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EDITORIAL

THE GENERAL PRACTITIONER IN TUBERCULOSIS CONTROL

It is well known but not widely appreciated that the private practitioner in his daily work is one of the most important, if not the most important, force in the control of disease. The millions of people who come to the offices of general practitioners throughout the Nation present at first hand all the variety of disease to which man is subject. In the course of a lifetime, the private practitioner is confronted with almost all known maladies, among which are a great number of unsuspected infectious diseases. It is the private physician's task not only to diagnose and to treat individual patients but also to protect the public health through the close supervision of those who endanger the physical well-being of the community.

Tuberculosis presents at once a challenge and an opportunity to the general practitioner. Thousands of persons who have tuberculosis go to private physicians for other illnesses, and no official agency ever sees them. Although the physician deals directly with the source material of tuberculosis, he often does not recognize the early stages of the disease, because he does not constantly search for tuberculosis with the tools at his command.

Too often it is assumed that the control of tuberculosis is solely the health department's domain of action. This is not true, nor can it ever be true, so long as men practice the ancient art of medicine.

This is the tenth of a series of special issues of PUBLIC HEALTH REPORTS devoted exclusively to tuberculosis control, which will appear the first week of each month. The series began with the Mar. 1, 1946 issue. The articles in these special issues are reprinted as extracts from the PUBLIC HEALTH REPORTS. Effective with the July 5 issue, these extracts may be purchased from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., for 10 cents a single copy. Subscriptions are obtainable at $1.00 per year; $1.25 foreign.

The family doctor in the city, the country doctor going about from farm to farm, the village doctor in his office over the drug store know the people, have their trust, and guide their physical destinies. The educational pamphlets of a hundred organizations cannot have the enduring effect nor the permeating persuasiveness of the doctor's personal word. Tuberculosis is so deeply a personal disease that news of its tragic onset or advance can be more calmly accepted when its source is the family doctor and not a stranger from a distant agency.

Participation by the private physician in the control of tuberculosis need be no trouble in terms of time or technique. There are many ways in which the private physician can contribute his talents as a professional man and his influence as a community leader in any integrated program of control. The routine use of the intracutaneous tuberculin test on every person who visits the physician's office should be the fundamental routine. Reactors to tuberculin should then have chest X-ray films made and interpreted by physicians with special training in chest diseases. The general practitioner can get expert help from sanatorium physicians, chest specialists, and radiologists in his area on all routine chest films. Regularity of such conferences with more highly specialized colleagues will provide many opportunities to develop skills in the interpretation of films. Local health departments and tuberculosis associations can make special consultants available for indigent patients.

When pulmonary abnormalities are discovered on X-ray films, careful clinical and laboratory studies are essential before final diagnosis can be made. General practitioners may find helpful counsel in the "Guide for the Disposition of Persons with Abnormal Pulmonary Findings on X-Ray Films," which is published in this issue. This Guide should be of particular assistance to rural physicians on whom is usually placed the entire burden of follow-up activities.

It has been estimated that nearly 4 percent of all persons who visit physicians' offices are coughing or expectorating. The alert physician will insist upon a sputum examination of all such patients. Such practice will be rewarded by the discovery of tubercle bacilli in 3 or 4 out of every 100 specimens examined. The country doctor will often be astonished to discover that a patient with slowly resolving pneumonia has an acid-fast reason for prolonged convalescence.

In less populous areas the general practitioner is required to carry on case finding and follow-up almost single-handed. He must give advice and encourage his patients and their families. Indeed, it is at this time that the practical philosophy of the private practitioner is of

great moment—at the height of that crisis which occurs upon the announcement of tuberculosis. It is at this time that the general practitioner can bring all his talents into play. He is aware of the whole person. He knows the patient's background, habits, aspirations, and desires. He does not think of his patient merely as a pair of lungs; he thinks of a man of spirit as well as of body who for a time has come, through tuberculosis, upon disaster.

Health agencies, private and governmental, have demonstrated the effectiveness of mass radiography as a case-finding procedure. Thousands of cases of tuberculosis, the discovery of which would have been delayed, have been brought to light by this new method. However, it is known that the overwhelming majority of these cases are being discovered in metropolitan areas. Because of distance, inaccessibility, or local reluctance, the rural areas of our country have not yet realized the benefits of organized case finding. It is here that the general practitioner can serve in eradicating tuberculosis.

Through the utilization of modern methods of case finding, the rural physician can extend the frontiers of medicine. Those physicians who have not had actual experience of these new techniques should be provided with training by the county medical society, the health department or the tuberculosis association. Postgraduate training and continuation study should also be provided, so that practitioners who are removed from centers of medical knowledge may take advantage of the latest information. By means of such training, the case finding of the general practitioner can be integrated with the case finding of official and private agencies. The private physician has a vital part to play in the campaign against tuberculosis, and the success of the whole movement may well be determined by the efforts and leadership of general practitioners.

HERMAN E. HILLEBOE,
Assistant Surgeon General, Associate Chief, Bureau of State Services.

GUIDE FOR DISPOSITION OF PERSONS WITH ABNORMAL PULMONARY FINDINGS ON X-RAY FILMS

By HERMAN E. HILLEBOE *Assistant Surgeon Gineral, Associate Chief, Bureau of State Services, United States Public Health Service,* and JOHANNES HOLM, *Chief, Tuberculosis Control Division, State Serum Institute of Copenhagen, Denmark, and Advisory Consultant, Tuberculosis Control Division, United States Public Health Service*

Millions of people in the United States have been examined by means of mass radiography since the early days of World War II. This new technique is rapidly achieving the long-sought goal of

obtaining X-ray examinations of the lungs of the majority of the adults in our country. This objective can easily be reached within 5 years if all our resources are mobilized and a national plan is executed with speed and efficiency.

Yet a word of caution must be given to prevent indiscriminate diagnoses of pulmonary tuberculosis on the basis of X-ray examination alone. The screening films of thousands of persons have shown lesions characteristic of early tuberculosis. Many of these persons have had no tuberculin tests performed, no sputum examined, and no history of symptoms taken to confirm or deny the suspicious film findings. Yet most of them have been labeled as tuberculous. This is scientifically unsound, and because such a practice tends to become commonplace, and because damage is done to people and to control programs, we should now take stock of our diagnostic criteria for the clinical determination of early tuberculosis.

Specialists in tuberculosis rightly insist that, before final diagnosis, every attempt be made to obtain sputum specimens, and that such specimens be submitted to meticulous examination; that is, by direct smear of actual sputum and, if this is negative, by culture or guinea pig inoculation. If sputum is not present, a stomach washing should be made and the contents examined by appropriate culture methods in laboratories that employ skilled bacteriologists. In cases of pleural effusion, the same procedure should be followed. This is possible only in an accredited laboratory which has been certified by some impartial central laboratory.

If, after such diligent search, no tubercle bacilli are found, the diagnosis should be limited to "suspected tuberculosis." This does not mean that many of the shadows found on the survey films are not the residue of a tuberculosis process that once was active. Nor does it mean that the person should not be followed for several years to watch for new evidences of disease activity. If changes occur on the X-ray film, frequent and careful observation is essential, and intensified search should be made for tubercle bacilli. Certainly such cases should be suspect until all doubt is resolved. Nevertheless, in order to be scientific in our practice of medicine and to avoid needless personal distress, we should refer to such persons as "suspects" until such time as tubercle bacilli can be demonstrated, and the diagnosis of tuberculosis proved. A firm stand of this sort should do much to clarify confused thinking in some mass radiography programs. To put it simply and candidly: Do not diagnose pulmonary tuberculosis on the basis of an original X-ray film alone.

One must study "suspected" cases by means of a careful history, including recent or present symptoms which are often caused by tuberculosis. For differential diagnosis it is essential to employ the

tuberculin test, using the intracutaneous method properly applied and interpreted by an experienced person. In the presence of a negative tuberculin test, other reasons than tuberculosis must be found for suspected shadows, even though their location or configuration strongly suggest disease. Indeed, even when a cavity is demonstrated on the film, a negative tuberculin test demands that some etiological factor other than tuberculosis be sought as the cause.

No person should be labeled with the diagnosis of pulmonary tuberculosis on the basis of incomplete evidence. "Suspected tuberculosis" on the X-ray film must be corroborated by a positive tuberculin test and by positive bacillary findings, before the unsuspecting person is told he has real pulmonary tuberculosis. Let treatment be delayed and judiciously deliberated until all the facts are in and all the evidence is evaluated. If such a practice is universally followed, physicians will gain considerably in accuracy and skill of diagnosis, and limited hospital resources will be conserved. Most important of all, the person suspected of having tuberculosis will be assured thorough study and scientific diagnosis, and will be treated for tuberculosis only if the disease is actually present. Medical judgments based on positive and complete evidence will give a final verdict that protects the individual and the public health.[1]

This Guide is presented for use, on a provisional basis, in case-finding and follow-up programs in tuberculosis control of the Tuberculosis Control Division. After a few years' experience we will be prepared to offer specific recommendations to the medical profession. It is based upon recent extensive experience in the United States and Denmark and presents both clinical and public health aspects of the subject.

Before case-finding programs are started, clinic and laboratory facilities must be provided in order to obtain information necessary for the diagnosis and disposition of cases. Carefully controlled studies have shown that the ordinary laboratory is not prepared to do diagnostic culture work with tubercle bacilli. Great care must be taken in the choice of a laboratory to be used for such work and in evaluating the results from different laboratories. This Guide requires the services of a laboratory of the highest quality.

The examinations recommended in the Guide need to be carried out only on persons with abnormal pulmonary findings on X-ray films; that is, those who have definite or suspicious parenchymal infiltrations demonstrated on the screening film and confirmed on the 14″ x 17″ celluloid film.

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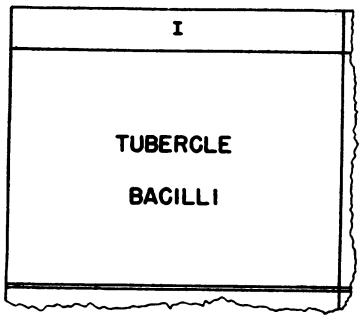
[1] This introductory passage includes excerpts from the editorial "What is Early Tuberculosis?" PUBLIC HEALTH REPORTS, vol. 61, No. 36 (September 1946).

INSTRUCTIONS

Accurate findings from four distinct examinations must be obtained and evaluated before accurate diagnosis and proper disposition can be made.



I. EXAMINATION FOR TUBERCLE BACILLI

A thorough examination for tubercle bacilli is so important that it commands first place in this guide. If the person is expectorating, the sputum should be examined by direct smear first. If the sputum is negative by direct smear, a culture should be made from the same specimen at once. For the person who really expectorates material from the lungs, no gastric lavage is necessary. For those persons who do not expectorate, a gastric lavage is required. This must be performed by an experienced person and the contents examined by culturing in a laboratory qualified to do this special culture work.

The terms in **Group I, Tubercle Bacilli,** are used as follows:

NO SPUTUM—means no expectoration.

SPUTUM SMEAR—Prepare slides from sputum. Stain slides by Ziehl-Neelsen stain.

- **NEGATIVE**—means no tubercle bacilli demonstrated.
- **POSITIVE**—means the demonstration of several acid-fast bacilli.

SPUTUM CULTURE—Use a culture medium of recognized efficiency.

- **NEGATIVE**—means no colonies of tubercle bacilli demonstrated. If no growth appears, the culture must not be labeled as negative until it is observed a minimum of 6 weeks after inoculation.
- **POSITIVE**—means the demonstration of typical colonies of tubercule bacilli. These colonies will usually appear 2 to 4 weeks after inoculation. Microscopy of smears from the colony must show typical tubercle bacilli.

GASTRIC CULTURE—The stomach of the fasting person must be thoroughly washed out with 200 to 300 cc. of sterile water in order to obtain a genuine sample. The culture is then made from the material in the same manner as described for sputum.

- **NEGATIVE**—means no colonies of tubercle bacilli are demonstrated by culture.
- **POSITIVE**—means that typical colonies of tubercle bacilli are demonstrated by culture from the stomach washings.

GUIDE FOR DISPOSITION OF PERSONS WITH ABNORMAL PULMONARY FINDINGS ON X-RAY FILMS.

