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OUR INADEQUATE TREATMENT OF THE MENTALLY ILL AS COMPARED WITH TREATMENT OF OTHER SICK PEOPLE

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The deplorable conditions in many State hospitals for the mentally ill and the huge costs of this type of illness have been recently discussed in several popular magazines and newspapers of wide circulation (*1*). If, as was said, we have been pound wise and penny foolish with regard to hospital care for mental patients, there are scarcely words available adequately to describe the almost total lack of efforts being made to prevent mental illnesses from occurring or developing to the point where hospitalization is necessary.

What is being done to stem the flow of men, women, and children into mental institutions? What is being done to prevent the breakdown of the 1 child out of every 20 who is now destined to spend some time in a mental hospital? What is being done to treat early disturbances of adults so that commitment may be prevented, and to help the childish old people adjust themselves to the community and the home?

Ordinarily a person ill in the physical sense, except in acute cases, receives considerable medical attention in the physician's office or at home, and hospitalization is reserved as a last resort. Why should mental disorders be an exception to this practice? Why are many mental cases allowed gradually to develop without any medical attention until commitment must be obtained? Why is it necessary for commitment to be sought in order to get medical attention for mentally ill persons? The majority of first admissions to our State hospitals have never seen a mental specialist until they arrive at the hospital, already seriously ill, with only the prospect of sharing a psychiatrist's time with perhaps 400 other patients. Why neglect the mentally ill until they have progressed to commitment to a hospital with only a 50–50 chance of not having to die in a mental institution?

We spend fortunes for hospital care of the end products of mental disturbances but only pittances for prevention. Blindly and foolishly we continue to pay $210,000,000 (*2*) a year of public funds to maintain our mentally ill in hospitals, but only about $5,000,000 (*3*) for the

support of mental hygiene clinics to prevent their commitment. To reverse the old adage about the forest and trees, we haven't been able to see the trees, represented by our individual early cases, because of the forest of mentally ill in the hospitals.

Besides the careless, and perhaps callous, indifference based on ignorance of the true conditions, and lack of appreciation of the magnitude of the problem, there are several reasons why this situation exists. One is the paucity of physicians trained in the care of mentally ill persons. Many cities of 50,000 to 100,000 population have no qualified psychiatrist in practice. Although 58 percent of all hospital beds in this country are occupied by mental cases (*4*), and 50 percent of all cases going to private physicians have emotional or mental disorders as an accompanying condition, only 1.2 percent of all physicians in this country are psychiatric specialists.

Then, too, psychiatrists are concentrated in certain areas, particularly the eastern centers of population. Even if they were distributed evenly according to population, which would provide 1 psychiatrist for every 57,247 persons in this country, in many sparsely settled districts they would be too far apart to be available to the entire population. Furthermore, of about 2,300 psychiatrists in this country, approximately 54 percent are employed in mental institutions, where they are for the most part unavailable for early treatment designed to prevent hospitalization (*5*).

Mental hygiene is peculiarly a job for a mental hygiene clinic team. Besides the psychiatrist, a social worker is needed to make investigations of the environmental conditions of the home, neighborhood, and school to which the patient is trying to adjust himself, and a psychologist is necessary to evaluate the intellect with which the psychiatrist must work in his treatment procedures.

The early treatment of emotional and mental disturbances which may result in incapacitating mental illness and hospital admission and the dissemination of the general principles of living which reduce the number of insolvable conflicts with the environment are the practical aims of mental hygiene. Although some psychoses are due to infections, toxic agents, including alcohol, and the deterioration of old age, most mental disturbances occur when persons are faced with what to them are intolerable situations. These may be overwhelming because of hereditary deficiencies of the individual, a weakness of the personality stamina resulting from educational and environmental forces, or a combination of these two factors. In a fully developed mental disorder this mechanism of flight from reality may be obscure; but it is likely that, at an earlier period, reaction to environmental stresses was shown as a behavior problem in a child or an emotional or neurotic reaction in an adult. The earlier these problems are recognized, the easier it is to correct them. Treatment

of the child's behavior problem or the adolescent's difficulty may prevent a fully developed mental disorder.

Let us consider the distribution of these teams operating as mental hygiene clinics. Since they are specialists and command relatively high salaries, they are found chiefly in the larger centers of population. Full-time clinical service for child guidance is provided in only 27 of the largest cities of the country and is almost nonexistent in small cities and rural areas. About one-fourth of the cities over 100,000 population and almost two-thirds of the cities between 50,000 and 100,000 population have no psychiatric clinic facilities for children or adults (*6*). Even in communities favored by mental hygiene clinics there is none in which the service is adequate to the need. So we see why it is that, today, all over the country, troubled individuals are progressing, or rather, regressing, without benefit of any early medical attention, to the point of hospitalization, with consequent depressing outlook for cure.

Another reason why early medical attention is not obtained for those developing mental illnesses is the unfortunate stigma that is attached to mental disorders of all kinds, a stigma dating back to the days when insanity was believed due to demons and evil spirits, but an anachronism today. Mental illnesses have been recognized by many as deserving of medical attention for some years, but the popular attitude still too frequently attaches a vague disgrace to individuals or families in which mental illness occurs. An unfortunate tendency persists to ignore early mental signs and to avoid facing the issue by consulting a specialist in mental illnesses.

Archaic, persecutory commitment practices, which include court appearances and sometimes jury trials, help to perpetuate this attitude. In too many States the mentally ill are thrown into jail, after which they are transported to the State hospital by sheriffs, who frequently substitute handcuffs, chains, and strait jackets for humane, intelligent handling, which could be furnished by the State hospital attendants. Why should a mentally ill patient be handled as a criminal in order to get for him the treatment that he needs?

In many ways mental disorders and venereal diseases have been placed in the same category in the public mind. Both are too often considered disgraces to individual and family, issues not to be faced but suppressed and concealed as long as possible. Treatment in both types of illness has tended to be long, unsatisfactory, and expensive, and the public has been confused and defrauded by the services of charlatans and quacks. Venereal diseases are now being recognized, by the public as well as the medical profession, as illnesses, apart from moral issues, which can be successfully treated and can perhaps eventually be eliminated as major problems if attacked according to the recognized principles of public health practice.

Venereal diseases can now be mentioned by name in the press and over the radio, and an increasing number of people are able to discuss these subjects frankly and intelligently. Mental illnesses, while lacking specific treatments such as are now available for certain venereal diseases, need no longer be treated by mere isolation and punishment.

Excellent mental hygiene work has been done in certain localities by private foundations, universities, State hospital and welfare groups; but from these scattered random efforts a cohesive national program can never be resolved. The National Committee for Mental Hygiene has been active in a crusade for mental health for more than 30 years. These efforts have included an educational program, the improvement of State hospitals, the development of mental hygiene techniques, with the establishment of demonstration clinics, and fellowships for special study. But it is not the responsibility of such an organization, supported by private subscriptions, to establish adequate mental hygiene service to the Nation any more than it is its function to provide other health services.

The recognition of mental illness as a public health problem offers the support of the Nation-wide system of Federal, State, and local health organizations which can successfully apply the knowledge we now possess concerning the control of this type of illness. Passage of the Federal Social Security Act in 1935 has made it possible for the United States Public Health Service to undertake the development of mental hygiene services as a part of organized health departments throughout the country (7).

Approximately $11,000,000 a year is allocated to the States by the Public Health Service for the extension of general public health work in the States. Under Surgeon General Thomas Parran an effort is being made to direct some of this money into mental hygiene programs, with the ultimate aim of a mental hygiene division in the health department of every State which has not already established an effective program of mental hygiene and a mental hygiene clinic for every community which now has a general public health program. The full-time public health organizations in the 1,665 counties out of a total of 3,071 in the United States may well form the basis for an extensive mental health service.

A mother will take her problem child with less prejudice and with less hesitation to the health center where she has gone for prenatal care and immunizations and vaccinations than she would to the outpatient department of an "insane asylum." The activities of health officers in this field identify mental disorders as problems for the medical profession and problems for which there is some hope in successful early treatment and in prevention.

A mental hygiene clinic is expensive, including as it does several specialists; and it is true that cases cannot be run through a mental hygiene clinic in the same numbers and in the same manner as they can in a vaccination clinic or a venereal disease clinic. But, expensive and time-consuming as mental hygiene efforts are, the results are correspondingly profitable.

