
Winston-Salem	1	4	4	----	0	0	0	0
South Carolina:								
Charleston	0	1	0	3	0	0	0	0
Columbia	0	1	0	----	0	0	1	1
Georgia:								
Atlanta	0	8	7	5	0	0	0	1
Brunswick	0	0	0	----	0	0	0	0
Savannah	0	1	0	----	0	0	0	0
Florida:								
Miami	0	2	2	----	0	0	0	1
St. Petersburg	----	0	----	----	0	----	----	0
Tampa	0	1	0	2	2	0	0	0
EAST SOUTH CENTRAL								
Kentucky:								
Covington	0	1	0	----	0	0	0	4
Tennessee:								
Memphis	1	5	5	----	0	0	0	1
Nashville	0	3	0	----	0	8	0	1
Alabama:								
Birmingham	0	5	0	----	2	3	0	3
Mobile	0	1	0	1	0	0	0	1
Montgomery	0	3	0	1	----	0	0	----

City reports for week ended September 27, 1930—Continued

Division, State, and city	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
		Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
WEST SOUTH CENTRAL								
Arkansas:								
Fort Smith	0	1	0			0	0	
Little Rock	0	1	0		0	0	0	0
Louisiana:								
New Orleans	3	8	15	3	0	0	0	10
Shreveport	0	1	0		0	0	0	2
Oklahoma:								
Oklahoma City	0	3	0		0	0	0	5
Tulsa	0	4	2			0	1	
Texas:								
Dallas	0	11	10	1	1	1	3	2
Fort Worth	0	3	0		0	0	0	1
Galveston	0	0	0		0	0	0	3
Houston	0	5	9		0	2	0	2
San Antonio	0	2	5		0	0	0	1
MOUNTAIN								
Montana:								
Billings	0	0	0		0	0	0	2
Great Falls	0	0	0		0	0	0	0
Helena	0	0	0		0	0	0	0
Missoula	0	1	0		0	0	0	1
Idaho:								
Boise	0	1	0		0	0	0	0
Colorado:								
Denver	3	10	7		0	0	0	3
Pueblo	1	1	0		0	1	2	0
New Mexico:								
Albuquerque	0	0	0		0	0	0	1
Arizona:								
Phoenix	0	0	2		0	0	0	1
Utah:								
Salt Lake City	1	3	0		0	2	0	0
Nevada:								
Reno	0	0	0		0	0	0	0
PACIFIC								
Washington:								
Seattle	7	3	1			0	6	
Spokane	8	2	0			2	0	
Tacoma	0	2	0		0	0	0	3
Oregon:								
Salem	0	0	1		0	0	0	0
California:								
Los Angeles	4	26	6	20	0	5	6	9
Sacramento	1	2	0	1	1	1	12	2
San Francisco	26	13	6		1	0	10	2

Division, State, and city	Scarlet fever		Smallpox			Tuberculosis, deaths reported	Typhoid fever			Whooping cough, cases reported	Deaths, all causes
	Cases, estimated expectancy	Cases reported	Cases, estimated expectancy	Cases reported	Deaths reported		Cases, estimated expectancy	Cases reported	Deaths reported		
NEW ENGLAND											
Maine:											
Portland	1	2	0	0	0	0	1	0	0	15	25
New Hampshire:											
Concord	0	1	0	0	0	0	0	2	0	0	6
Vermont:											
Barre	0	0	0	0	0	1	0	0	0	0	2
Burlington	0	0	0	0	0	0	0	1	0	0	5
Massachusetts:											
Boston	24	17	0	0	0	13	3	1	0	41	180
Fall River	1	2	0	0	0	1	1	2	0	1	27
Springfield	2	0	0	0	0	4	0	0	0	4	39
Worcester	5	5	0	0	0	3	0	0	0	9	35

City reports for week ended September 27, 1930—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuberculosis, deaths reported	Typhoid fever			Whooping cough, cases reported	Deaths, all causes
	Cases, estimated expectancy	Cases reported	Cases, estimated expectancy	Cases reported	Deaths reported		Cases, estimated expectancy	Cases reported	Deaths reported		
NEW ENGLAND—continued											
Rhode Island:											
Pawtucket	0	0	0	0	0	0	0	0	0	0	18
Providence	2	0	0	0	0	4	2	0	0	3	46
Connecticut:											
Bridgeport	2	4	0	0	0	1	0	0	0	3	24
Hartford	1	1	0	0	0	0	0	0	0	1	30
New Haven	1	4	0	0	0	0	1	0	0	3	10
MIDDLE ATLANTIC											
New York:											
Buffalo	8	5	0	0	0	6	2	2	1	24	119
New York	41	16	0	0	0	78	34	12	1	97	1,192
Rochester	3	0	0	0	0	1	2	0	0	6	67
Syracuse	3	3	0	0	0	1	1	0	0	7	36
New Jersey:											
Camden	2	2	0	0	0	0	1	0	0	0	30
Newark	5	3	0	0	0	6	2	2	0	24	85
Trenton	1	6	0	0	0	3	0	1	0	1	29
Pennsylvania:											
Philadelphia	28	22	0	0	0	18	12	12	2	13	426
Pittsburgh	19	13	0	0	0	2	2	0	0	9	177
Reading	0	0	0	0	0	2	0	0	0	0	28
EAST NORTH CENTRAL											
Ohio:											
Cincinnati	7	11	0	0	0	8	2	2	0	6	148
Cleveland	17	19	0	0	0	9	3	2	0	18	164
Columbus	5	2	0	4	0	2	1	0	0	0	76
Toledo	5	9	0	1	0	2	2	1	0	2	74
Indiana:											
Fort Wayne	1	0	0	0	0	1	1	1	0	0	36
Indianapolis	7	11	0	0	0	5	2	1	0	2	----
South Bend	2	2	0	0	0	1	0	0	0	0	25
Terre Haute	1	0	0	0	0	0	0	0	0	0	16
Illinois:											
Chicago	45	62	0	0	0	45	6	3	0	54	666
Springfield	2	0	0	0	0	0	1	0	0	1	13
Michigan:											
Detroit	37	33	0	0	0	22	4	3	0	63	218
Flint	7	15	0	0	0	2	1	1	0	1	35
Grand Rapids	5	9	0	0	0	0	1	0	0	1	37
Wisconsin:											
Kenosha	0	3	0	0	0	0	0	0	0	2	9
Madison	0	1	0	0	----	----	0	0	----	1	----
Milwaukee	13	9	0	0	0	4	0	1	0	18	101
Racine	3	12	0	0	0	1	0	1	0	4	13
Superior	1	0	0	0	0	1	0	0	0	2	9
WEST NORTH CENTRAL											
Minnesota:											
Duluth	5	1	0	0	0	1	0	0	0	9	26
Minneapolis	26	4	0	0	0	2	2	0	0	1	87
St. Paul	11	2	1	0	0	2	1	1	0	2	45
Iowa:											
Davenport	1	0	0	2	----	----	0	0	----	0	----
Des Moines	3	2	0	1	----	----	0	0	----	0	24
Sioux City	1	3	0	0	----	----	1	0	----	1	2
Waterloo	1	1	0	0	----	----	0	0	----	1	----
Missouri:											
Kansas City	7	----	0	----	----	----	2	----	----	----	----
St. Joseph	2	2	0	0	0	1	0	0	0	1	23
St. Louis	13	14	0	0	0	21	5	6	1	1	204
North Dakota:											
Fargo	2	0	0	0	0	0	0	0	0	3	6
Grand Forks	1	1	0	0	----	----	0	0	----	0	----
South Dakota:											
Sioux Falls	1	0	0	1	----	----	0	0	----	0	9