U.S.P.H.S. – T.C.D. – 10-1-46

GROUP	I	II	III	IV	DISPOSITION OF CASE		
						CLINIC OR PRIVATE PHYSICIAN	
	TUBERCLE BACILLI	CAVITY ON X-RAY FILM	TUBERCULIN TEST (0.0001mg. PPD) INTRACUTANEOUS	PRINCIPAL SYMPTOMS: TEMPERATURE FATIGUE OR WEIGHT LOSS	SANATORIUM CARE	SPECIAL STUDY OR FOLLOW-UP FOR TUBERCULOSIS	GENERAL MEDICAL SUPERVISION
		+ PRESENT S SUSPECT O ABSENT	+ REACTOR O NON-REACTOR	+ ANY ONE OR ALL PRESENT. O ALL ABSENT.			
0	NO SPUTUM OR SPUTUM SMEAR NEGATIVE	O	O	O	NOTE: MAY OMIT SPUTUM OR GASTRIC CULTURE ONLY IF II, III, IV ALL ARE O.		✓
1	SPUTUM CULTURE NEGATIVE OR NO SPUTUM, BUT GASTRIC CULTURE NEGATIVE	O	O	+		✓	
2		+	O	+ OR O		✓	
3		S OR O	+	+ OR O		✓	
4		+	+	+ OR O		✓	
5	NO SPUTUM BUT GASTRIC CULTURE POSITIVE	S OR O	+	O		✓	
6		S OR O	+	O	✓	NOTE: SAME AS GROUP 5; BUT IF LIVING CONDITIONS POOR OR CHILDREN EXPOSED, SEND TO SANATORIUM.	
7		S OR O	+	+	✓		
8		+	+	+ OR O	✓		
9	POSITIVE SPUTUM (SMEAR OR CULTURE)	+, S, OR O	+	+ OR O	✓		

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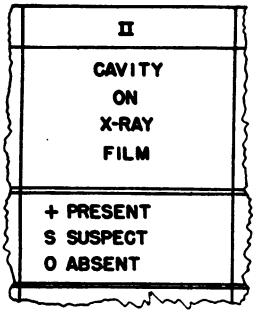
II. IDENTIFICATION OF CAVITY ON X-RAY FILM



Determination of cavity must be made on a 14" x 17" film and not on the basis of a screening film of any type.

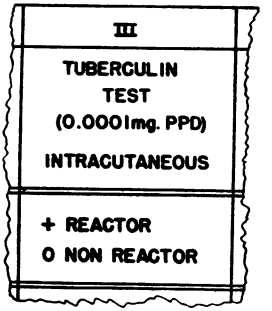
The inclusion of three classifications for identification of cavity, + **Present,** s **Suspect,** and **0 Absent**—instead of just positive and negative as formerly—is based on experience which has shown that error in identification of cavities is significantly reduced if the group **Suspect** is included. If a cavity is immediately recognized on a film, the findings are called + **Present;** if no cavity is seen, it is called **0 Absent.**

If there is a question of the presence or absence of a cavity, the film is classified as **Suspect.** Further X-ray study with special views is then indicated.

The terms in **Group II, Cavity on X-ray Film,** are used as follows:

+	**PRESENT**	means the presence of a cavity in one or both lungs.
s	**SUSPECT**	means any shadow suspect for cavity that cannot be classified.
0	**ABSENT**	means no definite or suspected evidence of a cavity in one or both lungs.

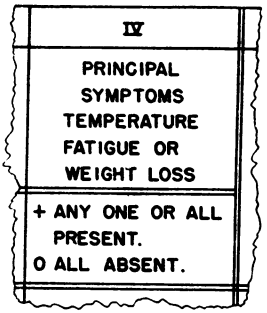
III. TUBERCULIN TEST



The single intracutaneous test may be used with a dosage of 0.0001 mg. of standardized PPD, or equivalent dosage of Old Tuberculin performed and read by a person experienced in this work.

The terms in **Group III, Tuberculin Test,** are used as follows:

+	**REACTOR**	means that reaction shows an induration (edema) with a diameter of at least 5 mm., 72 hours after injection.
0	**NON-REACTOR**	means no induration, or an area of induration less than 5 mm. Persons who show redness alone must be labeled **Non-Reactors.** Occasionally there may be doubt as to the interpretation of the reaction. If this is the case, the second test (using 0.002 mg. standardized PPD) may be employed. This should be called a "Reaction" only if there is typical induration of 10 mm. or more.



IV. PRINCIPAL SYMPTOMS

Tuberculous persons may exhibit a variable number of pulmonary and nonpulmonary symptoms and physical findings. However, clinical experience has shown that many symptoms that occur singly or in groups are of little help in determining the disposition of cases. These include such symptoms as pains in the chest, blood in the sputum, history of tuberculosis in the family, physical examination of the chest, and laboratory findings of increased sedimentation rate and blood changes.

The study of hundreds of cases of clinical tuberculosis indicates that the principal symptoms that must be considered are: (a) temperature elevation, (b) fatigue, and (c) loss of weight—within the last half year and not otherwise explainable.

Sputum, the principal symptom, is fully considered under **Group 1**.

The terms in **Group IV, Principal Symptoms,** are used as follows:

TEMPERATURE	means at least a one-degree rise above normal afternoon temperature for a period of 1 week, or a history of elevated temperature within the last 6 months—as determined by a physician or nurse.
FATIGUE	means weariness without apparent cause. If present, this should be considered a positive symptom.
WEIGHT LOSS	The amount of weight lost will depend upon the usual weight of the person. The significance of the amount of weight lost will be left to the discretion of the clinician.
+ ANY ONE OR ALL PRESENT	If any one or all of these three symptoms occur, + **Present** will be applicable.
0 ALL ABSENT	If there is no rise in temperature, no fatigue, no weight loss, **0 Absent** will be applicable.

ROUTINE PROCEDURE FOR DISPOSITION OF CASES

When there are abnormal pulmonary findings on the small screening film, a 14″ x 17″ celluloid film must be taken. There are two reasons for this. First, there may have been error in identification of the original small film. Second, the additional examination helps in the definitive identification of the lesion detected on the screening film. It will also aid in determining whether a cavity is **Present** or **Absent.**

Procedure of the first visit

A. Take a careful history with special attention to the principal symptoms, and make a permanent record of the findings.

B. Perform a tuberculin test as outlined and instruct the person to return in 72 hours for reading of the test.

C. Ask the person if he is expectorating. If so, give him a container and instruct him how to collect sputum. On the morning of his next visit he is to cough up the material produced on first arising. Instruct the patient to bring the container to the clinic, when he comes to have the tuberculin test read. If the person is not expectorating, plan for gastric lavage at the time of reading the tuberculin test. Explain the importance of the gastric lavage. For the gastric lavage, no food should be taken after 8 p. m. of the previous evening and no food or fluids on the morning before the clinic visit.

Second visit (72 hours after first visit)

A. Read and record the results of the tuberculin test, according to the procedure outlined.

B. If there is sputum, send specimen to the laboratory at once for examination, as outlined. The patient is then requested to return in 6 weeks unless sputum results require that he be notified sooner. If, at any time during the interval between visits, he develops symptoms, he should come immediately to the clinic.

C. If no sputum is obtained and the patient is a nonreactor to the tuberculin test, no gastric lavage is necessary. The present examination is complete. The patient is requested to return to the clinic in 6 weeks for another tuberculin test. If sensitivity to tuberculin has not developed by the time of the third examination, the case can be referred to general medical supervision as nontuberculous. Some other cause must be found for the shadows on the X-ray film.

D. If there is no sputum and the patient is a reactor to the tuberculin test, a gastric lavage should be done the next day and the gastric washings sent to the laboratory for culture. (Direct smear is not of value in the examination of gastric contents.) The patient is given an appointment after gastric lavage is performed and is requested to return in 6 weeks unless otherwise notified, or unless he develops symptoms.

Third visit

A. If the direct smear of the sputum is positive, the person should be notified at once to return for his third visit. Disposition to a sanatorium for immediate care is indicated. Great care must be exercised not to frighten the person unduly and to explain the diagnosis and need for treatment.

B. If, in 2 to 4 weeks, culture of the sputum or gastric lavage is reported positive, notify the patient to come in at that time for disposition. If, however, a positive report is received only after 4 to 6

weeks, the person can come in at the time already scheduled for his next visit.

C. If the culture of sputum or gastric lavage is negative, disposition of the patient can be made at his next regular appointment.

FOLLOW-UP AND DISPOSITION OF CASES IN NINE GROUPS

The Guide shows nine principal groups into which the results of a person's examinations may fall. There are corresponding recommendations for disposition of cases in each group. Procedures for public-health nurses and medical social workers in the follow-up program are being prepared for distribution.

Examples: **Group 0** indicates that the person has either no sputum or the sputum smear was negative; that no cavity is present on the X-ray film; that he is a nonreactor to the tuberculin test; and that he presents no temperature elevation, fatigue, or weight loss. This results in a recommendation to refer the case for general medical supervision because of lack of evidence of tuberculosis.

Group 9 indicates that the person has a positive sputum (gastric culture is not necessary); that X-ray findings are either "cavity present," "cavity suspect," or "cavity absent"; that he is a reactor to tuberculin; and that he may or may not have temperature elevation, fatigue, or weight loss. The recommended disposition is sanatorium care.

Explanation of groups (read across the chart)

Groups 7, 8, 9. Findings for each of these groups make it imperative that immediate sanatorium care be recommended. If no sanatorium beds are available, a bed in a general hospital should be found and the patient placed under the care and treatment of a chest specialist.

If no hospital bed can be found, it will be necessary to treat the patient temporarily at home. In the event that home care is necessary (but it should be avoided if at all possible), there is urgent need for expert and continuous medical care by a physician trained in chest diseases.

Special attention must be given to members of the household to protect them from contact with the patient or his infectious discharges. In addition, it is especially important for a public-health nurse to instruct the patient and family in contagious-disease technique and for such a nurse to visit at regular intervals to see that the instructions are followed and that contacts are examined regularly. If special social, economic, or emotional problems are discovered, refer the situation to a medical social worker, if one is available; if not, the public-health nurse can call in the proper social agency in the community.

Groups 5 and 6. Both groups represent persons who have a positive

gastric culture, whose X-ray findings are "cavity suspect" or "absent," who are reactors to tuberculin and who do not have any principal symptoms.

Group 5. Even with a positive gastric culture and the other indicated findings, the recommendation will be **Special Study or Follow-up for Tuberculosis,** providing the patient has satisfactory home conditions and no children are exposed.

As clinical observations are continued, changes in findings may occur which will place the patient either in **group 7, 8,** or **9,** whereupon sanatorium care will be recommended, or in **group 3** or **4,** whereupon follow-up alone is sufficient.

Group 6. The recommendation for persons with these findings who have inadequate living conditions or are exposing children to infection will be sanatorium care.

Group 4. If there is negative sputum or negative gastric lavage but a cavity is present, such an individual can best be studied in a tuberculosis clinic until final diagnosis is made. Such cases, in spite of reacting to the tuberculin test, can be classed only as **Suspect** with positive tuberculin tests, since tubercle bacilli cannot be demonstrated.

Group 3. This particularly is the type of case that must be followed in a clinic because it might be an early case of tuberculosis requiring further study before final disposition. In order to make final disposition, there must be interval examination to determine the significance of any findings. A person who is in **group 3** should be notified to return in 3 months for interval examination or whenever he might become ill. At this next 3-month's visit, certain examinations must be repeated:

(a) Repeat the search for tubercle bacilli in the usual manner. Repeat the X-ray examination in order to determine whether there are changes in X-ray findings. If there are tubercle bacilli or a cavity is present, the case can be reclassified.

(b) If tubercle bacilli and cavity are absent the patient can be told to return in 6 months.

(c) At the next clinic visit in 6 months, repeat the same procedure (see (a)) and, if tubercle bacilli and cavity are still absent, 1 year may elapse before another reexamination.

(d) When the patient returns 1 year after the last examination (c), repeat the procedure described in (c). If tubercle bacilli and cavity are still absent, the case need no longer be followed.

Groups 1 and 2. Such cases require special study by an expert in chest disease and are rarely tuberculous. Have patient return in 6 weeks for another tuberculin test. If the second test is also negative, the case should be studied further for differential diagnosis of non-tuberculous chest disease.