These results are profitable with regard to both financial and human, or social, values. If, as estimated, every commitment prevented saves a State from $5,000 to $7,000, it is only necessary to eliminate three commitments a year to correctional institutions or mental institutions to pay the budget of a mental hygiene clinic. There is no doubt that the efforts of such a clinic group throughout a year accomplish this, without taking into account the improved individual and family adjustments in which institutionalization might never be a problem, a divorce saved now and then, and a suicide prevented here and there. One-third of the problems referred to good child guidance clinics are solved, and an additional third are definitely improved.

It was estimated a few years ago in Indiana that if that State's present system of community mental hygiene services were extended to cover the entire State, from 15 percent to 20 percent of patients treated, who would under ordinary circumstances be committed, would not become institutional cases, thus saving the State approximately $583,440 each year. It is estimated that this State could save $284,452 more annually by the discharge of approximately 15 percent of the institutional cases if adequate mental hygiene clinics existed for parole supervision (*8*).

The Public Health Service hopes to establish a neuropsychiatric research institute which will investigate some of the unsolved problems in this field of mental health. At the present time the estimated total annual expense for research into nervous and mental diseases in this country is only about $2,000,000 (*9*), a much smaller amount than is justified by the total national cost of mental illness, which amounts to $777,000,000 (*10*) every year, including both maintenance costs and economic loss due to unemployment. The Federal Government alone has paid out close to $1,000,000,000 (*11*) for the care and pensioning of neuropsychiatric veterans of the last war.

The mental hygiene consultant of the Public Health Service is finding ready acceptance among the State health officers of responsibility in this field of mental disorders. Since none of the money available under the Social Security Act as grants-in-aid to the States is earmarked for mental hygiene purposes, readjustment of State budgets, with a decrease in the amount spent for other health purposes, is necessary in order to establish mental hygiene programs.

Difficult as this rearrangement is, it is being done in 12 States for the fiscal year 1942, as compared with 4 which previously had a mental hygiene program in the State health department.

A special training program to equip men to do this type of work is being started at one of the leading universities this year, and a limited number of fellowships using Federal funds are available through State health departments.

The President's Committee on Medical Care, reporting in 1938, recommended that Congress make available increasing amounts up to $10,000,000 a year for a field program of mental hygiene similar to special annual appropriations for venereal disease control. Appropriations for this purpose, however, have not been made.

In tumultuous, troubled times such as we are now experiencing, and in the days ahead, when joy gives way to anxiety and hope gives way to despair, an increase in mental disease is to be expected. Neuropsychiatric conditions are the fifth most important cause for rejection by the Selective Service boards and have resulted in approximately 63,000 men, up to May 1, 1941, being thrown back into community life with known nervous and mental disorders (*12*). Most of these individuals will receive no attention, although many of them are early cases in which treatment would be most promising.

The post-war period will be one of difficult adjustment for men relieved from military duties. It is hoped that we may have some better method of meeting this problem than mere custodial care at the hospital level and pensioning.

Faced with the enormous problem of mental disorders, a country-wide mental hygiene program is not the least of the needs that confront the Nation. Perhaps, just as the present successful campaign against venereal diseases resulted from our experience in the first World War, the present emergency will focus attention on mental disorders and result in a national, all-out, determined attack on this type of illness. It is inconceivable that this huge problem of emotional and mental illness will not eventually have accorded it the interest and financial support that it deserves.

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DERMATITIS FROM CUTTING OILS [1]

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Cutting oils are the most frequent causes of dermatitis among machinists and metal workers.

Cutting oils are divided into two large groups, the insoluble and the soluble. The insoluble ones are used mainly as lubricants, aiding the tools in the cutting operation, and the soluble ones are used mainly as cooling agents. Mineral oils and greases are also used in machine shops for lubricating moving parts and for rust prevention. These oils and greases may produce the same types of lesions as the cutting oils.

The soluble cutting oils in general consist of mineral oil about 60 to 95 percent, soap about 5 to 30 percent, and volatile matter from 0 to 10 percent.

The mineral oil content may be a paraffin or naphthenic type of oil. The soap content may consist of sodium or potassium salts, of fatty acids, rosin, sulfonic acids or sulfonated vegetable or animal oils. The soluble oil may contain more than one type of soap and the proportions vary with individual manufacturers.

The volatile contents consist of materials which are used as mutual solvents or cutting agents which stabilize the mixture of oil and soap. They may consist of alcohol or glycol phenol, nitrobenzene, cresol, and similar materials and water. Some of these compounds serve to inhibit the growth of bacteria and fungi in the oil.

The principal function of a soluble oil is that of cooling the cutting tools so that they do not lose their temper and break or chip; the secondary function is that of lubricating. Soluble cutting oils are mixed with water, in varying proportions (3 to 60 parts of water per part of oil) and allowed to flow continuously over the cutting operation.

The insoluble cutting oils—fatty oils, such as lard oil—were first used to lubricate cutting tools. Since petroleum has become generally available, it has been added to or substituted for these fatty oils in order to offset their high cost and their tendency to turn rancid.

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

Sulfur was chemically combined with the mineral oil-fatty oil blend to increase the film strength and to provide the added lubrication which was needed for cutting tougher steels and at higher speeds.

The insoluble type of cutting oil consists principally of 55 to 100 percent of mineral oil, 0 to 30 percent of fatty oil, 0 to 10 percent of sulfur, and 0 to 5 percent of chlorine. The mineral oil content may be of a paraffin or naphthenic type. The fatty oil content may be oleic acid, lard oil, fish oils, and vegetable oils. The purpose of the fatty oil is to act as a sulfur carrier and to increase the lubricating properties of the oil. The presence of the sulfur enables deeper cuts to be made in the metal without harming the cutting edge. Chlorine performs, in general, the same function as sulfur, but tends to cause corrosion and rusting of steel.

Firms that sell the vegetable or animal oils to the cutting oil manufacturers may incorporate into them chemicals known as inhibitors, which prevent the oil from becoming rancid. These inhibitors must be of a type that will not rust iron and are frequently phenolic compounds of the type of phenolic amines. The insoluble cutting oils may also contain small percentages of these inhibitors.

ACTION OF CUTTING OILS ON THE SKIN

All petroleum oils have the property of defatting the skin. The defatting action is somewhat lessened by the animal or vegetable content of the cutting oil, but since these oils are contained in comparatively small percentages in cutting oil, their action does not altogether counteract the defatting action of the mineral oil content. All oils may plug up pores of the skin and form comedones.

The chlorine content may be sufficiently high to irritate the skin.

The sulfur content of a cutting oil may cause a dermatitis because of the actual effect of the sulfur itself on the skin, or because the sulfur may be converted into such compounds as hydrogen sulfide or sulfur dioxide by the action of the heat generated by the cutting operation.

The animal or vegetable oils, especially when they are rancid, may irritate or sensitize the skin of some of the workers.

The phenols, cresols, nitrobenzene, and other inhibitors are usually not present in sufficient amounts to be primary irritants, but they may act as sensitizers and cause allergic eczemas.

The type of skin has a marked influence on the worker's susceptibility to dermatitis from the cutting oils. A greasy skin having active sebaceous glands is less apt to be defatted than is a dry skin. A smooth, hairless skin is less apt to develop comedones than a hairy one.

Cutting oils, after being used, contain many steel slivers which

may wound the skin of the workers, especially if old, dirty towels and old, used waste full of slivers are employed for drying the hands.

TYPES OF DERMATITIS FROM CUTTING OILS

Comedones of the hands and fingers occur in nearly all workers handling cutting oils unless they are particularly careful about washing their hands after work. Folliculitis is the most frequent type of dermatitis caused by cutting oils. Folliculitis occurs generally on the extensor surface of the forearms and of the thighs where oil-soaked sleeves and trousers have closest contact with the skin. The bacteria found in the lesions are usually the pathogenic staphylococci, which are usually found in ordinary boils and which may be found on the intact skin. It is true that workers sometimes expectorate into cutting oils and that the oils may even be contaminated with the colon bacillus. However, we believe that it is the bacteria on the skin which causes the folliculitis from cutting oils and not the bacteria which may be in the oil. The United States Public Health Service has analyzed samples of sterilized and unsterilized insoluble cutting oils used by workers who have had cutting oil dermatitis and found no significant number of staphylococci or streptococci. A large square-end, rod-like organism containing spores and some indefinite forms suggestive of yeasts and molds were found in the samples.