City reports for week ended September 27, 1930—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuberculosis, deaths reported	Typhoid fever			Whooping cough, cases reported	Deaths, all causes
	Cases, estimated expectancy	Cases reported	Cases, estimated expectancy	Cases reported	Deaths reported		Cases, estimated expectancy	Cases reported	Deaths reported		
WEST NORTH CENTRAL—contd.											
Nebraska:											
Omaha	2	7	0	7	0	3	0	0	0	3	34
Kansas:											
Topeka	2	0	0	0	0	0	0	0	0	0	13
Wichita	3	0	0	0	0	1	1	0	0	0	26
SOUTH ATLANTIC											
Delaware:											
Wilmington	2	0	0	0	0	0	0	0	0	0	39
Maryland:											
Baltimore	8	3	0	0	0	16	8	3	1	11	178
Cumberland	0	2	0	0	0	1	1	0	0	0	14
Frederick	0	0	0	0	0	0	0	0	0	0	3
District of Col.:											
Washington	8	3	0	0	0	6	3	2	0	2	127
Virginia:											
Lynchburg	1	0	0	0	0	0	1	2	0	0	5
Norfolk	0	2	0	0	0	1	0	1	1	0	----
Richmond	7	7	0	0	0	5	1	4	0	0	42
Roanoke	3	4	0	0	0	0	0	2	0	5	18
West Virginia:											
Charleston	1	0	0	0	0	0	1	1	1	1	25
Wheeling	2	1	0	0	0	0	0	0	0	0	16
North Carolina:											
Raleigh	0	1	0	0	0	2	0	0	0	0	18
Wilmington	1	0	0	0	0	0	0	0	0	0	12
Winston-Salem	4	2	0	0	0	2	1	1	0	2	22
South Carolina:											
Charleston	0	1	1	0	0	3	2	3	0	0	29
Columbia	1	1	0	0	0	1	0	1	0	0	11
Georgia:											
Atlanta	6	5	0	0	0	3	2	2	3	0	63
Brunswick	0	0	0	0	0	0	0	0	0	0	5
Savannah	0	0	0	0	0	6	1	7	0	0	32
Florida:											
Miami	0	2	0	0	0	0	0	1	1	0	19
St. Petersburg	0	----	0	----	0	0	0	----	1	----	7
Tampa	0	1	0	0	0	1	0	0	0	1	12
EAST SOUTH CENTRAL											
Kentucky:											
Covington	1	1	0	0	0	2	0	0	1	0	26
Tennessee:											
Memphis	3	2	0	0	0	4	4	0	0	0	50
Nashville	2	5	0	0	0	2	4	0	1	0	50
Alabama:											
Birmingham	6	8	0	0	0	1	3	2	0	6	62
Mobile	0	2	0	0	0	0	0	0	0	0	17
Montgomery	1	1	0	0	----	----	0	1	----	0	----
WEST SOUTH CENTRAL											
Arkansas:											
Fort Smith	1	1	0	0	----	----	0	0	----	0	----
Little Rock	2	0	0	0	0	2	1	1	0	0	----
Louisiana:											
New Orleans	3	5	0	1	0	7	4	7	0	0	144
Shreveport	1	0	0	0	0	0	0	1	1	0	24
Oklahoma:											
Oklahoma City	1	8	0	1	0	1	2	1	0	0	51
Tulsa	4	3	0	0	----	----	1	6	----	0	----
Texas:											
Dallas	3	8	0	0	0	1	2	1	0	3	45
Fort Worth	1	0	0	0	0	0	1	0	1	0	29
Galveston	1	0	0	0	0	0	0	0	0	0	12
Houston	1	0	0	0	0	3	1	0	0	0	67
San Antonio	0	1	0	0	0	10	1	0	2	1	47
MOUNTAIN											
Montana:											
Billings	1	0	0	0	0	0	0	1	1	1	8
Great Falls	1	3	0	0	0	0	0	0	0	0	11
Helena	0	2	0	0	0	0	0	0	0	0	4
Missoula	0	0	1	0	0	0	0	0	0	0	6

City reports for week ended September 27, 1930—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuberculosis, deaths reported	Typhoid fever			Whooping cough, cases reported	Deaths, all causes
	Cases, estimated expectancy	Cases reported	Cases, estimated expectancy	Cases reported	Deaths reported		Cases, estimated expectancy	Cases reported	Deaths reported		
MOUNTAIN—contd.											
Idaho:											
Boise	0	0	0	0	0	0	1	0	0	0	4
Colorado:											
Denver	6	4	0	0	0	5	3	2	0	24	74
Pueblo	1	0	0	0	0	0	0	1	0	0	9
New Mexico:											
Albuquerque	0	2	0	0	0	4	2	2	0	0	9
Arizona:											
Phoenix	1	2	0	0	0	2	0	0	0	0	8
Utah:											
Salt Lake City	3	2	0	0	0	0	2	1	0	15	15
Nevada:											
Reno	0	0	0	0	0	0	0	0	0	0	3
PACIFIC											
Washington:											
Seattle	6	14	1	0	----	----	2	1	----	8	----
Spokane	3	2	1	1	----	----	1	0	----	1	----
Tacoma	1	1	1	3	0	2	1	1	0	0	19
Oregon:											
Salem	1	0	1	0	0	0	1	0	0	0	----
California:											
Los Angeles	12	12	0	0	0	20	3	2	0	23	242
Sacramento	2	2	0	1	0	1	1	1	0	5	21
San Francisco	3	6	0	3	0	4	1	1	0	14	169

Division, State, and city	Meningococcus meningitis		Lethargic encephalitis		Pellagra		Poliomyelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, estimated expectancy	Cases	Deaths
NEW ENGLAND									
Maine:									
Portland	0	0	0	0	0	0	0	7	1
Massachusetts:									
Boston	0	0	1	0	0	0	3	17	1
Worcester	0	0	1	1	0	0	0	0	0
Connecticut:[1]									
Hartford	0	0	0	0	0	0	1	1	0
MIDDLE ATLANTIC									
New York:									
Buffalo	0	0	0	0	0	0	1	4	0
New York	3	2	2	2	0	0	20	4	0
Rochester	0	0	0	0	0	0	1	9	3
Syracuse	0	0	0	0	0	0	1	12	4
Pennsylvania:									
Philadelphia	1	0	1	1	0	0	1	4	0
Pittsburgh	0	1	0	0	0	0	0	1	0
EAST NORTH CENTRAL									
Ohio:									
Cincinnati	0	0	0	0	0	0	0	6	2
Cleveland	2	1	0	0	0	0	1	21	5
Columbus	0	0	0	0	0	0	0	3	0
Toledo	0	0	1	1	0	0	0	3	0
Indiana:									
Indianapolis	1	1	0	0	0	0	0	3	0
Illinois:									
Chicago	4	2	0	0	1	1	3	25	1
Michigan:									
Detroit	5	1	2	0	0	0	4	6	0
Grand Rapids	0	0	0	0	0	0	1	0	1
Wisconsin:									
Kenosha	0	0	0	0	0	0	0	1	0
Milwaukee	0	0	0	0	0	0	0	16	0

[1] Typhus fever, 2 cases: 1 case at New Haven, Conn., and 1 case at Savannah, Ga.