Group 0. This includes persons with abnormal pulmonary X-ray findings only. Tuberculosis can be ruled out because principal symptoms are absent, no tubercle bacilli have been demonstrated, tuberculin testing shows no reaction, and X-ray findings reveal no cavity. To determine the origin of the abnormal X-ray shadows, refer these patients to the clinic or private physician for general medical supervision, for differential diagnosis, and for follow-up.

FOLLOW-UP PROCEDURES FOR PERSONS IN WHOM TUBERCLE BACILLI HAVE BEEN DEMONSTRATED IN THE GASTRIC LAVAGE

Persons in whom tubercle bacilli have been demonstrated in the gastric lavage should be followed with at least one thorough examination for tubercle bacilli each year (even if the lesions show retrogression and there are no symptoms), until three negative gastric-lavage cultures have been taken a year apart. If at any time during these annual examinations the gastric lavage culture becomes positive, the program of 3-month, 6-month, and 1-year examinations must again be initiated with proper disposition at each visit.

CONTACTS

Examine all household, occupational, and other intimate contacts of persons whose findings fall into **groups 5, 6, 7, 8,** and **9.** Consider disposition of contacts as follows:

Take a chest X-ray film and give a tuberculin test (0.0001 mg. PPD) to all contacts at first visit.

A. Contacts with abnormal pulmonary findings

Follow procedure outlined above.

*B. Contacts who show **no** abnormal X-ray findings and who are **nonreactors** to tuberculin*

These contacts to positive sputum cases must return for subsequent tuberculin testing until a minimum of 1 year after the last exposure to an infectious case. Such follow-up testing should be done every 3 months during exposure and two times thereafter at intervals of 6 months after contact is broken. If the nonreactor becomes a reactor, follow closely by X-ray examination every 6 months for a minimum of 2 years and do everything possible to reduce or cut off exposure. These are the contacts most likely to develop genuine tuberculosis.

*C. Contacts who show **no** abnormal X-ray findings but who are **reactors** to tuberculin*

These contacts to positive sputum cases must have an X-ray examination after 6 months; then, if negative, one examination each year until a minimum of 1 year has elapsed since the last exposure to

an infectious case. More frequent examinations may be indicated if exposure is intense and living conditions poor.

It is unwise to send any person to a sanatorium who is merely suspected of having pulmonary tuberculosis. Sanatorium care should be recommended only after demonstration of a positive direct smear or a positive culture of the sputum or gastric lavage. Even if the patient has symptoms, or a recently elevated temperature, he can be observed in the tuberculosis clinic or in a local hospital. Not until tubercle bacilli are demonstrated should a patient be sent to the sanatorium for treatment. The patient is thus spared the psychological trauma and the personal risk of being hospitalized for a disease he does not have. Furthermore, a considerable number of scarce sanatorium beds will be reserved for infectious cases that are amenable to remedial care.

It is hoped that this Guide will help tuberculosis workers improve their diagnosis and disposition of cases, and at the same time provide the most effective utilization of the energies of private physicians, limited tuberculosis clinic services, and insufficient sanatorium beds.

VARIATION WITH AGE IN THE FREQUENCY OF TUBERCULOUS PULMONARY CALCIFICATION [1]

By ROBERT H. HIGH, *Assistant Surgeon*, and HENRY B. ZWERLING, *Surgeon (R)*, *Field Studies Section, Tuberculosis Control Division*

Calcification in the pulmonary parenchyma, the tracheobronchial lymph nodes, or in both, frequently follows tuberculous infection in children. Adults with tuberculosis, on the other hand, develop calcification much less commonly. Indeed, in adults the usual mode of healing is by fibrosis. These observations, which are often made, suggest strongly that the frequency of tuberculous pulmonary calcification is a variable of age.

It would seem pertinent, therefore, to study thoracic calcification in children of various ages who have developed tuberculosis. There is rarely an opportunity to watch the progress of a sizable group of children, beginning when they are tuberculin-negative and continuing after development of the disease. The results of such an investigation are presented in detail below.

MATERIAL AND METHODS

In 1936 the Health Division of the Office of Indian Affairs and the Henry Phipps Institute of the University of Pennsylvania began a

[1] From the Health Division, Office of Indian Affairs, Department of the Interior, Washington, D. C.; the Henry Phipps Institute, University of Pennsylvania, Philadelphia; and the Field Studies Section, Tuberculosis Control Division, U. S. Public Health Service.

study, directed by Dr. Aronson, to test the effect of BCG vaccination in preventing the development of tuberculosis. Three thousand and seven tuberculin-negative Indian children, living on reservations in Arizona, North Dakota, South Dakota, Wyoming, and Alaska, were selected for the purpose. At the beginning of the study, their ages ranged from less than 1 to 20 years. Of the 3,007 cases, 1,550 were vaccinated with BCG (introducing an artificial effect and thus excluding them from the present report), and the remaining 1,457 served as "controls." At approximately yearly intervals, chest roentgenograms and tuberculin tests (PPD given intradermally in doses of 0.00002 mg. or 0.005 mg.) were made on all children under observation.

Detailed results of this study have been published (*1*, *2*, *3*). The most recent report is that of Aronson and Palmer (*4*), who have summarized the data obtained during the first 6 years of the program. In those years tuberculosis developed in 185 children of the control group, and 13 additional cases of tuberculosis were found among a small part of the group, which was studied 1 or 2 years longer. Pulmonary tuberculosis was diagnosed in 190 instances, and extrapulmonary or disseminated forms, in the remaining 8. Data on the latter were included in tabulating the material presented here, in order to learn the approximate frequency with which pulmonary calcification follows all forms of the disease. The present report is based on data collected in observing this group of 198 children.

As only limited laboratory services were available for the demonstration of tubercle bacilli, most of the cases were not proved by bacteriologic examination; and in a few instances, tuberculosis was diagnosed from clinical or post mortem data alone. For the most part, however, diagnosis was based on roentgenographic evidence and on the demonstration of sensitivity to the standard first dose of PPD (0.00002 mg.). It is realized that these criteria are not completely reliable: tuberculous parenchymal lesions, tracheobronchial lymphadenopathy, or both, as revealed by roentgenography, can be simulated by other pathologic conditions. But it is unlikely that many nontuberculous lesions would have occurred in association with the change of the tuberculin reaction from negative to positive.

The lesions observed in the roentgenograms were classified as follows: *primary complex*, when a parenchymal lesion was noted in the presence of ipsolateral, enlarged tracheobronchial lymph nodes; *lymphoglandular*, when enlarged lymph nodes were noted in the absence of a parenchymal lesion;[2] *minimal*, when parenchymal

[2] For purposes of tabulation, this and the preceding type were, in some instances, grouped together as *cases showing adenopathy*.

lesions were noted in the absence of enlarged lymph nodes;[3] *pleural effusion*, used as generally accepted; and *all other types*, used to include the remaining cases—miliary tuberculosis, extrapulmonary tuberculosis, and so forth.

Pulmonary calcification was described when the shadow was of such opacity, irregularity, and location as not to be confused with vascular markings or beginning calcification of the costal cartilages. Furthermore, it was described only when the diagnosis seemed unequivocal. Additional films confirmed the presence of calcification in most instances.

FINDINGS

It is well known that morbidity and mortality from tuberculosis are extremely high among American Indians. A measure of prevalence is afforded by the data collected on 185 cases of tuberculosis found during the first 6 years of observation on the 1,457 Indian children in the control group. The distribution of these cases is shown in table 1. It should be added that 28 deaths from tuberculosis occurred in this group during the 6-year period.

The rate for cases showing adenopathy declines markedly with advancing age, whereas the rate for those showing minimal lesions or pleural effusions increases progressively in the older age groups. For the age group 21 to 25, no satisfactory interpretations can be made, as the person-years of observation were less than 100. In figure 1 the rates are shown for those cases occurring between the ages of 1 to 20 years.

TABLE 1.—*Number of cases and attack rates per 1,000 person-years of observation by type of tuberculosis and by age at time of first positive roentgenogram*

Type of tuberculosis	Number of cases						Attack rates per 1,000 person-years					
	All ages	Age					All ages	Age				
		1–5	6–10	11–15	16–20	21–25		1–5	6–10	11–15	16–20	21–25
Adenopathy	122	33	52	34	2	1	16.0	28.2	19.0	12.3	2.3	14.3
Minimal	29	0	3	14	11	1	3.8	0	1.1	5.1	12.6	14.3
Effusion	24	2	5	10	7	0	3.2	1.7	1.8	3.6	8.0	0
All other types	10	1	3	3	3	0	1.3	.9	1.1	1.1	3.4	0
All types	185	36	63	61	23	2	24.3	30.7	23.0	22.0	26.4	28.6
Person-years of observation	7,617	1,171	2,734	2,770	872	70						

[3] The term *reinfection minimal*, however, cannot be applied here, in view of the time at which tuberculin conversion occurred. Of the lesions classified as "minimal," 59.4 percent were seen at the time of conversion to positive to the first dose, 0.00002 mg. PPD. In only 12.5 percent was sensitivity to the first dose of PPD demonstrated 2 years before a lesion appeared on the roentgenogram, and in no case did sensitivity precede the appearance of a lesion by 3 years.

At all ages under 15, adenopathy—either alone or, more commonly, associated with a parenchymal infiltration—was the lesion most frequently observed. In the age group 16 to 20, on the other hand, adenopathy is the least frequently observed lesion. The fact that all lesions of this type appeared in recent tuberculin converters suggests

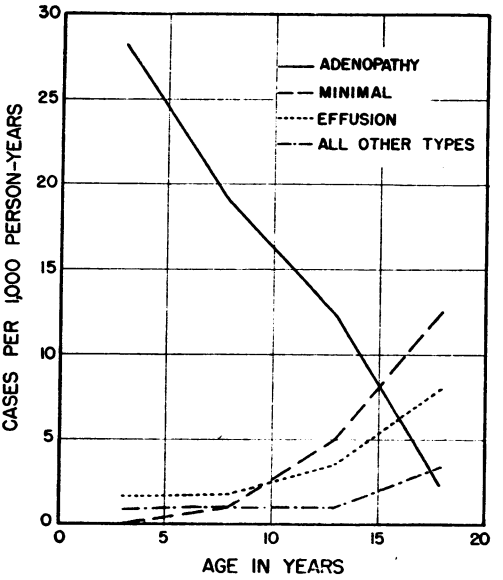


FIGURE 1.—Tuberculosis cases per 1,000 person-years, by type of tuberculosis and by age at time of first positive roentgenogram.

that the development of adenopathy may be directly related to age. It will be seen in table 1 that among the 36 cases of tuberculosis found in children 5 years of age or younger, there were 33, or 91 percent, with enlarged lymph nodes; while in those over 15, only 2 out of 23, or 9 percent, were cases showing adenopathy.

Turning to the observations on the total group of 198 cases, table 2 presents data by age and type of lesion, and shows the proportion of tuberculous lesions that ultimately calcified. A classification by sex was not included, as preliminary studies revealed no statistically significant difference between males and females in the development and calcification of lesions. It is immediately apparent that the proportion of lesions developing calcification decreased markedly with advancing age. This is true for lesions of every type. Among children less than 5 years of age, for example, 76.5 percent of all lesions ultimately calcified; and among those in the age group 15 to 19, calcification occurred in only 2.4 percent. It should be emphasized that between these groups, there is a steady decline in the percentage of children showing calcification. The difference is illustrated in figure 2.

In considering the entire group, one finds that the various types of tuberculous lesions have apparently calcified with different frequency. In the cases where adenopathy was noted, for instance, calcium developed almost three times as frequently as in those showing minimal lesions, and almost five times as frequently as in those showing pleural

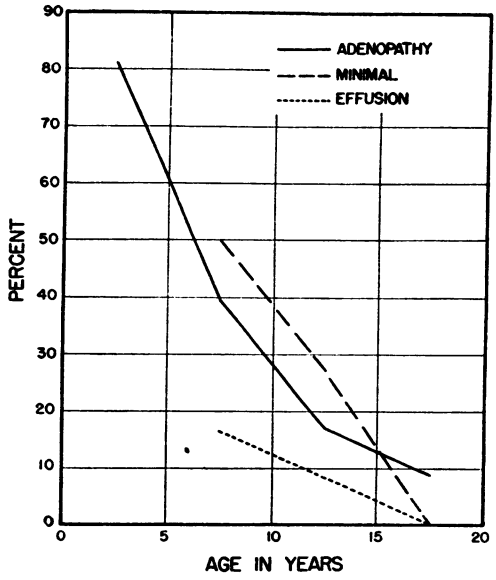


FIGURE 2.—Percentage of persons with tuberculosis who developed calcification, by type of tuberculosis and by age at time of first positive roentgenogram.

effusion. Where other types of disease were observed—that is, advanced or disseminated—no pulmonary calcification occurred.