The insoluble cutting oils as a class are not suitable for the growth of bacteria because they contain such a large percentage of petroleum oil and because many of them also contain an inhibitor which has antiseptic properties. Soluble petroleum oils, when diluted for use, are not well suited as culture media because of the fact that they consist mostly of water and a small percentage of the soluble oil. It is possible, however, for bacteria to grow in those having a high content of fatty oils. The lard oils contain animal or vegetable oils and therefore bacteria grow more easily in them. (Lee and Chandler [1] described a short Gram-negative rod which they found in cutting oils and which they called *Pseudomonas oleovorans*. This organism has not been shown to be a pathogen.)

Infected follicles may develop into boils and even carbuncles and infection of wounds of the skin caused by metallic slivers may cause the development of boils or even cellulitis. Bacteriologic examination of the infected follicles or boils usually shows the ordinary pyogenic bacteria, which are found in ordinary boils and which are usually present on the skin.

The defatting action of cutting oils on the skin may cause drying, cracking, and fissuring. The open fissures are subject to secondary infection and its train of symptoms—boils, lymphangitis, and even

[1] Lee, Melba, and Chandler, Asa C.: A study of the nature, growth and control of bacteria in cutting compounds. J. Bact., 41: 373 (1941).

septicemia. Dry skins and senile skins are most likely to be affected in this manner because the skin glands are not sufficiently active rapidly to replace the fat extracted from the skin by the mineral oils.

Metal slivers in the oils and in the waste used for drying the hands often become imbedded in the skin and may be the site of secondary infection.

Some of the mineral oils have keratogenic properties and cause the appearance of small, flat, brownish papillomas on the hands, arms, and other parts touched by the oil or oil-soaked clothing. We have found that about 10 percent of all workers in oils and greases have these growths. They are usually not troublesome and the worker may not be aware of their presence.

Certain mineral oils are carcinogenic, but fortunately the mineral oils of North America are low in carcinogenic properties. Nevertheless a few cases of skin cancer are caused in the United States by mineral oils. Cancers caused by oils are usually of the prickle cell type. They are usually multiple and occur on the parts exposed to the oils or the oil-soaked clothing, the hands, arms, neck, and scrotum. They do not as a rule have early metastases.

Allergic eczemas are the least frequent types of cutting oil dermatitis. They are caused by hypersensitivity to the animal or vegetable oil, or inhibitor contained in the cutting oil, or by allergy induced by the disinfectant, which in many instances is added to cutting oils when they become rancid. We have seen cutting oils that contain as high as 5 percent of a phenolic disinfectant which had been added to it from time to time during the prolonged period that the cutting oil was in the machine.

Dermatitis among workers exposed to cutting oils may not be caused by the cutting oil, but by the harsh soaps, bleaches, and solvents which the workers use to wash their hands after work. Careless workers are apt to use such solvents as kerosene and gasoline to remove dirt and grease from their hands quickly. Others will use sand soap, soaps containing a high percentage of alkali, and even such substances as bleaching powder to clean up after work. Skins that are naturally dry will not stand such cleansers without at least becoming chapped or fissured.

PREVENTION

Prevention of dermatitis from cutting oils consists chiefly in cleanliness of the person, of the clothes, of the machines, and of the oil. For personal cleanliness, workers with cutting oils should be provided with adequate washing facilities, hot and cold running water, and shower baths, and they should be compelled to use them under supervision.

Clean work clothes should be provided daily and the anterior

surfaces of the body and the arms should be protected by aprons and sleeves made of impermeable material, such as the new synthetic resins.[3]

Toilet or liquid soaps should be provided for the workers and placed in convenient locations in the wash room. There should also be provided places where workers who have dry or fissured skins, or acute or chronic dermatitis can wash after work with a cleanser that will not further defat or irritate the skin. Such a cleanser has been devised and consists of a neutral sulfonated castor oil, to which 2 percent of a wetting agent such as a fatty alcohol sulfate is added.[4] The sulfonated castor oil is a good emulsifier and the wetting agent is a good detergent and works in hard or soft water. Its defatting action is counteracted by the sulfonated castor oil. If it is desired to remove dyes or stains from the skin, the addition of 1 to 2 percent of trisodium phosphate or sodium hexametaphosphate to this mixture will increase its cleansing powers and not materially increase its irritating powers. The use of kerosene, gasoline, or other fat solvents and strong bleaches and scouring soaps should be prohibited for skin cleansing.

The machines should be kept as free from old grease and dirt as possible by washing daily.

The oil should be changed frequently, at least once a week, and either discarded, or, if it is to be reused, it should be screened to remove metal, sterilized to remove bacteria, and neutralized to remove rancidity. Such sterilization can best be done by a central sterilizing system, connecting with each of the machines, and recirculating the oil, or if this is not possible, the oil may be removed from each individual machine and carried to the central sterilizing system. Additional antiseptics should not be added to used or rancid oils. Such a practice increases the irritating properties of the oil and is not necessary if the oil is filtered, neutralized, and heat sterilized.

Clean towels should be given to the workers every day, or they should have free access to clean waste. The towels should be so laundered that all slivers are removed. Waste should be discarded and not laundered because it is impossible to remove all slivers from waste.

PROTECTIVE OINTMENTS

Since the most frequent type of occupational dermatitis caused by cutting oils is a folliculitis and boils, cleanliness is much more important in the prevention of dermatitis than are protective ointments. When protective ointments are used, they should be of the type that will fill the pores of the skin with an innocuous vegetable or

[3] Schwartz, Louis, Warren, Leon H., and Goldman, Frederick H.: Clothing for protection against occupational skin irritants. Pub. Health Rep., 55: 1158–1163 (1940).

[4] Schwartz, Louis: A new industrial skin cleanser. Pub. Health Rep., 56: 1788–1790 (1941).

animal fat to prevent the entrance of the mineral oil. Such an animal or vegetable fat will also act to protect the skin from the defatting action of the mineral oil, because the mineral oil must first dissolve out the protective ointment before it will act on the fat of the skin. Such a protective ointment should also contain a small percentage of a wetting or emulsifying agent such as the fatty alcohol sulfates, in order to make it easily removable from the skin with water. A small percentage of a harmless preservative, such as sodium perborate, is also desirable in order to prevent the fat in the ointment from becoming rancid.

Protective ointments which form films on the skin, insoluble in oil, are not as good as are those described above, because films insoluble in oil are soluble in water and the perspiration which forms beneath them washes them off. Protective ointments which form films soluble in oil are not desirable because the cutting oil washes them off. The sulfonated oils will wash away both types of films. Films will crack when the hands and fingers are flexed and leave areas of the skin unprotected.

Workers should be prohibited from expectorating into the oil, or from contaminating it in any other way.

EDUCATION

The safety director or the physician in charge of the medical service of the plant should make it his duty to give occasional lectures to the workers as to the hazards of dermatitis from cutting oils and make them acquainted with the methods of prevention outlined above. In this educational program, the placing of placards in suitable places calling the attention of the worker to the dermatitis hazards of cutting oils and the methods of prevention has been found to be effective.

DIFFERENTIAL DIAGNOSIS

Cutting oil folliculitis and boils must be differentiated from folliculitis, boils, and furunculosis occurring from nonoccupational causes. Cutting oil folliculitis and boils occur on parts of the body where there is most frequent contact with the cutting oils or with soiled clothes. The extensor surfaces of the forearms, thighs, and legs are the favored sites of cutting oil dermatitis. The condition may, however, occur on other parts of the body coming in contact with oil-soaked clothing. Nonoccupational boils usually occur on other portions of the body, such as the back of the neck and the back. Occupational folliculitis and boils are usually multiple, whereas nonoccupational types are more apt to be a succession of solitary boils or furuncles. In occupational folliculitis and boils, evidences of oil comedones can usually be seen on the parts affected. These do not occur in nonoccupational types.

In differentiating the allergic eczematoid type of cutting oil dermatitis from eczemas of unknown origin, the site of the lesions must be considered, as well as the result of patch tests with the oil. Allergic occupational eczemas due to cutting oil usually begin on the arms and hands or on parts of the body which have contact with oil or oil-soaked clothing. Patch tests with the particular cutting oil handled by the worker should be positive if the oil is the cause. Such positive patch tests should be followed with patch tests with each of the ingredients of the cutting oil to determine the actual chemical in the oil to which the worker is sensitive.