City reports for week ended September 27, 1930—Continued

Division, State, and city	Meningococcus meningitis		Lethargic encephalitis		Pellagra		Poliomyelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, estimated expectancy	Cases	Deaths
WEST NORTH CENTRAL									
Minnesota:									
Duluth	0	0	0	0	0	0	0	1	0
Minneapolis	0	0	0	0	0	0	0	4	0
Iowa:									
Sioux City	0	0	0	0	0	0	0	5	1
Waterloo	0	0	0	0	0	0	0	2	0
Missouri:									
St. Joseph	0	0	0	0	0	0	0	1	0
St. Louis	2	0	0	0	0	0	0	0	0
South Dakota:									
Sioux Falls	0	0	0	0	0	0	0	1	0
Kansas:									
Topeka	0	0	0	0	0	0	0	1	0
Wichita	0	0	0	0	0	0	0	7	0
SOUTH ATLANTIC									
Maryland:									
Baltimore	0	0	0	0	0	0	1	1	0
Virginia:									
Lynchburg	0	0	0	0	0	0	0	1	0
Norfolk	0	0	0	0	0	0	1	4	0
Richmond	0	0	0	1	0	0	0	0	0
North Carolina:									
Raleigh	0	0	0	0	3	2	0	0	0
Winston-Salem	0	0	0	0	0	1	0	0	0
South Carolina:									
Charleston	0	0	0	0	0	1	0	0	0
Georgia: [1]									
Atlanta	0	0	0	0	1	1	0	0	0
Florida:									
Miami	0	0	0	0	1	0	0	0	0
Tampa	0	0	0	0	0	0	0	1	1
EAST SOUTH CENTRAL									
Kentucky:									
Covington	0	0	0	0	0	0	0	1	0
Tennessee:									
Memphis	0	0	0	0	0	0	1	2	0
Alabama:									
Birmingham	1	1	0	0	0	2	0	0	0
WEST SOUTH CENTRAL									
Louisiana:									
New Orleans	0	0	0	0	2	2	0	0	0
Oklahoma:									
Tulsa	0	0	0	0	0	0	0	1	0
Texas:									
Dallas	0	0	0	0	1	1	0	0	0
Fort Worth	0	0	0	0	0	1	0	0	0
Houston	0	0	0	0	0	1	0	1	1
MOUNTAIN									
Colorado:									
Denver	1	1	0	0	0	0	1	0	0
New Mexico:									
Albuquerque	0	0	0	0	0	1	0	0	0
Utah:									
Salt Lake City	1	2	0	0	0	0	0	2	0
PACIFIC									
Washington:									
Seattle	1	0	0	0	0	0	1	1	0
California:									
Los Angeles	1	1	0	0	1	0	1	6	4
San Francisco	1	1	1	0	0	0	1	22	2

[1] Typhus fever, 2 cases: 1 case at New Haven, Conn., and 1 case at Savannah, Ga.

The following table gives the rates per 100,000 population for 98 cities for the 5-week period ended September 27, 1930, compared with those for a like period ended September 28, 1929. The population figures used in computing the rates are approximate estimates, authoritative figures for many of the cities not being available. The 98 cities reporting cases have an estimated aggregate population of more than 32,000,000. The 91 cities reporting deaths have more than 30,500,000 estimated population.

Summary of weekly reports from cities, August 24 to September 27, 1930—Annual rates per 100,000 population, compared with rates for the corresponding period of 1929 [1]

DIPHTHERIA CASE RATES

	Week ended—									
	Aug. 30, 1930	Aug. 31, 1929	Sept. 6, 1930	Sept. 7, 1929	Sept. 13, 1930	Sept. 14, 1929	Sept. 20, 1930	Sept. 21, 1929	Sept. 27, 1930	Sept. 28, 1929
98 cities	[2] 39	62	41	[2] 64	45	66	47	75	[4] 57	83
New England	49	45	35	[3] 46	55	47	31	49	51	76
Middle Atlantic	31	54	31	45	28	41	38	54	33	60
East North Central	46	75	49	86	64	95	75	96	75	90
West North Central	27	25	34	38	55	58	47	64	[4] 56	100
South Atlantic	[2] 60	90	60	92	62	133	42	114	92	112
East South Central	13	116	54	75	27	116	27	137	34	137
West South Central	71	137	60	133	49	61	67	149	146	164
Mountain	69	17	43	70	34	26	26	70	60	26
Pacific	19	27	38	34	26	22	14	19	31	65

MEASLES CASE RATES

98 cities	[2] 20	14	24	[2] 12	16	16	16	15	[4] 19	13
New England	18	20	33	[3] 21	38	16	18	31	42	18
Middle Atlantic	23	8	28	7	20	12	17	7	13	10
East North Central	8	22	13	16	9	20	14	17	13	13
West North Central	27	8	30	2	15	6	19	6	[4] 33	10
South Atlantic	[2] 30	13	26	2	5	7	20	7	9	13
East South Central	13	7	27	14	7	7	0	7	74	0
West South Central	11	8	0	4	4	11	0	8	11	11
Mountain	34	44	51	26	34	61	43	26	26	44
Pacific	35	19	40	46	19	39	21	51	19	24

SCARLET FEVER CASE RATES

98 cities	[2] 42	41	43	[2] 52	51	54	62	68	[4] 72	95
New England	51	38	55	[3] 83	51	52	71	49	80	99
Middle Atlantic	28	16	25	25	27	16	47	25	33	42
East North Central	48	63	47	70	85	90	91	121	118	161
West North Central	42	44	57	67	34	58	44	92	[4] 76	108
South Atlantic	[2] 67	45	66	64	51	47	40	66	57	105
East South Central	115	34	67	41	40	96	40	28	128	75
West South Central	15	72	67	34	26	91	56	72	56	72
Mountain	86	61	34	17	77	70	69	113	94	139
Pacific	31	46	33	77	73	72	78	68	87	84

[1] The figures given in this table are rates per 100,000 population, annual basis, and not the number of cases reported. Populations used are estimates as of July 1, 1930 and 1929, respectively.
[2] Columbia, S. C., not included.
[3] Pawtucket, R. I., not included.
[4] Kansas City, Mo., not included.