The data presented in table 2 should be regarded as an incomplete measure of the actual frequency with which lesions calcified. It has been mentioned that the children were examined only at yearly intervals; it must be added that the disease developed at about the same rate in all cases throughout the study; and it may be seen from this that all cases were not followed for the same length of time. Adjustment was made for the variation in periods of observation, using a modified life-table technique. The usual procedure of assigning a half year's experience when a case is lost between examinations was precluded, of course, by the nature of the examination itself: a case was regarded as contributing experience only until last examined. Those cases that developed calcification were withdrawn as of the time at which calcification was first observed.

The interval between the first appearance of a lesion on the roentgenogram and the development of clearly demonstrable thoracic calcification is presented in table 3. It will be noted that readily dis-

cernible calcium deposits appeared, for the most part, during the second and third years after a lesion was seen. Very few lesions developed calcification by the end of the first year, but a moderate

TABLE 3.—*Development of intrathoracic calcification in persons with tuberculosis by yearly intervals from time of first positive roentgenogram*

Interval in years from time of first positive roentgenogram (years)	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Number without observed calcification at beginning of interval	Number without observed calcification withdrawn from observation during interval		Number under observation at end of specified interval	First evidence of calcification at specified interval		Cumulative percentage of calcification at the end of specified interval
		Total [1]	Deaths		Number	Percentage of (4)	
1	198	54	20	144	3	2.1	2.1
2	141	35	8	106	25	23.6	25.2
3	81	20	1	61	16	26.2	44.8
4	45	15	2	30	5	16.7	54.0
5	25	5	0	20	0	0	54.0
6	20	17	0	3	0	0	54.0
7	3	3	0	0	0	----------	54.0

[1] Including deaths.

number calcified in the fourth. No lesions calcified later than 4 years after the active lesion was first observed. Of those followed for 4 years or longer, 54 percent developed calcification.

When the lesions are analyzed by age distribution, using the life-table technique as in table 3, the age difference in rates of calcification, noted in table 2, is again revealed. This analysis is summarized in table 4 and in figure 3. It will be seen that the percentage of lesions with calcification is greater than shown in table 2, as a result of the correction for cases withdrawn from observation during the study. The frequency with which calcification was noted depended, of course, upon the length of the observation periods. However, the cases in each group were observed for comparable periods of time.

If the percentage of lesions that developed calcification in each age group is plotted, the resulting line is almost straight. It can be represented by the following equation, derived by the method of least squares:

$$Y=99.425-4.93X$$

X in the equation represents the age in years when the lesion is first observed, and Y the percentage of survivors expected to show calcification 4 years or more after such observation. For practical purposes, this equation can be expressed more simply as:

$$Y=100-5X$$

In other words, for every increase of 1 year in the age at which the lesion is first seen, there is a 5-percent less chance that calcification

TABLE 2.—*Number of persons with tuberculosis,[1] number and percentage developing calcification by type of tuberculosis and by age at time of first positive roentgenogram*

Type of tuberculosis	Number of persons with tuberculosis												Percentage with calcification					
	All persons						With calcification											
	All ages	Under 5	5–9	10–14	15–19	20 and over	All ages	Under 5	5–9	10–14	15–19	20 and over	All ages	Under 5	5–9	10–14	15–19	20 and over
Adenopathy	125	16	56	41	11	1	43	13	22	7	1	0	34.4	81.3	39.3	17.1	9.1	0
Minimal	32	----	2	11	17	2	4	----	1	3	0	0	12.5	----	50.0	27.3	0	0
Effusion	27	----	6	12	8	1	2	----	1	1	0	0	7.4	----	16.7	8.3	0	0
All other types	14	1	2	5	6	----	0	0	0	0	0	----	0	0	0	0	0	----
All types (total)	198	17	66	69	42	4	49	13	24	11	1	0	24.7	76.5	36.4	15.9	2.4	0

[1] This and the following tables are based on all the cases included in table 1 and on an additional 13 cases which were discovered during a longer period of observation. More conventional age groupings are used than in table 1.

TABLE 4.—*Development of intrathoracic calcification in persons with tuberculosis by yearly intervals from, and by age at time of, first positive roentgenogram*

Interval in years from time of first positive roentgenogram	Number under observation at end of specified interval						Number showing first evidence of calcification at specified interval						Percentage showing first evidence of calcification at specified interval						Cumulative percentage of calcification at the end of specified interval					
	All ages	Under 5	5–9	10–14	15–19	20 and over	All ages	Under 5	5–9	10–14	15–19	20 and over	All ages	Under 5	5–9	10–14	15–19	20 and over	All ages	Under 5	5–9	10–14	15–19	20 and over
1	144	16	54	45	26	3	3	0	1	2	0	0	2.1	0	1.9	4.4	0	0	2.1	0	1.9	4.4	0	0
2	106	15	43	31	15	2	25	5	14	6	0	0	23.6	33.3	32.6	19.4	0	0	25.2	33.3	33.9	23.4	0	0
3	61	10	24	18	8	1	16	6	7	2	1	0	26.2	60.0	29.2	11.1	12.5	------	44.8	73.3	53.2	31.9	12.5	0
4	30	4	10	10	6	0	5	2	2	1	0	0	16.7	50.0	20.0	10.0	0	------	54.0	86.7	62.6	38.7	12.5	------
5	20	2	6	6	6	0	0	0	0	0	0	0	0	0	0	0	0	------	54.0	86.7	62.6	38.7	12.5	------
6	3	1	0	0	2	0	0	0	0	0	0	0	0	0	------	------	------	------	54.0	86.7	62.6	38.7	12.5	------
7	0	0	0	0	0	0	0	0	0	0	0	0	------	------	------	------	------	------	54.0	86.7	62.6	38.7	12.5	------

will develop. It can be stated further, on the basis of this evidence, that persons who develop lesions before the age of 5 have seven chances in eight of developing calcification; that those aged 5 to 9

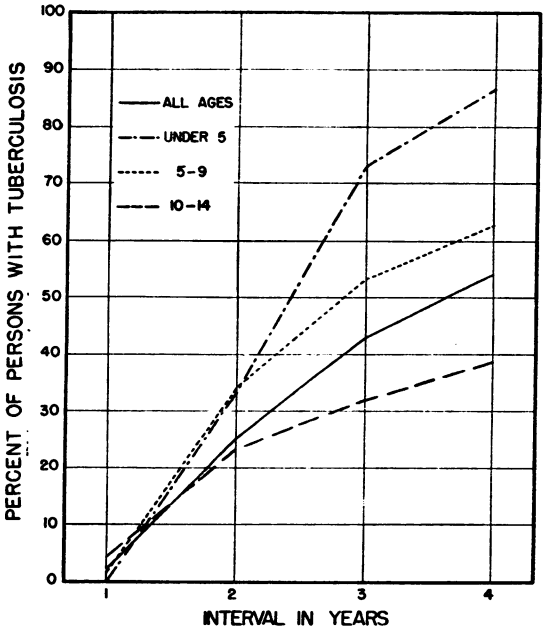


FIGURE 3.—Percentage (cumulative) of persons with tuberculosis showing calcification up to and including each yearly interval from time of first positive roentgenogram, by age at time of first positive roentgenogram.

have five chances in eight; those 10 to 14, three chances in eight; those 15 to 19, one chance in eight.

Data for the 49 cases in which calcium developed are presented in table 5. The rapidity with which a lesion calcifies apparently increases

TABLE 5.—*Percentage (cumulative) of persons with calcification following tuberculosis who showed calcification by the end of specified interval by interval in years from time of first positive roentgenogram to appearance of calcification and by age at time of first positive roentgenogram*

Age	Total persons with calcification following tuberculosis	Interval from time of first positive roentgenogram to appearance of calcification							
		1 year	2 years	3 years	4 years	1 year	2 years	3 years	4 years
		Number of persons				Cumulative percentage at the end of specified interval [1]			
Under 5	13	0	5	6	2	0	38.5	84.6	100.0
5-9	24	1	14	7	2	4.2	62.5	91.7	100.0
10-14	11	2	6	2	1	18.2	72.7	90.9	100.0
5-19	1	0	0	1	0	0	0	100.0	100.0
All ages	49	3	25	16	5	6.1	57.1	89.8	100.0

[1] Based on persons with calcification as shown in second column (total).

as the age at which it is observed advances. For example, in the age group under 5 years, no calcium was seen at the end of the first year, and by the end of the second, it was seen in only 38.5 percent. The lesions observed in the two following age groups, however, calcified more frequently in the first and second years. For all age groups,

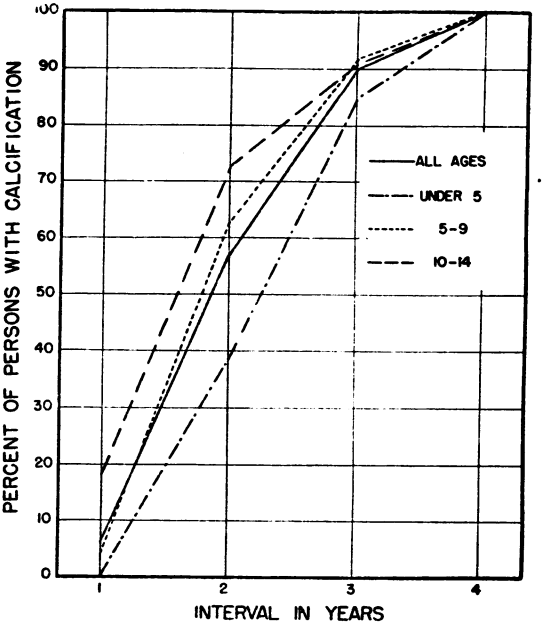


FIGURE 4.—Percentage (cumulative) of persons with calcification following tuberculosis who showed calcification by the end of specified interval, by interval in years from time of first positive roentgenogram to appearance of calcification, and by age at time of first positive roentgenogram.

the proportion that had calcified was the same at the end of the third year; and by the end of the fourth, all cases of calcification had appeared.

In order to determine whether there was a difference in the frequency with which parenchymal and hilar lesions calcified, a study of the individual lesions, as noted on the roentgenograms, was undertaken. Observation was confined to the 149 cases in which single or multiple lesions were clearly discernible. Roentgenograms were excluded when widespread, coalescing, or when otherwise obscure lesions were present. One might assume that a person with two or more individual lesions would be more likely to develop calcification than another person with only one lesion; and since two or more lesions were present in many instances, the assumption must be investigated. Results of the study are given in table 6.

No significant difference was observed in the calcification of hilar and parenchymal lesions within each age group; but significant differences did exist among the various age groups, as previously stated.