Fungus infections and phytids of the hands and forearms offer a difficult problem in differential diagnosis, but here again patch testing in conjunction with the history, site, and morphology of the lesions will usually lead to a correct diagnosis.

TREATMENT

Comedones, folliculitis, and boils caused by cutting oils can be successfully treated by cleanliness and antiseptics. Cleanliness consists in a daily change of work clothes and frequent washing of the parts affected. Pus should be evacuated by surgical means and moist dressings of solutions of boric acid, bichloride of mercury 1 to 1,000, or potassium permanganate 1 to 2,000, if judiciously used, will usually clear up the condition.

The allergic types of cutting oil dermatitis are best handled by removing the affected worker from contact with cutting oils and then treating the dermatitis. Antiseptic lotions should be used for the moist types of dermatitis and ointments for the dry, chronic types. Moist dressings of boric acid, Burow's solution, bichloride of mercury 1 to 1,000, and the like, for the acute moist types, and boric acid ointment, zinc ointment, Lassar's paste, and coal tar preparations are suggested for the dry stages or dry eczematoid types.

For dry, defatted skins an ointment consisting of a vegetable or animal fat should be given the worker to rub into his hands before and after work. The ointment suggested above as a protective may also be used for this purpose.

THE LEAD AND ARSENIC CONTENT OF URINES FROM 46 PERSONS WITH NO KNOWN EXPOSURE TO LEAD OR ARSENIC[1]

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A recent investigation of the lead and arsenic content of the blood and urine from a large number of individuals living in an apple growing

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

locality in the State of Washington has been reported (*1*). All of the 683 individuals, for whom analyses were made, had a potential exposure to lead arsenate either by ingestion of apples having on their surface some lead arsenate spray residue and/or by inhalation of dust, spray, or particulate matter containing lead arsenate. The number of persons eating no apples at any time during the year was so small that a satisfactory control group could not be found among the individuals studied.

The purpose of the present investigation was to secure urinary lead and arsenic values for children and adults having no known exposure to lead or arsenic and to compare the values with those for similar groups.

A group of 46 individuals working in or living near the National Institute of Health, Bethesda, Md., was studied. These persons were either personnel of the laboratory staff or members of their families. None of these, so far as it could be ascertained, had had any appreciable exposure to lead or arsenic compounds.

From 28 adults (27 males and 1 female) and 18 children (14 boys and 4 girls) from the households of some of these adults, morning specimens of urine were collected during the month of March. Lead- and arsenic-free bottles were issued on a given day and the individuals were requested to fill the bottles (250-cc. capacity) with the first morning specimen passed the following morning and return it to the laboratory. Nearly all the adults filled the bottles on the first voiding; the remainder made subsequent additions until 250 cc. were obtained. Urine was collected from the children 2 to 4 days later and the same care was taken to avoid contamination of the specimens. With most of the children, however, several voidings were necessary to fill a bottle.

Analyses of these specimens were started on the same day they were brought to the laboratory. The condition and color of each specimen was noted and recorded, the pH and specific gravity were measured, and the phosphate, lead, and arsenic content was determined. The modified Leconte uranium acetate method (*2*, *3*) was used for total phosphate analyses, a photometric dithizone method for lead (*1*, *4*), and the Gutzeit method (*1*) for arsenic.

EXPERIMENTAL RESULTS

The determinations made on the specimens are summarized in table 1 which gives both the minimal and maximal as well as average values. It can be seen that the urines for the two groups are remarkably similar both in range and in average values. The averages for the pH measurements on the two groups may differ significantly (critical ratio of the two means=2.5); for all the other kinds of determinations shown the averages do not differ significantly for corresponding groups (critical ratios of means are less than 2.0). However, this

similarity in values for adults and children holds only with average values for groups since a child with a specimen showing a low concentration for a given constituent frequently comes from a family in which the parent's specimen shows a considerably elevated concentration value for the same constituent. This can be seen from inspection of table 2 in which urinary lead concentration values are compared for corresponding members of families.

TABLE 1.—*Analytical findings for 46 residents of Bethesda, Md., having no known exposure to lead or arsenic*

Measurement	Range in values		Average values		
	Children	Adults	18 children	28 adults	Group of 46
Age	2–12½	21–52	7½	33	23
pH	5.2–6.8	5.2–6.7	6.1	5.8	5.9
Specific gravity	1.007–1.025	1.006–1.027	[1] 1.018	1.016	[2] 1.017
P_2O_5 (g./100 cc.)	.06–.43	.06–.31	.22	.19	.20
As (mg./l.)	.000–.065	.000–.060	[1] .014	[3] .015	[4] .014
Pb (mg./l.)	.000–.048	.008–.051	[1] .026	.030	[2] .028

[1] Based on 17 observations.
[2] Based on 45 observations.
[3] Based on 26 observations.
[4] Based on 43 observations.

Similarly, considering urinary arsenic concentration values, in nine families for which specimens from one parent and the child (or children) were available, the child of the parent with the highest urinary arsenic concentration showed no urinary arsenic while the parent of the child with the highest value had no detectable quantity of arsenic in the specimen examined. However, the mean arsenic values for the two groups, adults and children, were practically identical, being 0.013 and 0.014 mg. per liter, respectively.

TABLE 2.—*Comparison of urinary lead concentration values for parent and child or children in corresponding families*

Family number	Urinary lead concentration in mg. per liter		Family number	Urinary lead concentration in mg. per liter	
	Parent	Child or children		Parent	Child or children
1	0.051	0.010	8	0.036	0.023
2	.018	.020, .035	9	.046	.015, .032
3	.038	.037	10	.008	.041
4	.018	.011			
5	.015	0.048, .035, .011	Total number	10	16
6	.015	0.041, .019, .030			
7	.046	.005	Average	0.029	0.026

It is evident, therefore, that similarity of environmental conditions existing within a household is not sufficient to produce uniformity in the urinary lead or arsenic concentration measurements of different members of a given family. Statistical treatment of the data, together with other experimental work to be reported later, give results which are consistent with the existence of diurnal variation in the urinary lead excretion of the individual studied.

Although both the urinary lead and urinary arsenic values for the groups of children and adults showed no significant differences between the groups at this time of year it is not certain that this equality will be realized at a different time of year when the average temperature is greatly increased. Measurements have been made in this laboratory at cold and warm periods of the year which indicate that the total daily output of urine may decrease as much as 48 percent during hot weather in spite of increased water consumption at that time. Unless the intake and output of water remain essentially the same for adults and children during hot weather it is unlikely that the concentration values will remain equal for the two groups.

It is of interest to note some of the references found in the literature dealing with common sources that may be responsible for the lead and/or arsenic found in nearly all of the specimens analyzed. Some, although not all, foods have been found to contain measurable amounts of lead and/or arsenic (*5*, *6*) and the increased arsenic content of some sea-food and related products has been studied (*10*, *11*). The lead content of air and of some types of gasoline (*7*, *8*) and of certain drugs (*9*) has also been investigated. Finally, the lead and/or arsenic content of tobacco has received some attention (*12*, *13*, *14*, *15*).

The eating, smoking, and drinking habits of the 46 persons studied in the present investigation naturally varied widely. About three quarters of the adults are occasional or inveterate users of tobacco. However, substantial agreement of urinary lead and arsenic values for the groups of children and adults appears to indicate that the consumption of tobacco has little effect on the concentration of these elements in the urine. Furthermore, apples do not form a large part of the diet of the individuals concerned so that this dietary item would not be expected to contribute a large share of the lead and arsenic found in the specimens.

Analyses of three sets of drinking water samples were made in an effort to evaluate the potential quantities of lead from this source. Lead was determined by the dithizone method (*1*) on 500 cc. samples. The average values for duplicate analyses are given in table 3. Sample No. 1 was obtained from a drinking fountain used by the laboratory personnel, No. 2 from a private well used by one of the families, and No. 3 from a faucet in a newly built dwelling occupied by one of the families.