Summary of weekly reports from cities, August 24 to September 27, 1930—Annual rates per 100,000 population, compared with rates for the corresponding period of 1929—Continued

SMALLPOX CASE RATES

	Week ended—									
	Aug. 30, 1930	Aug. 31, 1929	Sept. 6, 1930	Sept. 7, 1929	Sept. 13, 1930	Sept. 14, 1929	Sept. 20, 1930	Sept. 21, 1929	Sept. 27, 1930	Sept. 28, 1929
98 cities	[2] 2	4	3	[2] 4	3	3	5	5	[4] 3	4
New England	0	0	0	[3] 0	0	0	0	0	0	0
Middle Atlantic	0	0	0	0	0	0	0	0	0	0
East North Central	0	10	3	10	2	4	9	10	3	3
West North Central	8	4	13	2	27	8	21	6	[4] 16	8
South Atlantic	[3] 0	0	4	0	0	2	0	0	0	0
East South Central	0	0	0	0	0	0	0	0	0	0
West South Central	4	4	0	0	0	0	0	0	4	0
Mountain	0	0	0	9	0	9	0	52	0	96
Pacific	12	14	14	14	9	12	5	17	19	10

TYPHOID FEVER CASE RATES

98 cities	[2] 25	27	21	[2] 18	27	21	22	22	[4] 18	20
New England	11	29	11	[3] 2	20	16	11	13	11	7
Middle Atlantic	21	28	22	20	25	18	16	14	14	12
East North Central	10	13	12	13	17	10	11	11	9	9
West North Central	19	23	13	12	21	17	28	6	[4] 16	23
South Atlantic	[3] 82	52	53	34	64	34	62	26	51	17
East South Central	47	103	54	55	54	89	54	0	20	82
West South Central	71	50	49	15	56	50	67	84	37	27
Mountain	43	17	9	44	60	70	0	340	43	313
Pacific	9	12	9	14	5	19	17	7	14	10

INFLUENZA DEATH RATES

91 cities	[2] 4	2	3	[2] 3	3	3	3	2	[4] 3	5
New England	0	0	0	[3] 2	0	0	2	2	2	2
Middle Atlantic	3	2	3	2	4	2	2	0	2	5
East North Central	4	2	2	6	3	2	3	2	2	4
West North Central	3	0	6	0	0	6	0	6	[4] 0	3
South Atlantic	[3] 7	2	7	4	2	2	0	2	4	6
East South Central	7	0	0	7	22	7	29	7	15	0
West South Central	8	4	11	0	0	12	8	0	4	12
Mountain	0	9	9	0	0	9	17	9	0	17
Pacific	3	0	0	3	0	0	0	9	6	3

PNEUMONIA DEATH RATES

91 cities	[2] 53	55	55	[2] 57	55	55	58	54	[4] 58	67
New England	47	49	51	[3] 44	62	36	51	29	35	72
Middle Atlantic	60	61	68	75	67	66	68	59	76	72
East North Central	50	51	36	44	43	47	43	47	48	54
West North Central	38	33	50	57	44	45	74	39	[4] 31	81
South Atlantic	[3] 52	56	62	64	53	52	51	66	51	60
East South Central	52	52	103	75	29	90	81	67	74	119
West South Central	38	98	54	31	61	55	50	51	77	94
Mountain	51	44	51	52	120	70	112	104	51	70
Pacific	55	28	34	31	31	41	49	57	49	38

[2] Columbia, S. C., not included.
[3] Pawtucket, R. I., not included.
[4] Kansas City, Mo., not included.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended September 20, 1930.—The Department of Pensions and National Health of Canada reports cases of certain communicable diseases for the week ended September 20, 1930, as follows:

Province	Cerebro-spinal fever	Dysentery	Influenza	Lethargic encephalitis	Poliomyelitis	Smallpox	Typhoid fever
Prince Edward Island [1]							
Nova Scotia	1		7				
New Brunswick							4
Quebec					3		30
Ontario	8		5		53		33
Manitoba					8		
Saskatchewan				1	6		6
Alberta					11	1	3
British Columbia		11			3	1	4
Total	9	11	12	1	84	2	80

[1] No case of any disease included in the table was reported during the month.

Quebec Province—Communicable diseases—Week ended September 27, 1930.—The Bureau of Health of the Province of Quebec, Canada, reports cases of certain communicable diseases for the week ended September 27, 1930, as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	1	Ophthalmia neonatorum	1
Chicken pox	31	Puerperal fever	1
Diphtheria and croup	41	Scarlet fever	56
Influenza	1	Tuberculosis	41
Measles	47	Typhoid fever	39
Mumps	5	Whooping cough	77

CUBA

Provinces—Communicable diseases—Four weeks ended August 30, 1930.—During the four weeks ended August 30, 1930, cases of certain communicable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	1	1		5	1		8
Chicken pox	1	1		1			3
Diphtheria		20	2	2	1	2	27
Malaria		13		3	14	31	64
Measles		1	1	1			3
Paratyphoid fever	3	2	1	2		7	15
Scarlet fever		6					6
Tetanus, infantile				1			1
Typhoid fever	3	31	15	50	8	21	128

DENMARK

Communicable diseases—July, 1930.—During the month of July, 1930, cases of certain communicable diseases were reported in Denmark as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	18	Paratyphoid fever	30
Chicken pox	10	Poliomyelitis	5
Diphtheria and croup	325	Puerperal fever	18
Erysipelas	264	Scabies	480
German measles	5	Scarlet fever	160
Influenza	2,406	Typhoid fever	12
Lethargic encephalitis	15	Undulant fever (Bac. abort. Bang)	27
Measles	1,609	Whooping cough	1,265
Mumps	546		

GERMANY

Bavaria—Vital statistics—First quarter of 1930.—The numbers of births, deaths, marriages, and deaths of infants under 1 year which occurred in Bavaria during the first quarter of 1930, as compared with the corresponding period of 1929 and 1913, are given in the following table:

Vital statistics for the first quarter of the years 1930, 1929, and 1913

	1930	1929	1913
Births	39,947	41,243	52,427
Deaths	25,719	32,023	34,209
Marriages	13,771	12,548	10,708
Deaths under 1 year	4,319	5,415	9,098

Estimated population of Bavaria during 1929, 7,510,435.

JAMAICA

Communicable diseases—Four weeks ended August 16, 1930.—During the four weeks ended August 16, 1930, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island of Jamaica outside of Kingston, as follows:

Disease	Cases		Disease	Cases	
	Kingston	Other localities		Kingston	Other localities
Cerebrospinal meningitis	----	2	Poliomyelitis	----	1
Chicken pox	2	16	Puerperal fever	----	1
Dysentery	----	1	Tuberculosis	36	55
Erysipelas	----	3	Typhoid fever	6	62
Leprosy	----	1			

PERU

Eradication of bubonic plague.—According to a decree dated September 5, 1930, promulgated by the President of the Provisional Government of Peru, the National Public Health Service is authorized to carry on a campaign to combat, until eradicated, bubonic plague in the Republic of Peru. The work will be carried on with the cooperation of the Pan American Sanitary Bureau, and Medical Director J. D. Long and Surgeon C. R. Eskey, officers of the United States Public Health Service, who have been designated technical adviser and epidemiologist, respectively, by the National Public Health Service of Peru for the purposes of the campaign.