TABLE 6.—*Calcification of individual lesions noted on the roentgenogram by type of tuberculosis and age at time of first positive roentgenogram and by site of lesion*

Type of tuberculosis and age at time of first positive roentgenogram	Total number of cases	Total number of lesions observed in roentgenogram		Total number cases showing calcification	Calcification at site of observed lesion				No calcification at site of observed lesion				Calcification at site other than observed lesion	
					Hilum		Parenchyma		Hilum		Parenchyma			
		Hilum	Parenchyma		Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage	Hilum	Parenchyma
PRIMARY COMPLEX														
All ages	86	88	87	26	21	23.9	19	21.8	67	76.1	68	78.2	2	1
Under 5	9	9	9	7	6	66.7	5	55.6	3	33.3	4	44.4	1	0
5–9	37	38	37	15	12	31.6	11	29.7	26	68.4	26	70.3	0	1
10–14	34	35	35	4	3	8.6	3	8.6	32	91.4	32	91.4	1	0
15–19	5	5	5	0	0	0	0	0	5	100.0	5	100.0	0	0
20 and over	1	1	1	0	0	0	0	0	1	100.0	1	100.0	0	0
LYMPHOGLANDULAR														
All ages	31	33	5	16	14	42.4	2	40.0	19	57.6	3	60.0	1	4
Under 5	6	6	2	6	4	66.7	1	50.0	2	33.3	1	50.0	1	1
5–9	12	12	0	6	6	50.0	0	0	6	50.0	0	0	0	0
10–14	7	8	2	3	4	50.0	1	50.0	4	50.0	1	50.0	0	2
15–19	6	7	1	1	0	0	0	0	7	100.0	1	100.0	0	1
20 and over	0													
MINIMAL														
All ages	32	0	35	4	0	0	5	14.3	0	0	30	85.7	1	0
Under 5	0													
5–9	2	0	2	1	0	0	1	50.0	0	0	1	50.0	1	0
10–14	11	0	13	3	0	0	4	30.8	0	0	9	69.2	0	0
15–19	17	0	18	0	0	0	0	0	0	0	18	100.0	0	0
20 and over	2	0	2	0	0	0	0	0	0	0	2	100.0	0	0

The cases showing only enlargement of tracheobronchial lymph nodes tended to calcify with somewhat greater frequency than the others. This seems a little unusual, particularly since the cases classified as *primary complex* necessarily showed such hilar enlargement. The reason for the tendency is obscure; but it may be suggested that, in general, a greater degree of adenopathy was responsible in cases of the former class. It is generally believed that large tuberculous lymph nodes are more liable to caseate than small ones, and that calcification frequently follows caseation. A review of the films revealed that the lymph nodes were larger in those cases where no parenchymal lesion was seen.

Among the 149 cases, pleural disease was concurrent with parenchymal or hilar disease in 34 instances. These are not presented in table 6, because no calcification of the pleura occurred.

MISCELLANEOUS OBSERVATIONS

The deposits of calcium assumed the pattern which is usually observed. The earliest deposits appeared as tiny granules, which gradually became larger. After about 2 years, the deposits tended to be grouped in a single homogeneous mass, irregular in contour, and somewhat smaller than that formed by the granules in the preceding stage. As time passed, additional films showed that this mass tended to lose its irregular outline and to appear as a homogeneous, round density, resembling a large blood vessel. In no instance did all calcium deposits disappear, but in a single case one parenchymal calcium deposit disappeared completely. Its position had been such that it was not obscured by osseous or mediastinal shadows, and its disappearance was confirmed by additional films.

As noted in other studies, more calcium was deposited on the right side than on the left. Of the 49 cases showing calcification, deposits occurred on the right side in 31 cases, on the left in 14, and on both sides in 4. Hence, the right side showed calcification in 35 of the 49 cases (71.4 percent), and the left in 18 (36.7 percent).

The distribution of calcium closely followed that of the initial lesions. The lesions by which the disease was classified occurred on the right side in 103 instances, on the left in 72, on both sides in 19, and on neither in 4. In summary, the initial lesions were noted on the right side in 61.6 percent of the cases, on the left in 46.0 percent.

It has been observed that cases with tuberculous lesions may develop calcification in areas where no antecedent lesion has been seen. There were 11 such instances in the present study. It must be mentioned, however, that in 7 of these cases, calcium also appeared at the site of the original lesion. In the remaining 4 cases, the original

lesion did not calcify. The calcification in 2 of these cases followed pleural effusions so large as to preclude the recognition of an associated parenchymal or hilar lesion. The third case was one in which pleural effusion and enlarged hilar lymph nodes were observed on one side, but in which calcium deposits were subsequently seen on the other. The fourth case presented enlarged lymph nodes, followed in 3 years by calcification on the same side, in the lung parenchyma.

In addition to the 198 cases of tuberculosis, there were 402 children who became sensitive to the first dose of tuberculin (0.00002 mg. PPD) during the period of observation. No evidence of tuberculosis was observed in this group, though more than 6 roentgenograms, as a rule, were taken of each person during the program. Nine, or 2.2 percent, of the 402 children developed calcification. Demonstrable tuberculosis, on the other hand, was followed by calcification in 24.7 percent of the cases—more than 11 times as frequently as among the converters. This suggests that most tuberculous calcifications are preceded by lesions demonstrable by roentgenography.

DISCUSSION

The relation of age to the development of tuberculous pulmonary calcification has previously received insufficient attention. Some reports that have appeared, however, include relevant data, and may be cited in support of the material presented here. Brailey (*5*) studied a group of 158 positive tuberculin reactors who were 2 years of age or younger, and noted that calcification appeared in 66.1 percent within 4 years of the first examination. From Myers' report on 20 years of experience at the Lymanhurst Health Clinic (*6*), it may be shown that among 767 children who became positive tuberculin reactors while under observation, successively fewer developed calcification in the older age groups. Gass et al. (*7*) studied a group of school children in Williamson County, Tennessee, in 1937, 1939, and 1941. Among those who acquired pulmonary calcification between the surveys, successively fewer of the older-age groups were affected; and no child of 15 or over developed calcification.

While none of these studies were conducted in the same way as the present one, they show that pulmonary calcification develops most frequently in young children. Holm (*8*), in discussing studies made in Denmark, contributes material similar to that given in the present report.

SUMMARY

A study of thoracic calcification following tuberculous infection was made on 1,457 American Indian children who served as a control group in testing the effectiveness of BCG vaccine. During the period

of observation, 198 cases of tuberculosis occurred, and calcification later developed in 49 of these. Deposits of calcium appeared 1 to 4 years after a lesion was noted on the roentgenogram, and usually during the second and third years after the lesion was first seen.

Very striking age differences were observed in the frequency with which calcification developed. In the youngest age group, calcification followed tuberculosis in 76.5 percent of the cases. The rate declined progressively in the older age groups, so that calcification only developed in 2.4 percent of the cases in whom the onset of the disease was between the ages of 15 and 19. Within a given age group, hilar and parenchymal lesions tended to calcify with about the same frequency.

No significant sex difference was noted.

More calcium was deposited on the right side of the chest than on the left—71.4 percent in the former and 36.7 percent in the latter. This distribution follows rather closely the sites of the original lesions—61.6 percent on the right side and 46.0 percent on the left.

Tuberculous lesions were followed by calcification in 24.7 percent of the cases; and tuberculin conversion with no demonstrable lesion, in 2.2 percent.

CONCLUSION

The frequency with which calcification follows pulmonary tuberculous lesions varies directly with the age in life at which the lesions are acquired.

ACKNOWLEDGMENT

The authors wish to thank Dr. Joseph D. Aronson and the Office of Indian Affairs, Department of the Interior, for permission to use the data upon which the report is based, and to express their deep appreciation for the valuable assistance of Mrs. Teresa Roache, Mrs. Sarah E. Yelton, and Dr. Carroll E. Palmer, of the Tuberculosis Control Division, United States Public Health Service.

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CALCIFICATIONS IN THE SPLEEN

OCCURRENCE IN HISTOPLASMIN AND TUBERCULIN REACTORS[1]

By Robert H. High, *Assistant Surgeon, United States Public Health Service*

Within the past 40 years, an extensive literature has developed concerning calcifications in the spleen. Excellent reviews have been presented by Bachman (*1*), Gray (*2*), Sweaney (*3*), and others; but it is unnecessary to review this past work for the purposes of the present brief report.

It is recognized that calcifications of the spleen can be produced by phleboliths, parasitic infections (chiefly echinococcus), abscesses, arteriosclerotic plaques, perisplenitis, infarcts, hemorrhage, and many other conditions. In the opinion of most authors, however, tuberculosis is by far the most frequent cause of such calcifications. Postmortem studies reported by Moorman (*4*) show that as many as 24.6 percent of spleens from tuberculous patients have calcifications. Other studies (*2*) have shown that as many as 57 percent of spleens removed at autopsy have calcifications, usually associated with pulmonary lesions consistent with a diagnosis of tuberculosis. Areas of calcification in the spleen have been described (*5*) as ranging from multiple nodules 1 to 5 mm. in diameter to large masses. It is usually stated that round nodules under 5 mm. in diameter are most frequently caused by tuberculosis or phleboliths.

Although many reports have appeared, most of them present but a few cases. From previous publications, there is no way of estimating the actual prevalence in a normal population. Hillyer (*6*) found 3 adults with definite splenic calcifications when he reviewed 350 films of the abdomen which were taken as diagnostic procedures for gastrointestinal or genitourinary complaints.

The present report deals with a group of calcifications in the spleen incidentally observed in a survey conducted to study the epidemiologic aspects of histoplasmin and tuberculin sensitivity (*7*).

[1] From the Tuberculosis Control Division.

FINDINGS

In 1945, the Tuberculosis Control Division of the United States Public Health Service, through the cooperation of the Board of Education, the City Health Department, and the Tuberculosis Society, of Kansas City, Mo., made histoplasmin and tuberculin skin tests and chest roentgenograms of over 17,000 persons. The subjects of this survey were 16,013 school children (13,522 white and 2,491 Negro) and 1,594 adults (1,458 white and 136 Negro). Round densities in the left upper quadrant of the abdomen were noted as incidental findings on 53 chest films. Many of these densities were rather indefinite, and although it was suspected that they represented calcifications in the spleen, no further investigation was possible.

It must be emphasized that this report is based upon the material used by Furcolow, High, and Allen (7) in the study of histoplasmin and tuberculin sensitivity. The roentgenograms were made in order to investigate the pulmonary area, and hence cannot be expected to give very satisfactory data on the prevalence of splenic calcification. Furthermore, so few cases of such calcification were observed that it was impossible to obtain reliable prevalence rates. The rates presented in this report are offered merely as preliminary estimates, in full appreciation of their possible divergence from those to be derived from subsequent findings.

In 15 children and 5 adults, the original chest-survey films were sufficiently definite to warrant a diagnosis of splenic calcification. Eight of the children were examined with additional films, including in each instance a Potter-Bucky diaphragm film of the splenic region. In all eight, the presence of splenic calcification was confirmed.

The ages of the 15 children in whom such findings were noted ranged from 7 to 17 years. Eleven children were white and four Negro. Seven instances were noted in males and eight in females. Thus, the frequency of splenic calcification in these school children was at least 0.9 per thousand.

In 14 instances, two or more round densities, none of which exceeded 5 mm. in diameter, were observed (fig. 1). In the remaining instance, several densities, 1 to 2 mm. round, were noted in addition to one large 18 x 31-mm. calcareous density (fig. 2). In three cases calcareous densities were also seen in the right upper quadrant of the abdomen. These, presumably, were intrahepatic. In 11 instances, calcification was also present in the thorax.

In 14 instances, intracutaneous tuberculin and histoplasmin tests were given, using 0.0001 mg. of PPD–S and 0.1 cc. of a 1 to 1,000 dilution of histoplasmin. These tests were considered positive if induration of 5 mm. or more was noted at the 48-hour reading. The results of these tests are presented in table 1, where the presence or

absence of thoracic calcification is also shown. It should be noted that 9 of the 14 cases (64.3 percent) did not react to tuberculin, whereas 5 (35.7 percent) did. Eleven (78.6 percent) reacted to histoplasmin, and three (21.4 percent) did not.

TABLE 1.—*Number of children and adults with splenic calcification by skin reaction to histoplasmin and tuberculin and by presence of thoracic calcification*

Skin reaction	Total	With negative chest		With thoracic calcification	
		Children	Adults	Children	Adults
H+ T−	9	1	1	7	0
H+ T+	7	0	1	3	3
H− T+	2	2	0	0	0
H− T−	1	1	0	0	0
Not tested	1	0	0	1	0
Total	20	4	2	11	3

Among the 1,594 adults studied, 5, ranging from 24 to 61 years of age, presented similar splenic calcification. Two white males, two white females, and one Negro male were affected. Their skin reactions to the tuberculin and histoplasmin tests, given and interpreted as above, are also noted in table 1. It is interesting that the rate of splenic calcification, 1.9 per thousand, is twice that for the children. This finding is in accordance with many previous reports noting a greater proportion of cases among adults.

Among the 19 cases tested, 9 (47.4 percent) reacted to tuberculin, whereas 16 (84.2 percent) reacted to histoplasmin. It would appear that tuberculosis is not the only, or even the most frequent, cause of splenic calcification in Kansas City, Mo.

It should be mentioned that 5 of the 20 splenic calcifications occurred in Negroes. The frequency of splenic calcification is twice as high in Negroes (1.9 per thousand) as in whites (0.8 per thousand). This observation suggests that there may be a racial difference in the frequency of splenic calcification. It should be repeated, however, that present data are inadequate to permit definite conclusions.