TABLE 3.—*Lead content of samples of drinking water*

No.	Mg. of lead per liter
1	0.004
2	.002
3	.033

The quantities of lead ingested in the course of a day from the first two water samples must be regarded as of negligible importance. The highest value found in drinking water (No. 3) was only a third of the United States Public Health Service (Treasury Department) limit of 0.1 mg. of lead per liter for drinking water supplied by interstate carriers (*16*). The possibility of lead in water in houses having new plumbing is recognized (*17*). Careless use of lead compounds used to calk pipe threads may result in leaving comparatively large

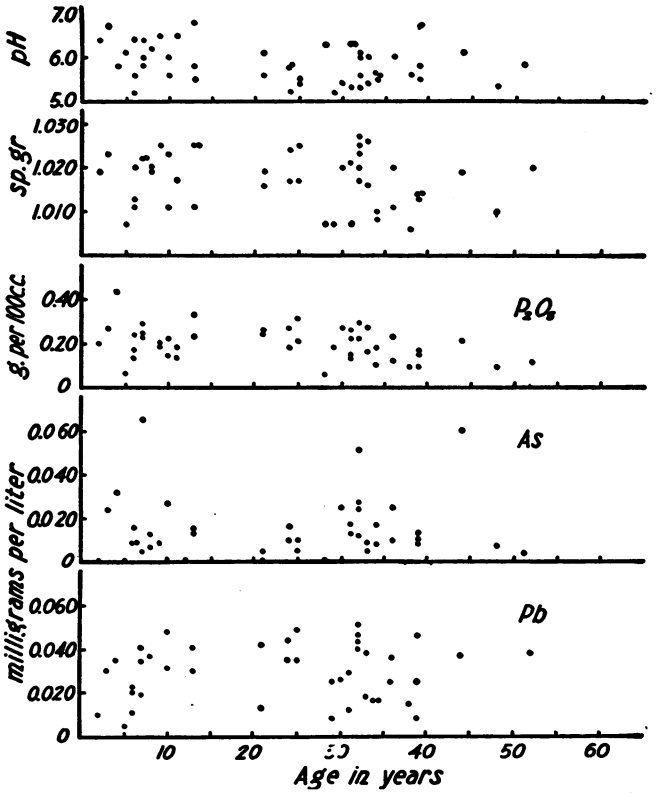

FIGURE 1.—Variation of certain urinary measurements with age of 46 individuals having no known exposure to lead arsenate.

amounts of these substances in the pipes in contact with water. Under these circumstances the water standing in the pipes may contain much more lead than a sample taken after the water has been allowed to flow (*22*). The not infrequent practice of using for drinking or culinary purposes the hot water derived from a domestic tank supply has also received attention.

The average values for urinary lead excretion for the two families for which the water analyses were available were identical within the limits of experimental error, being 0.024 and 0.026 mg. per liter, respectively.

Considering the urine measurements in more detail, the individual

values for the hydrogen ion concentration (expressed in terms of pH), specific gravity, phosphate (calculated as P_2O_5), arsenic (as As), and lead (as Pb) concentrations are given in figure 1, where these measurements are plotted against the age of the individuals concerned. It can be seen that age trends are not prominent. It will be noted also that all of the individuals [2] had an appreciable quantity of lead in the urine and all but four (2 children and 2 adults) had a measurable quantity of urinary arsenic. The comparatively large var-

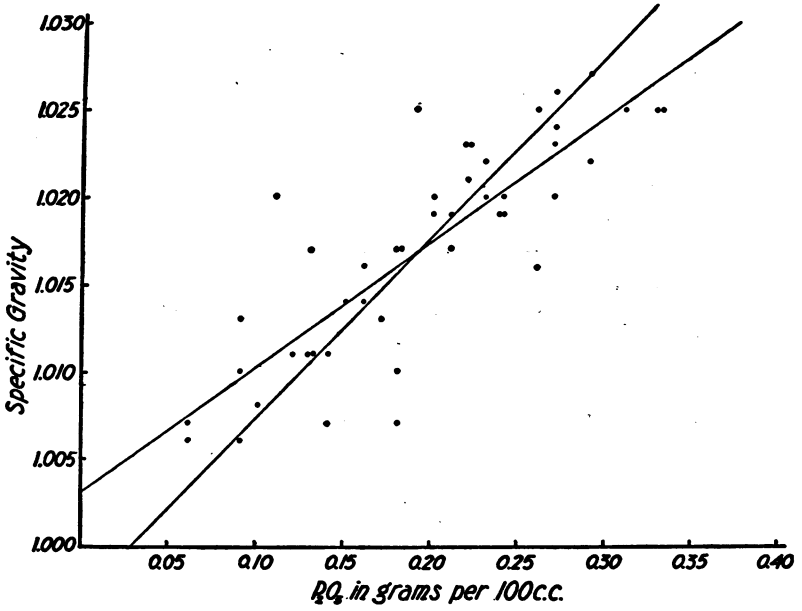

FIGURE 2.—Relation between specific gravity and P_2O_5 concentration of urine (based on urine specimens from 45 individuals).

iation for concentration ranges with lead, arsenic, and phosphorus is likewise of interest.

Scatter diagrams, made by plotting one set of measurements in turn against another set, failed to reveal any close correspondence between the five variables except in one case. Figure 2 shows such a diagram for specific gravity and P_2O_5 values. This is of interest in connection with the known dependence of the specific gravity on the urea and sodium chloride content (*18*). The regression lines shown in the figure are obtained by the method of least squares, using first the specific gravity and then the phosphate concentration as the independent variable. This diagram indicates a marked tendency for the specific gravity to increase as the phosphate content increases.

From the random nature of the scatter diagrams for lead and arsenic concentration values it is probable that the small quantities of lead

[2] Lead and arsenic were determined on 45 and 43 specimens, respectively.

and arsenic found in the urines of nearly all the individuals studied are not derived largely from lead arsenate.

Finally, it is of interest to compare the urinary lead and arsenic values for the groups included in this study with those for men and women among the consumer groups studied in Wenatchee, Wash., (*1*) at comparable times of the year. Of these 98 Wenatchee residents none had occupational or industrial exposure to lead arsenate but all but 6 percent ate apples which had been sprayed with lead arsenate containing material. The average yearly consumption was estimated to be about 300 apples per person, the men consuming about 10 percent more and the women about 10 percent less than this figure. Table 5 shows the average values for urinary lead and arsenic concentrations. It appears that the difference in urinary arsenic excretion for the Bethesda and Wenatchee residents may be explained both by the high consumption of apples by the latter group and also by the rapid elimination of arsenic from the blood stream by way of the kidneys. The earlier rise of the urinary arsenic levels and the relatively small influence of the consumption of apples on the urinary lead levels which have been shown elsewhere (*1*) appear to explain satisfactorily these group differences.

TABLE 5.—*Comparison of average urinary lead and arsenic concentration values for 4 groups of persons with no known industrial or occupational exposure to lead or arsenic*

Location	Group	Urinary lead		Urinary arsenic	
		Number of analyses	Mg./l.	Number of analyses	Mg./l.
Bethesda, Md.	Children	17	0.026	17	0.014
Do.	Adults	28	.030	26	.015
Wenatchee, Wash.	Women	57	.029	55	.041
Do.	Men	41	.035	40	.050

SUMMARY AND CONCLUSIONS

The urinary lead and arsenic concentration values were determined on morning specimens from 46 persons (28 adults and 18 children) having no known exposure to lead arsenate. Urinary phosphate, pH, and specific gravity measurements were also made. A wide range in concentration values and in other determinations was found. The children showed nearly the same range of values as the adults, the averages for the children being slightly less than for the latter group. However, no uniformity was found in the urinary lead or arsenic measurements for different members of a given family. A close correspondence between specific gravity and phosphate concentrations of the urine specimens was shown.

Comparison was made of lead and arsenic values for the 46 non-exposed individuals in this study and for men and women of the pre-

viously studied consumer groups in the lead arsenate spray residue investigation. These figures showed about the same average lead values for the four groups but significantly lower average urinary arsenic values for the groups living outside of the apple growing district.

In this experiment, with a population having no known occupational or industrial exposure to lead or arsenic, no evidence was discovered to indicate that children as a group differ significantly from adults in their urinary lead and arsenic concentration values.