PORTO RICO

San Juan—Communicable diseases—Five weeks ended September 13, 1930.—During the five weeks ended September 13, 1930, cases of certain communicable diseases were reported in San Juan, Porto Rico, as follows:

Disease	Cases	Disease	Cases
Diphtheria	5	Tuberculosis	79
Malaria	7	Typhoid fever	6
Measles	1		

TRINIDAD (BRITISH WEST INDIES)

Health conditions—Year 1929.—During the year 1929 health conditions on the island of Trinidad were good notwithstanding a limited outbreak of poliomyelitis in the Siparia district. The general death rate was 19.4 per 1,000 population, which is slightly less than that for the preceding year. The venereal diseases, malaria, dysentery, and tuberculosis are the most prevalent diseases. Venereal-disease infection continued high. The death rate from malaria during 1929 was the lowest on record, and dysentery and diarrhea and enteritis were also less prevalent than during the preceding year. There was also a decrease in the number of cases of typhoid fever, 228 cases being reported during 1929 as compared with 295 in 1927 and 250 in 1928. Hookworm infection was still high.

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

From medical officers of the Public Health Service, American consuls, International Office of Public Hygiene, Pan American Sanitary Bureau ,health section of the League of Nations, and other sources. 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.

CHOLERA

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27	Oct. 4, 1930
Afghanistan	C						[1] P						P						
China:																			
Canton	C			3	2		2	1											
	D						1												
Shanghai	C													8	4		22		
	D														1	1	1		
Tientsin	C												2	1					
Swatow	C			3	7					1									
India	C	10,817	41,462	56,311	37,102	6,728	5,520	5,701	8,172	7,199	11,597	11,993							
	D	5,866	27,906	44,878	25,711	3,712	3,095	3,133	3,882	3,676	6,033	6,345							
Bassein	C		7	7											1				
	D		5	5															
Bombay	C	4								3	2	3	6						
	D									2	1	1	4						
Calcutta	C	354	647	609	327	81	53	49	37	18	10	17	18	8	10		9		
	D	220	414	372	179	54	28	23	23	7	7	10	6	3	3		6		
Negapatam	C				1														
	D				1														
Rangoon	C	2	1	9	6	1				1					1				
	D	2	1	3	4	1				1									
India (French):																			
Chandernagor	C	1	6	6	3			1											
	D	3	5	6	2														
Karikal	C	12	1		3														
	D	9	1		3														
India: Portuguese	C											1							
Indo-China (see also table below):																			
Pnompenh	C		2	1	40	9	16	7		5	3	3							
	D	6		5	18	6	9	5	2	3	2	1							
Saigon and Cholon	C	14	76	160	48	7	1	1	1	2	2	1							
	D	6	55	101	23	3					1								

Philippine Islands:[2]																			
Ports—																			
Cebu	C				1	20	13	13	9	5	4			1	1				
	D					9	7	12	8	4	4			1	1				
Iloilo	C						1	11	17	9	19	32	4	3	4	1	2	1	
	D							9	13	10	18	19	9	4	5	2	3	2	
Manila	C			1							1			16	13	5	4	2	1
	D													5	6	1			1
Provinces—																			
Antique	C									14	3		3	2	21	12	8	12	
	D									7	3		2	1	10	12	7	8	
Bohol	C										54	54	30	30	26	4	1		
	D										29	32	18	12	11	2			
Bulacan	C				3		1	1	2	1				1	1	1			2
	D				1									1					1
Capiz	C							2	1	1	1			1					
	D							1	1	1	1								
Cebu	C			1	355	313	222	115	63	55	26	4		1	16	8			
	D			1	170	131	83	60	29	32	15	3		1	6	4			
Iloilo	C				2	20	45	85	159	143	209	127	92	71	57	66	46	18	7
	D				1	10	23	64	96	77	143	91	65	45	44	37	26	15	6
La Union	C						1												
Leyte	C				47	11													
	D				19	11													
Masbate	C				10	14	32	25	21	15	19								
	D				3	2	18	5	10	5	9								
Misamis, Occidental	C					1	2												
	D					1	2												
Negros, Occidental	C			10	140	62	163	172	171	151	79	45	68	40	47	21	15	8	10
	D			7	88	42	90	122	114	97	64	33	43	32	32	18	9	6	5
Negros, Oriental	C					1	1	6	15	6	2								
	D						1	3	9	1	3								
Nueva Acija	C								1										
	D								1										
Pampanga	C				2	2								1					
Pangasinan	C					2		1		1									
	D					2				1									
Rizal	C				1		1												
	D				1		1												
Samar	C										5	3	10			2	1	4	
	D										5	3	8			2		4	
Surigao	C										15	2	11	1					
	D										9	2	6	2					
Tarlac	C									1									
Siam	C	1	20	33	27	8	3	8	1		1	1	1						
	D		13	21	19	4	1	3	1		1		1						
Bangkok	C	2	15	9	12		3	4	1	1		1							
	D	1	4	3	5		1	1	1	1		1							
Nagara Pathom	C		10																
	D		2																

[1] An outbreak of cholera was reported in June, 1930, in Afghanistan.
[2] Figures for cholera in the Philippine Islands are subject to correction.

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

CHOLERA—Continued

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27	Oct. 4, 1930
Siam—Continued.																			
Songkla	C						8		2										
	D						4		2										
On vessel:																			
S. S. Malwa from Shanghai	D																1		
S. S. Sassari at Massoua, from Jeddah	C			1															
	D			1															
On small boat at Port Cebu, from Bantayan Island	C				1														
	D				1														

Place		February, 1930	March, 1930	April, 1930	May, 1930	June, 1930 1–10	11–20	21–30	July, 1930 1–10	11–20	21–31	August, 1930 1–10	11–20	21–31	Sept. 1–10, 1930
Indo-China (French) (see also table above):															
Annam [1]	C	4	52	60	23	2	14				1		3		
Cambodia [1]	C	90	81	24	88	56	88				43	37	22		28
Cochin-China [1]	C	65	82	48	671	147	126				46	22	5		9

[1] Reports incomplete.