The prevalence of splenic calcification as stated above probably represents a minimal estimate of the occurrence in this population. Thirty-three suspicious cases were noted, in addition to the 20 described, but no further examinations could be undertaken. Moreover, it is obvious that the technique used for chest roentgenography is unsatisfactory for examination of the spleen. It is therefore highly probable that many additional instances of splenic calcification would have been observed had the splenic area been routinely examined with Potter-Bucky diaphragm films. It is very interesting, however, that the prevalence of such calcification is at least 0.9 per thousand in children and 1.9 per thousand in adults.

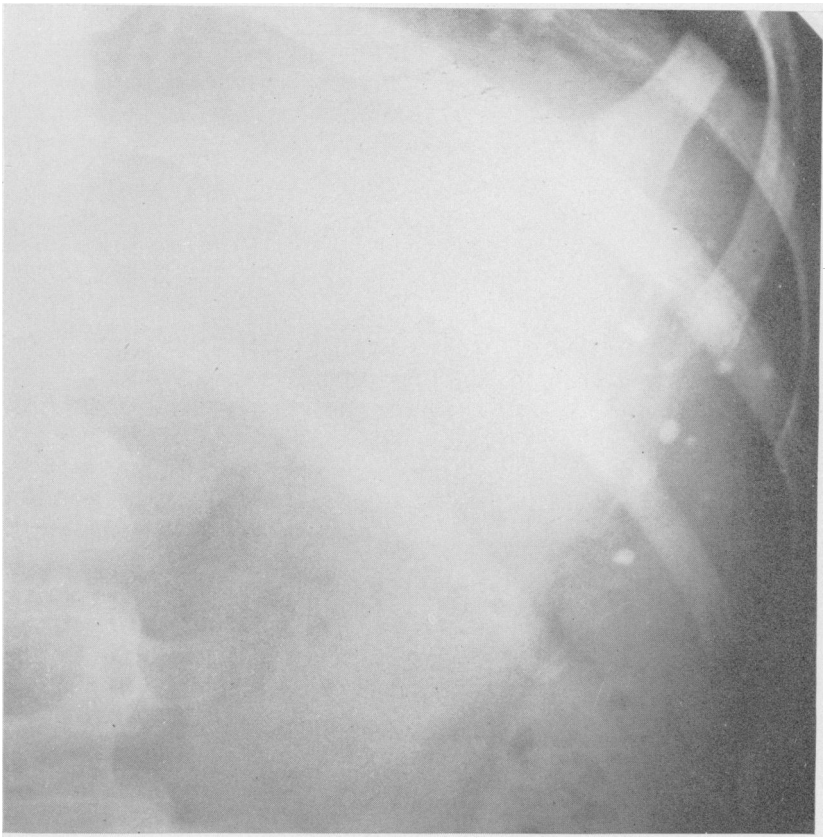

FIGURE 1.—Multiple calcification in the spleen (histoplasmin-positive, tuberculin-negative).

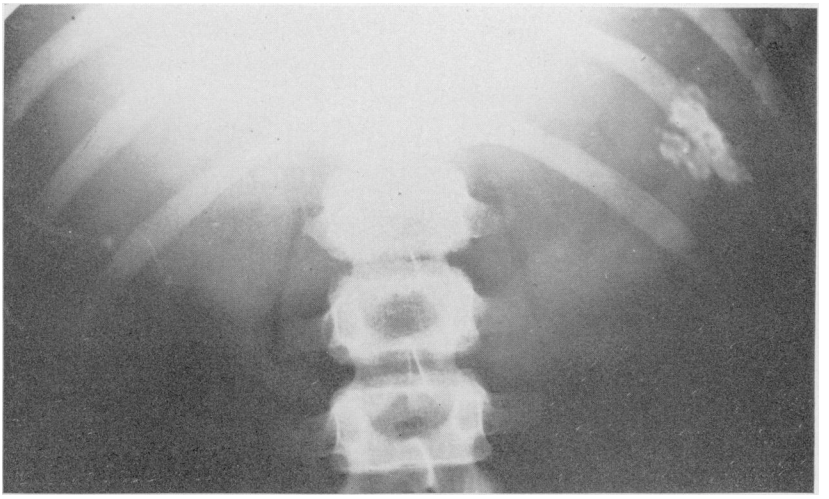

FIGURE 2.—Massive calcification in the spleen; possible calcification in the liver (histoplasmin-positive, tuberculin-negative).

DISCUSSION

It is the purpose of this report to point out that splenic calcifications have been observed as an incidental finding in a survey conducted in Kansas City, Mo. Since less than half of these cases were noted in tuberculin reactors, it appears that in this locality tuberculosis is probably not the most common cause of such calcification.

Kansas City, Mo., is an area where pulmonary calcifications are frequently found in nonreactors to tuberculin. Palmer (*8*, *9*), and Christie and Peterson (*10*, *11*) have shown that there is a close correlation between cutaneous reactions to histoplasmin and nontuberculous pulmonary calcifications. Furcolow et al. (*7*), reporting the more general findings from the material on which the present study is based, noted that approximately 40 percent of the white and a somewhat lower percentage of the Negro school children in Kansas City, Mo., reacted to histoplasmin. Further, results of the survey showed that approximately 80 percent of those with pulmonary calcification reacted. It is interesting to note that 78.6 percent of the children with splenic calcification reacted to histoplasmin. The percentage of histoplasmin reactors among those with splenic calcification is approximately twice as high as among the general school population and is almost identical with the percentage of histoplasmin reactors among those showing pulmonary calcification.

The findings presented here suggest that the agent causing the nontuberculous pulmonary calcifications may likewise cause splenic calcifications. Further, these findings suggest that the causative agent, or some of its products, has been spread by hematogenous routes. Since three instances of calcification, presumably intrahepatic, were also noted, it may be suspected that the liver is similarly affected by blood-borne agents.

SUMMARY

1. Twenty instances of splenic calcification were observed as incidental findings in a survey conducted in Kansas City, Mo.

2. It is suggested that tuberculosis is not the most frequent cause of such calcifications in that area.

3. The frequency with which splenic calcifications were noted in histoplasmin reactors suggests that the agent producing the sensitivity to histoplasmin is frequently responsible.

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REHABILITATION

"Restoration is at least as much a matter of spirit as of body, and must have as its central truth: Body and spirit are inextricably conjoined. To heal the one without the other is impossible. If a man's mind, courage and interest be enlisted in the cause of his own salvation, healing goes on apace, the sufferer is remade; if not, no mere surgical wonders, no careful nursing, will avail to make a man of him again. Therefore I would say: 'From the moment he enters the hospital, look after his mind and his will; give him food; nourish him in subtle ways; increase that nourishment as his strength increases. Give him interest in his future. Light a star for him to fix his eyes on, so that, when he steps out of the hospital, you shall not have to begin to train one who for months, perhaps years, has been living, mindless and will-less, the life of a half-dead creature.'

"That this is a hard task none who knows hospital life can doubt. That it needs special qualities and special effort, quite other than the average range of hospital devotion, is obvious. But it saves time in the end, and without it success is more than doubtful. The crucial period is the time spent in the hospital. Use that period to recreate not only the body, but mind and will power, and all shall come out right; neglect to use it thus and the heart of many a sufferer and many a would-be healer will break from sheer discouragement. A niche of usefulness and self respect exists for every man however handicapped; but that niche must be found for him. To carry the process of restoration to a point short of this is to leave the cathedral without spire. To restore him, and with him the future of our countries, that is the sacred work."

JOHN GALSWORTHY.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease wilhout knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED NOVEMBER 16, 1946

Summary

For the country as a whole, a slight decrease was recorded in the incidence of poliomyelitis. A total of 463 cases was reported, as compared with 489 last week and a 5-year (1941–45) median of 221. Increases occurred in 5 of the 9 geographic divisions—the Middle Atlantic, South Atlantic, South Central, and Mountain areas. Of 26 States reporting 5 or more cases currently, 14 showed an increase (145 to 222), while a decrease occurred in the other 12 States (288 to 198). States reporting 10 or more cases are as follows (last year's figures in parentheses): *Increases*—New York 44 (31), Michigan 33 (27), Minnesota 29 (27), Kansas 26 (15), Virginia 12 (1), Mississippi 10 (4), Louisiana 13 (4), Washington 11 (9); *decreases*—Illinois 47 (49), Wisconsin 18 (31), Iowa 11 (29), Missouri 26 (31), Nebraska 13 (14), Oklahoma 11 (13), Texas 18 (26), California 28 (34).

The total number of cases reported since March 15, the approximate average date of lowest seasonal incidence, is 23,423, as compared with 18,228 for the corresponding period of 1944 and a 5-year median for the period of 11,541. With 30 percent of the population, the North Central area has reported 12,717 cases, or 53 percent, of the total for the year to date, 23,889 cases.

Totals reported since the respective approximate dates of lowest seasonal incidence for diphtheria, measles, meningococcus meningitis, scarlet fever, smallpox, typhoid and paratyphoid fever, and whooping cough are below both the corresponding figures for last year and the 5-year medians. The total for influenza for the period since the seasonal low is 14,940, as compared with 21,848 for the same period last year and a 5-year median of 14,276.

Of 9 cases of Rocky Mountain spotted fever reported for the week, 6 were reported in Kentucky and 1 each in New Jersey, Pennsylvania, and Tennessee. The total for the year to date is 564, as compared with 461 last year and a 5-year median of 451.

Deaths recorded during the week in 93 large cities of the United States totaled 8,692, as compared with 8,663 last week, 8,836 and 9,143, respectively, in 1945 and 1944, and a 3-year (1943–45) average of 9,010. The total for the year to date is 414,560, as compared with 411,700 for the corresponding period last year.

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Telegraphic morbidity reports from State health officers for the week ended Nov. 16, 1946, and comparison with corresponding week of 1945 and 5-year median

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

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median 1941–45	Week ended—		Median 1941–45	Week ended—		Median 1941–45	Week ended—		Median 1941–45
	Nov. 16, 1946	Nov. 17, 1945		Nov. 16, 1946	Nov. 17, 1945		Nov. 16, 1946	Nov. 17, 1945		Nov. 16, 1946	Nov. 17, 1945	
NEW ENGLAND												
Maine	6	17	1	------	------	------	180	1	5	2	0	1
New Hampshire	0	0	0	------	------	------	51	9	9	0	0	0
Vermont	1	0	0	------	------	------	59	------	2	0	1	0
Massachusetts	13	4	3	------	------	------	170	160	160	1	1	7
Rhode Island	1	0	1	1	4	------	3	------	2	0	0	0
Connecticut	2	1	1	4	1	1	7	9	13	1	3	3
MIDDLE ATLANTIC												
New York	16	7	13	[1] 10	[1] 2	[1] 5	243	88	124	7	10	12
New Jersey	8	1	4	3	3	5	19	12	15	4	2	2
Pennsylvania	15	6	10	3	6	1	164	295	222	5	8	8
EAST NORTH CENTRAL												
Ohio	12	67	16	5	12	8	82	12	21	1	6	6
Indiana	7	24	20	------	109	22	8	4	16	0	3	1
Illinois	6	7	8	1	3	4	15	159	34	5	8	8
Michigan [2]	4	11	9	1	------	------	52	125	117	2	4	5
Wisconsin	3	3	3	28	167	28	47	23	66	2	3	2
WEST NORTH CENTRAL												
Minnesota	9	10	9	------	------	------	2	11	11	0	3	2
Iowa	3	32	4	------	4	2	6	9	18	1	4	0
Missouri	10	13	12	------	10	3	1	21	8	1	2	2
North Dakota	0	4	2	------	10	2	2	7	7	0	0	1
South Dakota	0	5	3	------	------	------	------	------	4	0	0	0
Nebraska	0	3	2	------	11	8	8	3	5	0	0	0
Kansas	6	9	8	2	------	1	6	8	9	0	2	2
SOUTH ATLANTIC												
Delaware	0	0	0	------	------	------	------	------	1	0	1	1
Maryland [2]	9	13	13	1	3	4	8	5	21	2	2	3
District of Columbia	0	0	0	------	------	1	2	------	1	0	0	0
Virginia	26	44	25	317	400	168	44	28	28	0	1	3
West Virginia	3	19	8	50	91	25	19	63	50	1	2	1
North Carolina	12	82	49	------	------	2	100	7	7	1	1	1
South Carolina	17	28	26	309	842	302	7	52	5	0	1	1
Georgia	12	23	25	14	53	35	26	4	8	0	0	2
Florida	18	11	16	11	4	3	9	1	8	1	2	2
EAST SOUTH CENTRAL												
Kentucky	30	16	11	------	5	1	------	86	11	0	1	1
Tennessee	12	44	15	30	45	26	9	1	15	4	2	4
Alabama	11	38	25	21	278	60	21	3	3	2	4	3
Mississippi [2]	12	28	18	------	------	------	------	------	------	1	1	1
WEST SOUTH CENTRAL												
Arkansas	9	24	18	12	61	53	9	8	8	1	1	1
Louisiana	13	15	13	1	76	15	2	2	2	4	4	1
Oklahoma	4	12	17	46	81	65	3	1	2	2	0	0
Texas	32	79	75	1,039	1,635	993	66	45	45	2	9	2
MOUNTAIN												
Montana	2	9	3	------	6	5	45	10	9	1	0	0
Idaho	1	1	1	8	12	1	10	101	18	0	0	0
Wyoming	3	0	0	15	8	6	1	3	3	0	0	0
Colorado	8	7	11	25	113	31	6	5	7	1	1	0
New Mexico	1	3	1	------	2	1	8	3	3	0	0	0
Arizona	4	2	5	130	55	84	16	1	3	0	0	0
Utah [2]	0	0	0	1	19	8	5	7	7	0	0	0
Nevada	1	0	0	------	------	------	1	------	1	0	0	0
PACIFIC												
Washington	3	4	4	------	------	------	9	210	49	0	1	1
Oregon	1	6	5	1	10	10	13	10	23	0	2	2
California	27	27	27	15	7	28	45	236	157	10	11	11
Total:	393	759	512	2,104	4,148	1,863	1,609	1,848	2,191	65	107	107
46 weeks	14,066	15,510	13,452	206,762	92,580	95,943	649,949	113,914	561,941	5,257	7,314	7,314