ACKNOWLEDGMENTS

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RAT-BITE FEVER IN WASHINGTON, D. C., DUE TO *SPIRILLUM MINUS* AND *STREPTOBACILLUS MONILIFORMIS*[1]

By CARL L. LARSON, *Assistant Surgeon, United States Public Health Service*

Rat-bite fever has been recognized as a clinical entity for a considerable time, but the causative agent has only recently been discovered. Robertson (*1*) states that the disease was described in the United States as early as 1840 by Wilcox and Watson. The discovery of *Spirocheta morsus muris* was made by Futaki and his associates (*2*) in 1916. Carter (*3*) had applied the term *Spirillum minus* to the organism which he observed in the blood of a naturally infected rat in 1887.

The disease is of world-wide distribution. In 1931, Bayne-Jones (*4*) reviewed the literature and found that there had been 81 cases in 23 States in the United States prior to that time. The first patient in this country from whom *S. minus* was recovered was reported by Shattuck and Theiler (*5*). The number of cases diagnosed on etiologic grounds increased slowly and, in 1932, Francis (*6*) noted that his patient was the sixth one so studied. Leadingham (*7*) had 5 individuals with rat-bite fever under his observation and *S. minus* was detected by animal inoculation in three instances. He states, "* * * the *Spirillum minus* has been identified with a clinical syndrome sufficiently characteristic to permit restriction in the use of the designation 'rat-bite fever' or 'sodoku' to the disease caused by this organism." There have been 19 previous cases in the United States from which the organism has been isolated (*4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16*).

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

Kaneko and Okuda (*17*) studied 2 human cases which came to autopsy. Liver damage was the most marked pathologic finding and spirillae were present in the kidney.

As no practical serological tests are available and *S. minus* has not been cultivated in artificial media, the diagnosis of rat-bite fever depends upon clinical evidence and inoculation of laboratory animals with blood or other materials derived from the patient. Care must be taken in interpreting the results of animal tests as *S. minus* may be present as an inapparent natural infection in the animals. Francis (*18*) pointed this out among mice and das Gupta (*19*) described a natural infection in a guinea pig.

S. minus has not been universally accepted as the cause of rat-bite fever. The controversy regarding this was opened in 1916 when Blake (*20*) published a report of a case from which *S. minus* could not be isolated. He obtained pure cultures of a streptothrix from the patient and pointed out the similarity with Schottmuller's results. At autopsy, Blake's patient presented myocarditis, endocarditis, interstitial hepatitis, and nephritis. It was apparent that, although the condition appeared to be rat-bite fever on clinical grounds, it was characterized by an entirely different etiologic and pathologic picture. Tunnicliff (*21*, *22*) discussed the occurrence of this organism among rats and suggested the name *Streptothrix muris ratti*. Strangeways (*23*) found it in 4 of 6 wild rats and 28 of 46 white rats. Pure cultures of *S. muris ratti* were obtained from 2 human cases by Tileston (*24*). In 1939, Dawson and Hobby (*25*), who had studied 2 patients with rat-bite fever from whom this organism was obtained, cast doubt upon the acceptance of *Spirillum minus* as the etiologic agent of this disease, stating, "Before it can be accepted * * * the spirillum theory requires more critical evaluation than it has yet received."

An outbreak of Haverhill fever (erythema arthriticum epidemicum) which appeared to be a milk-borne infection was investigated by Place and Sutton (*26*). This new clinical entity was characterized by sudden onset, recurrent fever, chills, rash, and arthritis. Parker and Hudson (*27*) studied the organism which was responsible for the outbreak and named it *Haverhillia multiformis* but it appears to be identical with *S. muris ratti*. In addition to these names, the organism has also been termed *Actinomyces muris* and *Streptobacillus moniliformis*.

Allbritten et al. (*28*) in their clinic studied a case of rat-bite fever due to *S. moniliformis*, and after a thorough review of the literature concluded that the disease might be due to either *Spirillum minus* or *H. multiformis* and that the clinical picture varies depending upon the organism involved. When due to the spirillum, a primary chancre, papular or large macular eruption, fever, and rarely arthritis characterize the course of the disease. When the streptobacillus is responsi-

ble, septicemia with arthritis and morbilliform and petechial cutaneous eruptions occur.

In view of the facts which have been brought out, it seems of value to present a series of cases of rat-bite fever which have been observed in Washington, D. C., for they not only include the first record of isolation of *S. minus* from a patient in Washington [2] but also are the only instances of infection with *Streptobacillus* in this locality.

CASE REPORTS

Case 1.—E. G., a colored female infant, aged 3½ months, was bitten on the ring finger of the right hand by a wild rat on July 24, 1940. The wound healed with no difficulty, but 19 days later the finger became red and swollen. By August 16, 1940, the finger showed increased swelling; the forearm was hot and edematous, a number of enlarged nodes appeared in the right axilla, diarrhea developed, and a temperature of 103.6° F. was observed. At this time the child was admitted to the service of Dr. Ong at Children's Hospital, Washington, D. C. Three days later the temperature was 101° F., the axillary lymphadenopathy had disappeared, and the finger was still swollen although no ulcer or break in the skin was apparent. Diarrhea accompanied each bout of fever, and swelling and discoloration of the finger fluctuated with the changes in temperature. At no time did a chancre develop at the site of the rat bite. Following an attack of fever beginning on September 2, the course was one of general improvement. In spite of frequent, careful examinations made with this in mind, no rash was discovered at any time. A significant weight loss occurred during the early part of the illness. The patient was discharged on October 10, 1940. Figure 1 shows the character of the temperature curve of this patient. The clinical diagnosis was rat-bite fever.

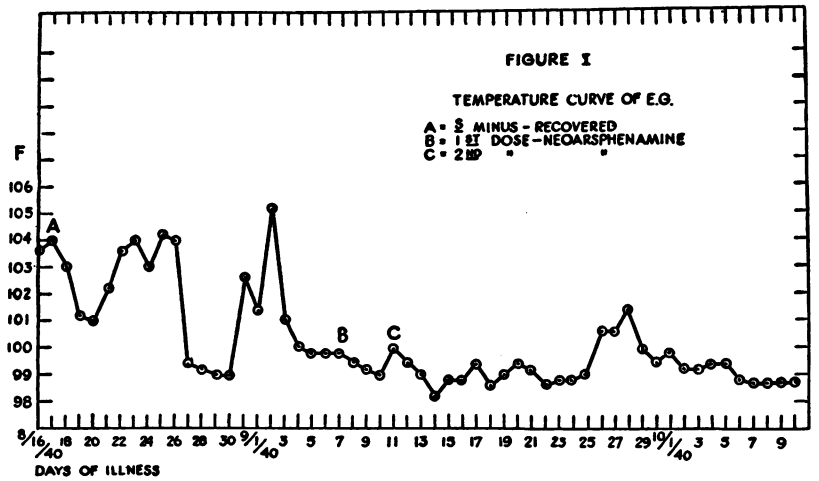
Examinations of the urine failed to reveal anything unusual. On admission blood studies showed hemoglobin 11 gm. per 100 cc., leucocytes 11,000 per cu. mm., and a differential count of 53 percent neutrophiles and 47 percent lymphocytes. On August 31, 1940, there were 7,200 white cells made up of 75 percent neutrophiles and 25 percent lymphocytes, and on September 18, 1940, the leucocytes numbered 6,900. They consisted of 45 percent neutrophiles, 1 percent eosinophiles, and 54 percent lymphocytes. Tuberculin, Schick, and Kahn tests were negative. X-rays failed to reveal evidence of joint damage.

During the early part of the illness, treatment was mainly symptomatic. The local wound was treated conservatively and, with the exception of administration of fluids by clysis, good nursing care was the only treatment employed. When a laboratory diagnosis had

[2] Since this paper was presented, *S. minus* was isolated from another case of rat-bite fever in Washington D. C. (Packchanian, A., and Sweet, L. K.: Rat-bite fever in Washington, D. C. Report of first proved case. Med. Ann. Dist. Columbia, 10:95 (1941)).

been made, neoarsphenamine was administered. This was given in 0.045 mg. and 0.060 mg. amounts on September 7 and 11, 1940, respectively.