PLAGUE

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930				August, 1930					September, 1930				Oct. 4, 1930
						5	12	19	26	2	9	16	23	30	6	13	20	27	
Algeria:																			
Algiers	C						1	2			1		6	1	2	6	2		
Constantine	C				1	1													
Oran	C							2	1	1	1	1	1	1	2	3	4		
Philippeville	C														1			1	
Azores: Ponta Delgada	C		8																
	D		5																
Belgian Congo	C					2				1		1		2	3				
	D					2				1		1			3				
British East Africa (see also table below):																			
Tanganyika	C		44																
	D		20																
Uganda	C	98	117	227	406		50	100	78	52	67								
	D	87	105	195	328		47	97	69	50	64								
Canary Islands: Las Palmas	D									1									
Ceylon:																			
Colombo	C	4	1	6	1	1	2					1	1	2				P	
	D	4	1	5	1	1	2					1	1	3					
Plague-infected rats		2	4		1														
Chile: Antofagasta	C	1	1																
	D	2	1																
China: Manchuria—Tungliau and Nungan	C												30			29	P		
Dutch East Indies:																			
Batavia and West Java	C	124	87	82	98	19	25	18	22	19	30	20							
	D	122	81	82	98	19	25	18	22	19	30	20							
Plague-infected rats		3	8	5	4						1				1	1			
Java and Madura	D	223	173	185	202	55	56	58	48	45	51	47	45	55					
Ecuador (see table below).																			
Egypt:																			
Alexandria	C	4	2	13	19	8	8	3	4	2	2	4	3	3	3	2	2	2	1
	D	1	2	3	9	2	3	2	3	2	1	3		5	1	2		3	1
Assiout	C		14	20	9	1	1												
	D		5	5	3	1	1												
Beheira	C	4																	
Beni-Suef	C	4											1						
Dakahlieh	C	5	5	11	1														
	D	1		2															
Gharbieh	C		1							3									
	D		1							1									
Girga	C		1				1							1					
Minieh	C			7	10	2	1												
	D			1	1														
Port Said	C		1	2	2				1	1									

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

PLAGUE—Continued

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27	Oct. 4, 1930
France:																			
Marseilles	C													2	2	1			
St. Ouen	C							1											
	D							1											
Gambia	C												4						
	D												4						
Greece (see also table below):																			
Patras	C	1			1			1											
Pyrgos	C	1																	2
Hawaii Territory, Hamakua, Hawaii: Plague-infected rats					1														
India	C	4,087	2,215	648	240	70	75	87	145	145	189	293							
	D	3,344	1,960	635	187	46	46	62	102	98	84	154							
Bassein	C	3												1		1			
	D	1												1		2			
Bombay	C	7	4	6	3				1						1				
	D	5	8	5	2				1						1				
Plague-infected rats		86	108	81	26	13	14	10	15	10	8	9	9	12	9	11	15		
Madras Presidency	C	157	44	38	39	10	1	10	26	35	21	7							
	D	87	19	11	22	4	1	9	17	13	9	1							
Rangoon	C	3	5	4	1				2	2	1			2	1	5	2		
	D	3	4	3	1				2	1	1			2	1	4	2		
Plague-infected rats		3	4	5	1	2	2		2	4		2	1	2		2	4		
India (Portuguese)	D			7								P							
Indo-China (see also table below):																			
Pnompenh	C		3	3	6		1	1			2		2	2	1				
	D	13	4	1	7		1	1											
Saigon and Cholon	C	1		1	2							1			1				
	D	1			1														
Iraq: Baghdad	C	7	6	37	28	10	4	2	2	4	3	2							1
	D	4	2	18	15	5		1	1		2	1							1
Japan: Osaka (vicinity of)—Plague-infected rats		4	3																
Kwang-Chow-Wan	C	7	37	34	31				4			1							
Madagascar (see also table below): Tamatave	C		1		1					2						P			
Morocco	C	55	121	71	3				1		8	7							
	D	18	45	34	4						1	1							

Nigeria: Lagos	C	13		2	5	1				2	1	3	1		1				
	D	5		2	5	1				2	1	3	1						
Plague-infected rats		25	7	17	11	2	3	6	7			4	4						
Senegal (see table below).																			
Siam	C	16	5	5						2	3		2						
	D	15	6	4						1	3		2						
Bangkok	C	5		5						1	2								
	D	4		4							2								
Nagara Pathom	C	1	10																
	D	1	9																
Nagara Rajsima	C	1	1							1			1		1				
	D	1	1							1			1		1				
Syria: Beirut	C				12		2	4	3		1		1	3	2				
Tripolitania	C											2							
Tunisia:																			
Sfax district	C		1	21	9			6			1								
Tunis	C	1	4	1	1		1	4											
	D				1		1												
Union of Socialist Soviet Republics:																			
Saisk Region	C				2			3	2	4		3							
	D				1			2	2	3		2							
Stavropol Region	C				1														
	D				1														
Union of South Africa:																			
Cape Province	C	1		P	1			1					1						
	D	1						1				1							
Orange Free State	C	5	1																
	D	3																	
Transvaal	C	1																	
	D	1																	

Place		March, 1930	April, 1930	May, 1930	June, 1930	July, 1930	August, 1930
British East Africa (see also table above):							
Kenya	C	85	16	171	107	97	27
Ecuador: Guayaquil	C	2	0	0	0		
	D	0	0	0	0		
Plague-infected rats		2	0	0	0		
Greece (see also table above)	C		1		1		
Indo-China (see also table above)	C	27	4		11	1	2
Madagascar (see also table above):							
Ambositra Province	C	25	14	1			
	D	20	12	1			
Antisirabe Province	C	38	46	19	3	8	
	D	36	45	19	3	8	
Itasy Province	C	4					
	D	4					
Miarinarivo Province	C	14	1	5	1	1	
	D	14	1	5	1	1	

Place		March, 1930	April, 1930	May, 1930	June, 1930	July, 1930	August, 1930
Madagascar (see also table above)—Con.							
Moramanga Province	C	5	3	1	3		
	D	5	3		3		
Tananarive Province	C	52	39	15	16	11	
	D	52	38	14	16	11	
Senegal:							
Baol [1]	C	18	24	13	2	62	79
	D	8	12	11	2	48	20
Dakar [1]	C		2	52	53	140	108
	D		2	42	117	122	90
Louga [1]	C		33	54	60	138	75
	D		10	27	21	103	33
Thies [1]	C	3	12	21	52	54	34
	D	2	9	8	35	30	20
Tivaouane [1]	C	11	71	135	43	119	110
	D	8	38	69	28	70	54

[1] Incomplete reports.

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

SMALLPOX

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27
Algeria:																		
Algiers	C	5	1	3			1				1		2					
Constantine	C				1			1										
Oran	C	1																
Arabia: Aden	C	3	1			1												
Bolivia: La Paz.[1]																		
British East Africa (see also table below):																		
Tanganyika	C	103	57	409	1,610	64	4		100	26	51	44	121					
	D	7	14	70	301	13	2		27	4		3	30					
British South Africa:																		
Northern Rhodesia	C	9	1	59														
	D		2	9														
Southern Rhodesia	C		66	155	79	1	12	18										
	D		1	13														
Canada:																		
Alberta	C	10	4				2	1	2	1							1	
Edmonton	C	4	3															
British Columbia—Vancouver	C	20	17	4	2	2	2	1	1	1	1		2			1	1	1
Manitoba	C	4	4	10	4									1				
Ontario	C	100	77	82	47	3	5	6	10	8	3	5	4	2	2	6		1
North Bay	C			1														
Ottawa	C	19	21	25	15	1	1	4	7	5	1	1		2	1	2		
Toronto	C			4	4		1									1		
Quebec	C				4	3					2	2	1					
Montreal	C									7								
Saskatchewan	C	47	41	39	22		2		3				8			1		
Regina	C			4														
Ceylon: Angoda, Western Province	C		6															
	D		2															
China:																		
Canton	C	6	3															
	D	2	3															
Chungking	C		P	P	1	P	P	P	P	P		P	P	P				
Foochow	C		P	P	P		P		P		P		P					
Hong Kong	C	38	18	12	4		2											
	D	25	23	9	3		1											