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

Telegraphic morbidity reports from State health officers for the week ended Nov. 16, 1946, and comparison with corresponding week of 1945 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever [2]		
	Week ended—		Median 1941–45	Week ended—		Median 1941–45	Week ended—		Median 1941–45	Week ended—		Median 1941–45
	Nov. 16, 1946	Nov. 17, 1945		Nov. 16, 1946	Nov. 17, 1945		Nov. 16, 1946	Nov. 17, 1945		Nov. 16, 1946	Nov. 17, 1945	
NEW ENGLAND												
Maine	1	0	0	35	33	18	0	0	0	0	1	0
New Hampshire	3	0	0	2	4	9	0	0	0	0	0	0
Vermont	3	2	0	5	7	3	0	0	0	0	1	0
Massachusetts	9	18	4	74	92	175	0	0	0	3	2	1
Rhode Island	1	1	1	14	5	8	0	0	0	0	0	0
Connecticut	4	1	1	17	20	32	0	0	0	1	1	1
MIDDLE ATLANTIC												
New York	44	18	18	162	200	237	0	0	0	2	4	6
New Jersey	5	12	6	46	67	67	0	0	0	0	0	3
Pennsylvania	6	15	8	106	149	163	0	0	0	3	4	4
EAST NORTH CENTRAL												
Ohio	5	12	8	168	240	240	0	0	0	3	0	2
Indiana	6	2	2	50	58	77	0	0	2	0	5	3
Illinois	47	7	12	108	136	162	0	1	0	0	2	2
Michigan [1]	33	4	4	141	103	129	0	0	0	1	0	1
Wisconsin	18	18	4	49	76	113	0	0	1	1	0	0
WEST NORTH CENTRAL												
Minnesota	29	7	3	26	50	50	0	0	0	0	0	0
Iowa	11	6	1	29	50	50	0	0	0	1	4	4
Missouri	26	25	3	23	60	62	1	0	0	0	0	0
North Dakota	4	0	0	5	10	11	1	0	0	0	0	0
South Dakota	1	0	0	2	10	12	0	0	0	0	0	0
Nebraska	13	4	4	27	3	15	0	0	1	0	0	0
Kansas	26	0	1	34	69	83	0	0	0	0	0	0
SOUTH ATLANTIC												
Delaware	1	0	0	6	5	7	0	0	0	0	1	0
Maryland [1]	4	2	2	24	47	47	0	0	0	0	3	3
District of Columbia	1	5	1	7	12	13	0	0	0	0	0	0
Virginia	12	1	1	35	152	84	0	0	0	2	2	2
West Virginia	2	0	1	29	88	79	0	0	0	1	0	0
North Carolina	6	4	4	33	76	83	0	0	0	0	1	2
South Carolina	0	2	2	11	24	14	0	0	0	1	0	1
Georgia	3	1	1	5	24	40	0	1	0	2	6	3
Florida	9	0	0	10	6	7	0	0	0	3	0	2
EAST SOUTH CENTRAL												
Kentucky	1	1	3	41	61	56	0	0	0	2	0	3
Tennessee	7	6	2	34	77	77	0	1	0	1	3	4
Alabama	3	1	1	23	31	33	0	0	0	1	2	1
Mississippi [1]	10	4	3	10	37	20	0	1	0	3	0	2
WEST SOUTH CENTRAL												
Arkansas	*8	1	1	1	12	12	0	3	0	1	0	4
Louisiana	13	1	1	4	30	10	0	0	0	3	4	8
Oklahoma	11	1	1	9	18	20	0	0	0	0	0	1
Texas	18	10	10	41	125	75	0	1	0	13	10	10
MOUNTAIN												
Montana	1	0	0	8	30	19	0	0	0	0	1	1
Idaho	3	1	0	22	15	15	0	0	0	1	0	0
Wyoming	1	0	0	4	1	5	0	0	0	0	1	0
Colorado	9	3	2	27	32	36	0	0	0	0	1	1
New Mexico	2	1	0	5	28	10	0	0	0	1	0	1
Arizona	1	2	0	3	16	11	0	0	0	1	0	1
Utah [1]	1	1	1	10	15	15	0	0	0	0	0	0
Nevada	0	0	0	0	0	1	0	0	0	0	0	0
PACIFIC												
Washington	11	14	4	42	30	30	0	0	0	2	2	1
Oregon	2	4	4	10	26	26	0	0	0	1	0	0
California	28	38	23	149	223	223	0	0	0	3	4	2
Total	463	256	221	1,726	2,683	2,683	2	8	9	57	65	94
46 weeks	*23,889	12,928	11,843	100,983	156,593	121,996	319	312	676	3,735	4,531	5,085

[1] Period ended earlier than Saturday.

[2] Including paratyphoid fever reported separately, as follows: Massachusetts 3 (salmonella infection), New York 1; Georgia 1; Florida 1; Texas 4; California 2.

*Correction: Arkansas, poliomyelitis, week ended October 19, 15 cases (instead of 16).

Telegraphic morbidity reports from State health officers for the week ended Nov. 16, 1946, and comparison with corresponding week of 1945 and 5-year median—Con.

Division and State	Whooping cough			Week ended Nov. 16, 1946							
	Week ended—		Median 1941–45	Dysentery			Encephalitis, infections	Rocky Mt. spotted fever	Tularemia	Typhus fever endemic	Undulant fever
	Nov. 16, 1946	Nov. 17, 1945		Amebic	Bacillary	Unspecified					
NEW ENGLAND											
Maine	20	64	55								
New Hampshire	10	15	15								1
Vermont	12	37	37								1
Massachusetts	120	130	130								2
Rhode Island	27	26	23								
Connecticut	23	48	54				1				1
MIDDLE ATLANTIC											
New York	258	290	310	4	2						3
New Jersey	103	224	186	2		1		1			2
Pennsylvania	130	210	164	1	1			1	1		1
EAST NORTH CENTRAL											
Ohio	53	101	142				1		1		2
Indiana	38	31	31	1			1		1		6
Illinois	90	78	150	4					1		10
Michigan [2]	160	171	171	2							
Wisconsin	238	102	153								2
WEST NORTH CENTRAL											
Minnesota	9	10	39	2							
Iowa	19	7	15								40
Missouri	17	21	26			1			3		2
North Dakota		3	10								
South Dakota		1	5								1
Nebraska	6	4	4								
Kansas	19	23	38						2		2
SOUTH ATLANTIC											
Delaware	7	1	1								
Maryland [2]	50	23	74								
District of Columbia	4	2	7								
Virginia	91	46	46			28			1		1
West Virginia	6	20	24								
North Carolina	37	50	69						1	1	
South Carolina	29	69	53	2	6				1	2	2
Georgia	1	12	12	1	1					8	3
Florida	34	3	7	4			1			10	4
EAST SOUTH CENTRAL											
Kentucky	7	45	52					6	2		1
Tennessee	25	39	23	1			1	1	3	3	2
Alabama	2	32	26				1			12	
Mississippi [2]										1	6
WEST SOUTH CENTRAL											
Arkansas	23	2	11						3	2	
Louisiana	4	2	2		1				1	18	4
Oklahoma	5	15	15	8	1	9					1
Texas	126	118	118	36	314	50				9	15
MOUNTAIN											
Montana	5		16								
Idaho		18	5								
Wyoming	11	3	3								
Colorado	2	38	38		1						4
New Mexico	10	1	2			1					
Arizona	8	1	3			25					
Utah [2]	5	10	18								1
Nevada	2										
PACIFIC											
Washington	22	38	38								
Oregon	3	6	17								1
California	40	78	122	2	4						2
Total	1,911	2,268	2,675	70	331	115	6	9	21	66	123
Same week, 1945	2,268			26	294	132	5	1	10	174	74
Average, 1943–45	2,358			41	528	170	10	[4] 1	9	[4] 110	
46 weeks: 1946	87,574			2,165	14,634	5,785	574	564	834	3,120	4,716
1945	111,507			1,740	22,502	9,857	579	461	650	4,655	4,458
Average, 1943–45	120,226		[4] 159,129	1,768	19,851	8,343	597	[4] 451	621	[4] 3,930	

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

WEEKLY REPORTS FROM CITIES [1]

City reports for week ended Nov. 9, 1946

This table lists the reports from 85 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

Division, State, and City	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
NEW ENGLAND												
Maine:												
Portland	0	0	------	0	------	0	2	0	3	0	0	------
New Hampshire:												
Concord	0	0	------	0	6	0	0	1	0	0	0	------
Massachusetts:												
Boston	11	0	------	0	5	0	10	8	8	0	0	31
Fall River	0	0	------	0	------	0	1	0	2	0	0	4
Springfield	0	0	------	0	2	0	1	2	2	0	0	13
Worcester	0	0	------	0	------	0	16	0	3	0	0	15
Rhode Island:												
Providence	0	1	------	0	1	0	1	0	2	0	0	35
Connecticut:												
Bridgeport	0	0	------	0	------	0	0	2	1	0	0	:
Hartford	0	0	------	1	1	1	0	1	2	0	0	2
New Haven	0	0	------	0	7	0	0	0	3	0	0	5
MIDDLE ATLANTIC												
New York:												
Buffalo	1	0	------	0	------	0	1	0	8	0	0	4
New York	10	2	2	0	19	1	38	11	43	0	2	36
Rochester	0	1	------	0	1	0	5	0	3	0	1	------
Syracuse	0	0	------	0	------	0	2	0	3	0	0	11
New Jersey:												
Camden	0	0	------	0	------	0	0	1	1	0	0	4
Newark	0	0	------	0	1	1	4	0	10	0	0	12
Trenton	0	0	------	0	3	0	2	0	1	0	0	------
Pennsylvania:												
Philadelphia	2	0	1	1	5	2	4	3	11	0	0	2
Pittsburgh	0	0	1	1	87	0	16	1	9	0	1	13
Reading	0	0	------	0	2	0	0	0	0	0	0	11
EAST NORTH CENTRAL												
Ohio:												
Cincinnati	1	0	------	1	------	0	2	2	10	0	0	4
Cleveland	1	0	3	0	41	1	8	9	18	0	0	9
Columbus	0	1	------	0	2	0	0	1	5	0	0	4
Indiana:												
Fort Wayne	0	0	------	0	6	0	0	0	1	0	0	2
Indianapolis	1	0	------	1	1	0	5	1	9	0	1	8
South Bend	0	0	------	0	------	0	0	0	0	0	0	------
Terre Haute	0	0	------	0	------	0	1	0	0	0	0	------
Illinois:												
Chicago	1	0	1	0	6	1	20	14	35	0	0	64
Springfield	0	0	------	0	0	0	2	2	1	0	0	3
Michigan:												
Detroit	0	0	------	0	6	1	6	5	34	0	1	43
Flint	0	0	------	0	------	0	2	4	0	0	0	4
Grand Rapids	0	0	------	0	1	0	2	0	3	0	0	8
Wisconsin:												
Kenosha	0	0	------	0	------	0	0	0	3	0	0	------
Milwaukee	0	1	------	0	5	0	2	1	12	0	0	62
Racine	0	0	------	0	------	0	0	0	8	0	0	1
Superior	0	0	------	0	------	0	0	0	1	0	0	------
WEST NORTH CENTRAL												
Minnesota:												
Duluth	1	0	------	0	------	0	0	4	1	0	0	9
Minneapolis	2	0	------	0	3	0	5	3	11	0	0	------
Missouri:												
Kansas City	0	0	------	0	------	0	5	3	2	0	0	------
St. Joseph	0	0	------	0	------	0	0	0	0	0	0	------
St. Louis	1	0	1	0	------	1	7	6	7	0	0	------

[1] In some instances the figures include nonresident cases.