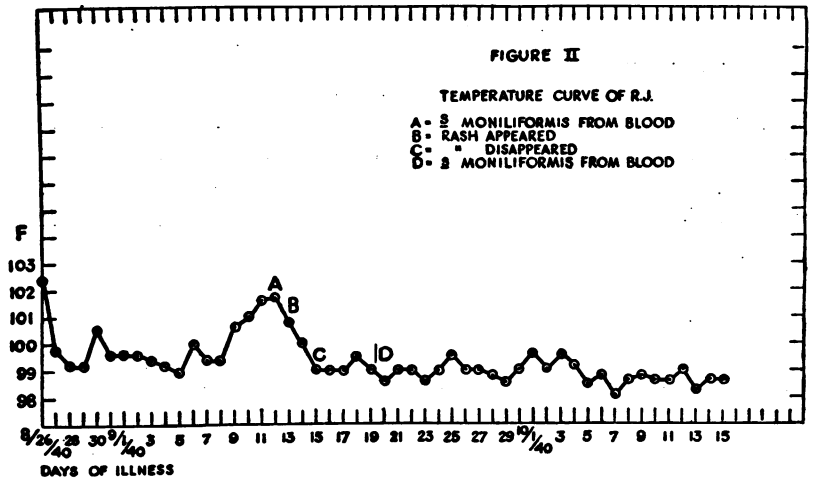
Blood for bacteriological examination was obtained on August 17, 1940. Direct examination using dark-field illumination did not reveal *S. minus*. The blood was injected intraperitoneally into 4 white mice, 2 white rats, and 2 guinea pigs, using 0.25, 1.0, and 3.0 cc., respectively. Blood was also inoculated into veal infusion broth containing 20 percent rabbit serum and incubated at 37° C. No growth resulted. Wet smears of the blood of each of the inoculated animals were observed by dark-field illumination at regular intervals.



Although each animal had been observed seven times before September 7, 1940, no organisms were detected until this date when they were observed in the blood of a single rat. The other rat was found to be infected 3 days later. None of the mice or guinea pigs developed organisms in the blood stream. Blood obtained on September 7, 1940, and subjected to the same procedure as the first sample remained negative. Serum taken on the same date was tested for the presence of antibodies against *Streptobacillus moniliformis* but none were detected. A number of stock rats have been examined in order to determine whether the colony was infected with spirillae. No infected animals were discovered. From our studies, we feel that the infection set up in the two rats injected with the patient's blood was directly due to the injection and that the patient was suffering from rat-bite fever.

Case 2.—R. J., a colored male infant, aged 7 months, was bitten on the right foot by a wild rat on August 26, 1940. He was taken to Children's Hospital on the same day and, as the foot was swollen and a number of puncture wounds were present, the child was admitted to

the service of Dr. Ong. On admission the patient's temperature was 102.6° F. The local wound healed and the child's condition was generally good until September 7, 1940, when he developed diarrhea and vomiting. Two days later a fever of 100.6° F. was noted. The following day, when the temperature was 101° F., the baby appeared very ill, with symptoms of anorexia, vomiting, and diarrhea. Considerable weight loss occurred. Clinically it was considered that this febrile period constituted the onset of rat-bite fever following an incubation period of about 14 days. On September 13, 1940, when the patient was still very ill, having a temperature of 101.6° F., a reddish maculopapular rash was discovered on the hands, arms, and



soles of the feet. The rash disappeared when firm pressure was applied. Two days later the rash had faded considerably, and by September 18, 1940, had become imperceptible and did not again return. During this period the baby was restless and extremely irritable, protesting whenever handled. There was no reaction at the site of inoculation during the febrile period and at no time was evidence of arthritis elicited.

Repeated urinalysis gave no results of note. Blood studies during the course of the illness showed a hemoglobin content ranging from 10 to 11 gm. per 100 cc. and a leucocyte count of 8,700 to 11,700 per cu. mm. The differential was 51–57 percent neutrophiles, 1–3 percent eosinophiles, 0–1 percent monocytes, and 40–45 percent lymphocytes. X-rays of the joints were essentially negative.

Specimens of blood for bacteriological studies were obtained on August 26, September 13, and September 20, 1940. These samples were subjected to the same procedures carried out on those received from the previous case. Although 12 mice, 6 white rats, and 6 guinea pigs were inoculated, *S. minus* was not found in any of the animals.

The first specimen of blood taken on the day of admission was bacteriologically sterile. *S. moniliformis* was obtained from both the other samples which were cultured on veal infusion broth containing 20 percent rabbit serum.

Extreme care was taken with these cultures as the organism may not grow well on original isolation. In the first instance, a few fine white colonies were imbedded in the blood clot which was suspended in the culture medium. They were so unimpressive that they might easily have been overlooked. Subcultures yielded a profuse growth, morphologically resembling *S. moniliformis*. Dr. F. Heilman of the Mayo Clinic concurred in the identification of the culture. The organisms reacted with a specific antiserum to the same titer as did two known strains of *S. moniliformis* kindly sent us by Dr. Heilman.

Case 3.—J. Y., a 21-year-old white male, employed at the National Institute of Health, Washington, D. C., as an animal caretaker, was bitten by a white rat, in the course of his work, on April 19, 1940. The wound bled freely and, after the initial pain, caused him no difficulty. About 10 a. m. on April 22 he complained of chilly sensations and of a severe headache localizing in the occiput and frontal regions. In a short time he became nauseated, vomited, and broke out in profuse perspiration. He vomited several times during the day and night previous to admission to the United States Marine Hospital, Baltimore, Md., on April 23. The patient felt tired, but had difficulty in sleeping. He was admitted to the service of Dr. K. F. Nelson in a toxic condition with a temperature of 38.6° C., pulse rate of 80, and respiratory rate of 16 per minute. There were a few enlarged cervical lymph nodes and the abdomen was tender. The liver was palpable 2 cm. below the costal margin on deep inspiration. It was questionable whether or not the spleen could be felt. The skin and extremities were essentially negative.

Laboratory examination at this time showed the urine to be essentially normal. The blood contained 15.2 gm. hemoglobin per 100 cc., 3,430,000 erythrocytes, and 6,950 leucocytes per cu. mm. The differential count was 16 percent small mononuclears, 4 percent large mononuclears, and 80 percent neutrophiles. Kline and Eagle tests were negative. The blood sedimentation rate was 18 mm. in 1 hour. X-rays of the chest revealed no lesions of significance.

The patient was given symptomatic and supportive treatment and under this therapy the temperature returned to normal on April 25, 1940, and remained so until April 27, when it rose to 38.8° C. He became irrational and developed a definite rash on the wrists, forearms, and ankles. The rash disappeared on pressure. Two days later the patient was subjectively better, the temperature was normal, and the rash had faded in intensity. On May 3 there was an elevation of temperature to 38.4° C. The patient's general condition was good,

but he complained of pain in the right elbow with limitation of movement. This joint was swollen and tender, but not red. He also developed pain in other joints, but this was not intense. The following day he complained of sore throat. The rash had disappeared by that time. The patient felt better on May 7, but was still running a low-grade fever of 37.6° C. Blood studies on this date showed hemoglobin 12.6 gm. per 100 cc., erythrocytes 4,000,000, leucocytes 8,400 with a differential of 33 percent small mononuclears, 1 percent large mononuclears, 3 percent transitional cells, and 63 percent neutrophiles and a sedimentation rate of 25 mm. in 1 hour.

From this time on the patient began a slow and steady convalescence. Pain of increasing and decreasing severity in various joints caused some discomfort. Sore throat of varying intensity also complicated convalescence. He became afebrile on July 1, and remained so for 4 days when he was discharged. At this time the Kline and Eagle tests were positive.

During the course of the illness, agglutination tests of the blood serum by Dr. F. Heilman of the Mayo Clinic and Dr. T. Brown of Johns Hopkins Hospital were carried out. Both reported complete agglutination of *S. moniliformis* at a titer of 1:160 and partial agglutination at 1:320. Two attempts to isolate the organism were unsuccessful. A sample of serum obtained on September 4 agglutinated three strains of *S. moniliformis* to a titer of 1:80. Wassermann and Kahn tests on this serum gave negative results.

This case presented symptoms of intermittent fever, rash, and arthritis. The fact that the wound healed promptly and failed to react during the febrile periods would indicate that *S. minus* was not involved. This, together with the appearance of arthritis, led to a clinical diagnosis of rat-bite fever due to *S. moniliformis* and the laboratory studies bore this out.