Manchuria—																		
Harbin	C		1	20	4													
Kwantung—Dairen	C		2	8	16	8												
	D			4	1													
Nanking	C	P	P	P	P		P	P	P	P	P	P	P	P				
Shanghai—																		
Foreigners only	C	2	3	2	5	1	1	1	1	3						18		
Including natives	D	10	10	5	3											2		
Swatow	D	6	3	6	4	1				1		2	1	1	1			
Tientsin	C	2	2	1														
Chosen (see table below).																		
Colombia:																		
Barranquilla	C	2	1							2								
Buenaventura	O		15	4	6	4	3	2	1		1	1						
Costa Rica:																		
Port Limon	C		6	2	2													
San Jose [2]	C	10	7	2														
Curacao (alastrim)	C	14	2	2	1	2						1						
Dutch East Indies:																		
Borneo	C	185	90	16	12		2											
	D	12	31	1	1													
Java—																		
Batavia and West Java	C	78	64	12	13	3	1	3	1	1	4	5	2			2	1	
	D	6	11	7	3	2	1	1	1	1	1	1	2			1		
East Java and Madura	C		4					1										
Sanggi Islands	C	5	160	26				11		36								
	D	1	24	5				3		2								
Sumatra	C	48																
	D	5																
Egypt: Port Said	O		1	1			2		1									
Great Britain:																		
England and Wales	C	1,700	1,427	1,417	926	156	138	126	109	70	86	95	93	99	106	75	61	
Ashton under Lyne	C	15	18	17	7		4	2	2					1				
Bradford	C		3	2		1												
Cardiff	C	2	3			2												
Leeds	C	16	2			1												
London	C	710	602	637	498	84	52	62	48	36	50	41	51	43	44	36	41	
London and Great Towns	C	1,239	1,066	1,078	753	127	96	102	83	61	73	71	80	79	84	51	54	
	D	3	3															
Sheffield	C	8	2															
Stoke-on-Trent	C	122	85	62	32	3	2	1	3	1	1					1	1	
Scotland	C				4		1								5			
	D						1											
Honduras: Naco	C															5		
India	C	39,329	34,843	22,835	12,962	2,258	2,006	1,853	1,513	11,313	1,282	1,236						
	D	9,109	6,983	5,451	3,531	733	580	577	458	368	327	290						
Bombay	C	718	430	218	113	17	15	11	7	6	2		2		1	1		
	D	431	270	161	79	13	9	9	2	5	2		2		1	2		
Calcutta	C	361	495	302	161	27	19	13	14	4		12	9	4	5	3	2	
	D	305	403	258	146	17	17	12	10	4		8	9	3	4	1	1	

[1] From Jan. 1 to May 31, 1930, 44 deaths from smallpox were reported in La Paz, Bolivia.
[2] 5 cases of smallpox were reported Apr. 14, 1930, in Costa Rica, outside of city of San Jose.

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

SMALLPOX—Continued

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27
India—Continued.																		
Cochin	C	291	183	37	16			4	2	2	4	4	4	1	1		1	
	D	35	13	8	4									1				
Karachi	C	33	30	14	7				1	2		2				1	3	
	D	47	17	4	3					2		1					2	
Madras	C	173	133	72	35	6	10	17	16	19		9	10	9	11	6	8	
	D	36	27	15	10	4	1	3	4	3		5	3	1	1	1	3	
Moulmein	C	146	43	89	21	4	10	3	1				2	1				
	D	41	7	21	7	3	4	1	2				1			1		
Negapatam	C			1	2			1		2		2		1	2		6	
Rangoon	C	10	4	6	9	2	1	1		1	1	1						
	D	5		1	2													
Tuticorin	C			5	1													
Vizagapatam	C	69	6	5	1													
	D	18	2	3														
India (French):																		
Chandernagor	C	6	10	24	19	3	1	4										
	D	2	2	7	3	1	1	1								1		
Karikal	C	24	19	12	11	1		1			8		1		5			
	D	7	8	3	8	1		1			8		1		2			
Pondicherry Province	C	21	24	40	23	4	9	8	5	7	8		7		7	1		11
	D	13	20	36	23	3	9	8	5	7	8		7		7	1		11
India (Portuguese)	C	38	44	47	28	1				1	6	1	1					
	D	2	8	8	10				1	1			1					
Indo-China (see also table below):																		
Pnompenh	C			3				1										
	D		2		1			1				1						
Saigon and Cholon	C	3	5	3	1						1							
	D	1	5	2						1	1	1		1				
Iraq:																		
Baghdad	C		8	1	1	1	1	1				1						
	D		1	1				1				1						
Basra	C		1															
Mossoul Liwa	C		22	21	4	47	20											63
	D		3	3	1	19	1											27

Ivory Coast (see table below).																		
Jamaica (alastrim)	C			2														
Japan: Tokyo	C		1															
	D		1															
Macao	D	3	2	1														
Mexico (see also table below):																		
Jalisco (State) Guadalajara	D	22	20	7	15	3	1				1							
Juarez	C	2			6						2							
	D				1						1							1
Mexico City and surrounding territory	C	106	99	80	77	10	12	4	11	2	3	3		7				
	D	31	47	32	17	2	3	1	2	1	3	2		1				
Progreso	C			1														
	D			1	1													
Morocco (see table below).																		
Nigeria: Lagos	C	1	1		1													
Poland	C		7	3	2													
Portugal:																		
Lisbon	C	7	8	8	10		7		6		6	19	25	7	8		12	
Oporto	C												1					
Rumania	C	2																
Siam	C		8	2		2		4										
	D		4					3										
Somaliland, British: Boales	C	2																
	D	6																
Spain	C				12		1	1	3	2					4	1		
Straits Settlements	C	5	5	11	9	1	2	7	1	1	1	1	5	1	2	2	1	
	D	1	2	3	2	1	1	1				1	1		1			
Sudan (Anglo-Egyptian)	C	60	42	19	67	8	1		3				42	102	5	20	1	25
	D	5	4	2	3		1						3	50	2			2
Sudan (French) (see table below).																		
Switzerland: Berne Canton	C								1	1	1							
Syria (see table below).																		
Taiwan: Taihoku (see table below).																		
Tunisia: Tunis	C	3	4	2	2				1									
Turkey (see table below).																		
Union of South Africa:																		
Cape Province	C	P	P	P					P									
Orange Free State	C	P						P	P	P								
Transvaal	C	P									P	P	P					
Upper Volta	C	2	3	1	13													
Zanzibar	C	13																
On vessel:																		
S. S. Elysia, at Port Sudan, from Bombay	C		1															
S. S. Naldera, at Port Said	C			1														
S. S. Manoa, from Honolulu to San Francisco	C					1												