City reports for week ended Nov. 9, 1946—Continued

Division, State, and City	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
WEST NORTH CENTRAL—continued												
Nebraska:												
Omaha	0	0	------	0	-------	0	1	7	7	0	0	2
Kansas:												
Topeka	1	0	------	0	1	0	0	1	1	0	0	------
Wichita	0	0	------	0	2	0	3	0	0	0	0	1
SOUTH ATLANTIC												
Delaware:												
Wilmington	0	0	------	0	1	0	1	0	2	0	0	8
Maryland:												
Baltimore	6	0	3	0	1	0	7	3	5	0	0	17
Cumberland	0	0	------	0	3	0	0	0	0	0	0	------
Frederick	0	0	------	0	13	0	0	0	1	0	0	------
District of Columbia:												
Washington	0	0	------	0	-------	0	5	2	0	0	2	15
Virginia:												
Richmond	0	0	------	0	9	0	0	0	2	0	0	1
Roanoke	2	0	------	0	-------	0	0	0	2	0	0	------
West Virginia:												
Charleston	0	0	------	0	-------	0	0	0	4	0	0	------
Wheeling	0	0	------	0	-------	0	3	0	0	0	0	1
North Carolina:												
Raleigh	0	0	------	0	-------	0	3	0	0	0	0	8
Wilmington	0	0	------	0	-------	0	0	0	0	0	0	------
Winston-Salem	0	0	------	0	39	0	0	0	3	0	0	4
South Carolina:												
Charleston	0	0	6	1	7	0	2	0	0	0	0	2
Georgia:												
Atlanta	0	0	2	0	-------	0	2	0	0	0	0	------
Brunswick	0	0	------	0	-------	0	0	0	1	0	0	------
Savannah	0	0	------	0	5	0	1	0	2	0	0	------
Florida:												
Tampa	2	0	------	0	-------	0	0	0	1	0	0	1
EAST SOUTH CENTRAL												
Tennessee:												
Memphis	1	0	------	2	-------	1	6	0	0	0	1	------
Nashville	2	0	------	0	-------	0	3	0	1	0	0	8
Alabama:												
Birmingham	2	0	------	0	-------	1	4	0	2	0	0	------
Mobile	1	0	1	1	1	0	1	1	1	0	0	------
WEST SOUTH CENTRAL												
Arkansas:												
Little Rock	0	0	------	0	1	0	2	0	0	0	0	------
Louisiana:												
New Orleans	3	0	1	0	-------	1	10	0	2	0	0	------
Shreveport	0	0	------	0	-------	0	5	0	0	0	0	------
Texas:												
Dallas	1	0	------	0	-------	1	3	0	4	0	1	3
Galveston	1	0	------	0	-------	0	2	0	0	0	0	------
Houston	1	0	------	0	-------	0	4	11	0	0	0	------
San Antonio	4	0	------	0	3	0	2	2	0	0	0	------
MOUNTAIN												
Montana:												
Billings	0	0	------	0	-------	0	1	0	0	0	0	------
Great Falls	0	0	------	0	1	0	2	0	0	0	0	------
Helena	0	0	------	0	-------	0	1	0	0	0	0	------
Missoula	0	0	------	0	1	0	2	0	0	0	0	------
Idaho:												
Boise	0	0	------	0	2	0	0	0	0	0	0	------
Colorado:												
Denver	2	0	3	0	3	1	2	1	5	0	0	------
Pueblo	0	0	------	0	-------	0	1	0	3	0	0	------
Utah:												
Salt Lake City	0	0	------	0	1	0	0	2	2	0	0	------

City reports for week ended Nov. 9, 1946—Continued

Division, State, and City	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
PACIFIC												
Washington:												
Seattle	0	0	------	0	-------	0	0	1	5	0	1	1
Spokane	0	0	4	1	1	0	1	2	3	0	0	------
California:												
Los Angeles	8	0	------	0	2	1	2	8	17	0	2	11
Sacramento	0	0	------	0	-------	0	0	2	0	0	0	1
San Francisco	0	0	2	0	3	1	10	1	8	0	1	6
Total	69	6	31	10	311	16	263	129	360	0	14	527
Corresponding week, 1945	74	------	60	12	436	------	276	------	598	0	18	693
Average, 1941–45	92	------	85	[2] 20	[3] 457	------	[2] 333	------	679	0	17	760

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

Anthrax.—Cases: Camden, 1.
Dysentery, amebic.—Cases: New York, 5; Philadelphia, 1; Detroit, 1; San Francisco, 1.
Dysentery; bacillary.—Cases: Philadelphia, 2; Detroit, 1; Charleston, S. C., 2; Memphis, 1; Los Angeles, 2.
Dysentery, unspecified.—Cases: San Antonio, 13.
Tularemia.—Cases: Cincinnati, 1; Memphis, 1; Los Angeles, 1.
Typhus fever, endemic.—Cases: Atlanta, 1; Tampa, 3; Nashville, 1; Mobile, 2; New Orleans, 3; Dallas, 1; Houston, 1.

Rates (annual basis) per 100,000 population, by geographic groups, for the 85 cities in the preceding table (estimated population, 1943, 33,912,200)

	Diphtheria case rates	Encephalitis, infectious, case rates	Influenza		Measles case rates	Meningitis, meningococcus, case rates	Pneumonia death rates	Poliomyelitis case rates	Scarlet fever case rates	Smallpox case rates	Typhoid and paratyphoid fever case rates	Whooping cough case rates
			Case rates	Death rates								
New England	28.9	2.6	0.0	2.6	58	2.6	81.4	36.8	68	0.0	0.0	284
Middle Atlantic	6.0	1.4	1.9	0.9	55	1.9	33.3	7.4	41	0.0	1.9	50
East North Central	2.4	1.2	2.4	1.2	41	1.8	30.4	23.7	85	0.0	1.2	129
West North Central	11.3	0.0	2.3	0.0	7	2.3	47.3	54.1	65	0.0	0.0	27
South Atlantic	16.5	0.0	18.2	1.7	134	0.0	41.4	8.3	38	0.0	3.3	94
East South Central	35.4	0.0	5.9	17.7	6	11.8	82.6	5.9	24	0.0	5.9	47
West South Central	28.7	0.0	2.9	0.0	11	5.7	80.3	37.3	17	0.0	2.9	9
Mountain	15.9	0.0	23.8	0.0	64	7.9	71.5	23.8	79	0.0	0.0	0
Pacific	13.2	0.0	9.9	1.6	10	3.3	21.4	23.0	54	0.0	6.6	31
Total	10.6	0.9	4.8	1.5	48	2.5	40.5	19.9	56	0.0	2.2	81

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended October 26, 1946.—During the week ended October 26, 1946, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox		5	1	133	198	39	37	125	85	623
Diphtheria		4	6	34	18	2	3		1	68
Dysentery, bacillary				2	2					4
German measles				1	6		3	4	7	21
Influenza					2	1			1	4
Measles		37	3	117	110	22	118	117	12	536
Meningitis, meningococcus								1	1	2
Mumps				54	219	15	102	20	99	509
Poliomyelitis		1	7	43	16	2	2	2	5	78
Scarlet fever		15	9	81	92	10		5	2	214
Tuberculosis (all forms)		9	6	128	46	16	11	3	109	328
Typhoid and paratyphoid fever			1	12	4	1			3	21
Undulant fever				2				5		7
Venereal diseases:										
Gonorrhea		15	15	152	120	39	47	40	81	509
Syphilis	1	11	6	139	79	13	12	13	33	307
Whooping cough		9		62	64	13	5	8	5	166

CUBA

Habana—Communicable diseases—4 weeks ended November 9, 1946.—During the 4 weeks ended November 9, 1946, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Chickenpox	4		Measles	16	
Diphtheria	15	1	Tuberculosis	10	2
Malaria	5		Typhoid fever	11	2

Provinces—Notifiable diseases—4 weeks ended November 2, 1946.—During the 4 weeks ended November 2, 1946, cases of certain notifiable diseases were reported in the Provinces of Cuba, as follows:

Disease	Pinar del Rio	Habana [1]	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	2	14	14	18		18	66
Chickenpox		3				1	4
Diphtheria	1	19	3	2		3	28
Hookworm disease		15		1			16
Leprosy		3				86	89
Malaria	2	14		1		25	42
Measles	1	13	8		1	1	24
Poliomyelitis			1	1	1	2	5
Tuberculosis (pulmonary)	16	50	24	66	11	52	219
Typhoid fever	36	36	9	35	20	43	179

[1] Includes the city of Habana.

NEW ZEALAND

Notifiable diseases—4 weeks ended October 5, 1946.—During the 4 weeks ended October 5, 1946, certain notifiable diseases were reported in New Zealand as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal meningitis	14	1	Ophthalmia neonatorum	1	
Dengue	1		Poliomyelitis	1	
Diphtheria	139	1	Puerperal fever	13	
Dysentery:			Scarlet fever	115	1
Amebic	5		Tetanus	3	
Bacillary	4		Trachoma	3	
Erysipelas	12		Tuberculosis (all forms)	193	66
Food poisoning	3		Typhoid fever	14	1
Malaria	4				

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

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

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

Cholera

Burma—Moulmein.—For the week ended November 2, 1946, 37 cases of cholera were reported in Moulmein, Burma.

Smallpox

China—Hong Kong.—Smallpox has been reported in Hong Kong, China, as follows: Weeks ended—November 2, 1946, 98 cases, November 9, 1946, 167 cases with 100 deaths, November 16, 1946, 217 cases with 137 deaths.

Straits Settlements—Penang.—For the period November 7 to 15, 1946, 56 cases of smallpox were reported in Penang, Straits Settlements.

Typhus Fever

Guatemala.—For the month of September 1946, 74 cases of typhus fever with 6 deaths were reported in Guatemala. Departments reporting the highest incidence are: Sacatepequez, 19 cases, 2 deaths; Chimaltenango, 17 cases, 1 death; Solola, 16 cases, 2 deaths.

Mexico.—For the month of September 1946, 107 cases of typhus fever were reported in Mexico. States reporting the highest incidence are: Federal District, 30 cases; Coahuila, 15 cases; Hidalgo, 11 cases; Nuevo Leon, 11 cases.

* * *

DEATHS DURING WEEK ENDED NOV. 9, 1946

[From the Weekly Mortality Index, issued by the National Office of Vital Statistics]

	Week ended Nov. 9, 1946	Corresponding week, 1945
Data for 93 large cities of the United States:		
Total deaths	8,663	8,974
Average for 3 prior years	8,732	
Total deaths, first 45 weeks of year	405,868	402,864
Deaths under 1 year of age	771	600
Average for 3 prior years	601	
Deaths under 1 year of age, first 45 weeks of year	29,699	27,296
Data from industrial insurance companies:		
Policies in force	67,327,836	67,302,402
Number of death claims	8,958	11,083
Death claims per 1,000 policies in force, annual rate	6.9	8.6
Death claims per 1,000 policies, first 45 weeks of year, annual rate	9.4	10.0

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