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

SMALLPOX—Continued

[C indicates cases; D, deaths; P, present]

Place		February, 1930	March, 1930	April, 1930	May, 1930	June, 1930			July, 1930			August, 1930			Sept. 1-10, 1930
						1-10	11-20	21-30	1-10	11-20	21-31	1-10	11-20	21-31	
Indo-China (see also table above)	C	434	26	261	305	80	133	----	----	----	238	59	34	----	54
Ivory Coast	C	----	7	----	----	----	----	----	----	----	----	----	----	----	----
Sudan (French)	C	213	609	521	274	76	----	----	----	34	----	----	----	39	----
	D	11	49	36	32	18	----	----	----	----	----	----	----	3	----
Syria: Beirut	C	18	17	19	7	6	1	----	----	----	2	1	----	----	----
Taiwan: Taihoku	C	43	58	12	----	----	----	----	----	----	----	----	----	----	----

Place		February, 1930	March, 1930	April, 1930	May, 1930	June, 1930	July, 1930
British East Africa (see also table above):							
Kenya	C	12	175	174	171	142	186
Uganda	C	109	----	----	78	----	----
	D	99	----	----	69	----	----
Chosen	C	263	236	253	107	----	3
	D	71	53	53	35	----	----
Seishin	C	4	----	5	2	1	2
	D	1	----	1	1	----	----

Place		February, 1930	March, 1930	April, 1930	May, 1930	June, 1930	July, 1930
France	C	23	8	58	51	----	----
Mexico: Durango (see also table above)	D	6	5	4	4	3	3
Morocco	C	74	17	10	18	5	3
Turkey	C	114	----	3	16	----	----
	D	42	----	----	----	----	----

TYPHUS FEVER

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27
Algeria:																		
Algiers	C	6	8	15	3	----	1	2	3	----	----	----	2	----	3	----	----	----
Constantine Department	C	11	15	6	12	----	1	1	----	----	----	----	3	----	----	----	----	----
Oran	C	----	----	3	4	----	2	1	----	----	1	----	----	1	----	----	----	----
Arabia: Aden	C	1	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
	D	1	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Bolivia: La Paz.[1]																		
Brazil: Porto Alegre	C	----	----	1	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Bulgaria	C	9	15	6	16	----	4	5	1	----	1	----	----	----	2	----	----	----
	D	----	1	2	1	----	----	1	----	----	----	----	----	----	----	----	----	----
China:																		
Manchuria—Harbin (see also table below)	C	4	52	13	8	2	----	----	----	----	----	----	----	----	----	----	----	----
Shanghai	C	1	----	----	----	----	----	----	----	----	1	----	----	----	----	----	----	----
Chosen (see table below).																		
Czechoslovakia (see table below).																		
Egypt:																		
Alexandria	C	----	1	----	2	1	----	----	----	----	----	1	----	----	1	1	----	1
Beheira Province	C	2	2	49	45	----	5	1	9	----	----	----	----	----	----	----	1	----
	D	----	----	13	4	----	1	----	1	----	----	----	----	----	----	----	----	----
Cairo	C	----	----	----	----	----	----	----	----	2	----	----	5	----	----	1	----	----
Port Said	C	----	----	----	----	----	----	----	----	----	----	----	4	----	1	----	----	----
Great Britain: Scotland—																		
Dunfermline	C	----	----	----	----	----	1	----	----	----	----	----	----	----	----	----	----	----
Glasgow	C	----	----	1	----	----	----	----	----	----	----	----	----	----	----	----	----	----
	D	----	----	1	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Greece (see table below).																		
Iraq: Baghdad Liwa	C	2	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Ireland:																		
Irish Free State—																		
Galway County—Oughterard	C	----	----	----	----	----	----	----	2	----	----	----	----	----	----	----	----	----
Kerry County—Dingle	C	----	5	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----
Leitrim County—Mohill	C	----	----	----	9	----	----	----	1	----	----	----	----	----	----	----	----	----

[1] 12 deaths from typhus fever were reported in La Paz, Bolivia, from Jan. 1 to May 31, 1930.

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

TYPHUS FEVER—Continued

[C indicates cases; D, deaths; P, present]

Place		Mar. 9–Apr. 5, 1930	Apr. 6–May 3, 1930	May 4–31, 1930	June 1–28, 1930	Week ended— July, 1930 5	12	19	26	August, 1930 2	9	16	23	30	September, 1930 6	13	20	27
Ireland—Continued.																		
Irish Free State—Continued.																		
Mayo County—																		
Ballina	C			2	1		1											
Castlebar	C												1					
Swinford	C			14														
Westport	C							2		1								
Roscommon County—																		
Roscommon	C							1										
Strokestown	C							1										
Wicklow County—Shillelagh	C				4	1		1										
Latvia (see table below).																		
Lithuania (see table below).																		
Mexico: Mexico City, including municipalities in Federal District	C	4	4	6	9	2		2	1	2	2	2	3	2				
	D	1	4	3	1					1	1							
Morocco	C	38	15	11	15		4	8	4				8		2			
	D	7	3															
Palestine	C	6	1	2	2	3	1	1	1		1	1	1		1	1		
Poland	C	228	243	171	117		24		12	10	15	2	7		3			
	D	13	15	5	11		4			1		1	1		1			
Portugal:																		
Lisbon	C		4															
Oporto	C	2	1									1						
Rumania	C	185	186	227	58	11	10	3	4	5								
	D	12	11	35	5	5	2	1		2								
Spain: Valencia	C				3		3											
	D						1											
Tunisia	C			6	18			14	10	2	5		3	1	1			
Turkey (see table below).																		
Union of South Africa:																		
Cape Province	C	P	P	P	P	P	P	P	P	P	P	P	P					
Natal	C	P	P	P	P	P	P	P	1	P	P	P						
Orange Free State	C	P		P	P	P	P	P	P	P	P	P	P					
Transvaal	C		P					P		P	P	P						
Yugoslavia (see table below).																		

Place		February, 1930	March, 1930	April, 1930	May, 1930	June, 1930	July, 1930
China: Harbin (see also table above)	C		37	204	240		
Chosen: Seoul	C	17		3	43	2	
Czechoslovakia	C	2	42	29	12	1	
Greece: Athens	C	6	3	1	3	3	6
Latvia	C				3	3	3
Lithuania	C	70	62	73	27	16	3
	D	5	4	4			
Turkey	C	3	1	3	16	2	
Yugoslavia	C	33	46	22	16	6	
	D	5	2	4	1		

YELLOW FEVER

Brazil:	Cases
Mage, on the Leopoldina Railway, between Rio de Janeiro and Nictheroy, Apr. 22, 1930	2
Campos, Rio de Janeiro Province, May 23, 1930	1
Para, June 23, 1930	2

Gold Coast:	Cases
July 10, 1930	1
Albosso, Aug. 5, 1930 (deaths)	1
Liberia, Monrovia, June 3, 1930	1
Nigeria, Lagos, July 12, 1930 (probably laboratory infection)	1