Study of the three cases suggests the need for inclusion of methods directly concerned with identification of *S. moniliformis* when considering the laboratory diagnosis of rat-bite fever. Samples of blood should be cultured on veal infusion broth with 20 percent rabbit serum and incubated at 37° C. and serum should be made available for an agglutination test. While the former procedure is simple, the latter one is somewhat difficult for considerable labor is required to procure a suitable stable antigen. We have obtained our antigens by grinding the organisms in a mortar and pestle, or in a ball mill for ½ to 1 hour and suspending the material in normal salt solution containing 0.02 percent formalin. Once a stable emulsion of organisms is prepared, the test is no more formidable than the routine ones commonly employed in most laboratories.

From our cases it is apparent that the clinical diagnosis of rat-bite fever may be rendered difficult by the absence of certain cardinal

features. The absence of a primary chancre at the site of inoculation, failure of a rash to appear, or the presence of an atypical temperature curve may cause hesitation in diagnosis; this can be offset by employing proper laboratory methods for confirmation.

CONCLUSIONS

Three cases of rat-bite fever occurring in Washington, D. C., were diagnosed by isolation of the organism in two instances and by serological evidence in the other. *Spirillum minus* or *Streptobaccillus moniliformis* may be the cause of rat-bite fever in this area.

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DEATHS DURING WEEK ENDED SEPTEMBER 20, 1941

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

	Week ended Sept. 20, 1941	Corresponding week, 1940
Data from 88 large cities of the United States:		
Total deaths	7,264	7,669
Average for 3 prior years	7,563	
Total deaths, first 38 weeks of year	321,301	322,202
Deaths per 1,000 population, first 38 weeks of year, annual rate	11.8	11.8
Deaths under 1 year of age	503	520
Average for 3 prior years	506	
Deaths under 1 year of age, first 38 weeks of year	19,941	19,056
Data from industrial insurance companies:		
Policies in force	64,464,679	64,843,013
Number of death claims	11,023	11,227
Death claims per 1,000 policies in force, annual rate	8.9	9.1
Death claims per 1,000 policies, first 38 weeks of year, annual rate	9.6	9.8

PREVALENCE OF DISEASE

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

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED SEPTEMBER 27, 1941

Summary

A slight decrease was reported in the incidence of poliomyelitis for the country as a whole—592 cases as compared with 596 for the preceding week and 595 for the next earlier week. The East North Central and West South Central areas recorded slight increases, while five geographic areas decreased and two (Middle and South Atlantic) reported the same numbers of cases as last week. The following named 12 States reported 15 or more cases (last week's figures in parentheses): New York, 115 (113)—New York City, 42 (52), State exclusive of New York City, 73 (61); Pennsylvania, 66 (70); Ohio, 42 (34); Tennessee, 39 (24); Alabama, 35 (57); Illinois, 31 (25); New Jersey, 29 (27); Michigan, 26 (20); Georgia, 17 (22); Minnesota, 16 (24); Massachusetts, 15 (20); Maryland, 15 (24). Indiana dropped out of this group during the current week.

For the corresponding week, 711 cases were reported in 1940 and 603 in 1937, in both of which years the peak was reached in the 37th week (second week of September). This year the peak appears to have been reached during the 35th week (last week in August). To date this year (first 39 weeks), 6,393 cases of poliomyelitis have been reported, as compared with 7,724 in 1937 and 6,363 in 1940 for the corresponding period.

The incidence of infectious encephalitis declined in all western States in the area of recent epidemic prevalence.

The number of reported cases of influenza continues higher than the 5-year (1936–40) median and is above that for any corresponding week during the past 5 years. Of 830 cases reported for the current week 310 cases (37 percent) occurred in Texas.

Only 1 case of smallpox was reported (in Michigan). A total of 1,214 cases has been reported to date this year as compared with 2,020 for the corresponding period in 1940, in which year the incidence was the lowest on record.

Four cases of Rocky Mountain spotted fever were reported, 3 in Virginia and 1 in North Carolina. Three cases of tularemia were reported in Utah. Of 93 cases of endemic typhus fever, 36 occurred in Georgia, 27 in Texas, and 9 in Alabama.

The crude death rate for the current week for 88 large cities in the United States is 10.3 per 1,000 population, as compared with 10.1 for the preceding week and a 3-year (1938–40) average of 10.7 for the corresponding week. The accumulative rate to date is 11.8, the same as for the corresponding period last year.

Telegraphic morbidity reports from State health officers for the week ended September 27, 1941, and comparison with corresponding week of 1940 and 5-year median

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

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Sept. 27, 1941	Sept. 28, 1940		Sept. 27, 1941	Sept. 28, 1940		Sept. 27, 1941	Sept. 28, 1940		Sept. 27, 1941	Sept. 28, 1940	
NEW ENG.												
Maine	0	1	1	-------	2	1	18	1	4	1	1	1
New Hampshire	0	0	0	-------	-------	-------	1	0	0	0	0	0
Vermont	0	0	0	-------	-------	-------	3	2	5	0	0	0
Massachusetts	2	2	3	-------	-------	-------	53	64	25	1	1	1
Rhode Island	3	0	0	-------	-------	-------	1	0	0	0	1	0
Connecticut	2	0	1	-------	1	1	8	2	4	1	0	0
MID. ATL.												
New York	6	14	14	-------	[1] 8	[1] 8	48	51	51	5	4	4
New Jersey	1	3	5	3	1	5	27	49	16	1	0	0
Pennsylvania	12	10	16	2	-------	-------	67	82	46	2	4	3
E. NO. CEN.												
Ohio	8	6	27	5	6	6	22	8	20	0	0	1
Indiana	9	8	10	11	4	10	3	5	3	0	0	0
Illinois	8	11	14	3	3	6	18	32	22	0	1	1
Michigan [2]	5	4	11	-------	5	2	38	55	15	0	1	1
Wisconsin	0	2	2	27	33	33	23	70	28	0	0	0
W. NO. CEN.												
Minnesota	2	1	4	2	-------	1	8	4	6	0	0	0
Iowa	0	2	5	-------	-------	-------	6	2	3	0	1	0
Missouri	20	3	4	-------	1	11	3	3	3	1	0	0
North Dakota	2	3	3	-------	-------	5	11	4	2	0	0	0
South Dakota	13	1	1	-------	1	-------	1	0	2	1	0	0
Nebraska	1	1	3	-------	-------	-------	4	19	2	0	1	0
Kansas	1	6	6	1	2	2	8	5	5	0	1	0
SO. ATL.												
Delaware	1	0	0	-------	-------	-------	2	2	1	0	0	0
Maryland [2]	4	3	6	2	-------	3	7	1	3	0	2	3
Dist. of Col	0	2	2	-------	-------	-------	3	2	3	0	0	0
Virginia [2] [4]	16	10	39	41	40	32	28	16	6	3	0	1
West Virginia [2] [3]	0	2	14	-------	7	7	23	2	2	0	1	1
North Carolina [4]	69	46	105	-------	2	2	41	11	11	0	0	0
South Carolina [2]	44	8	23	185	147	147	18	0	0	3	0	0
Georgia [2]	38	18	38	20	11	5	19	11	1	1	0	0
Florida [2]	6	5	10	16	8	3	2	1	1	1	1	1
E. SO. CEN.												
Kentucky	12	14	24	-------	2	3	11	33	12	0	0	2
Tennessee [2]	19	10	34	7	5	13	20	5	4	0	2	1
Alabama [2]	40	16	39	4	2	7	7	16	6	0	1	1
Mississippi [2] [3]	11	10	19	-------	-------	-------	0	-------	-------	0	0	0
W. SO. CEN.												
Arkansas	21	12	20	17	16	5	18	1	1	1	0	0
Louisiana [2]	6	2	14	12	-------	3	0	2	1	1	2	1
Oklahoma	12	9	7	17	11	12	31	1	1	0	0	0
Texas [2] [3]	33	34	34	310	71	71	21	6	13	0	2	2
MOUNTAIN												
Montana	7	0	0	8	-------	-------	14	16	16	0	0	0
Idaho	0	0	0	-------	-------	2	2	1	1	0	0	0
Wyoming	0	0	0	4	-------	-------	0	0	3	0	0	0
Colorado	4	4	5	55	-------	-------	9	6	7	1	0	0
New Mexico	0	3	3	-------	-------	-------	6	1	3	1	0	0
Arizona	1	0	2	36	43	18	29	11	3	0	0	0
Utah [2]	0	0	0	-------	2	2	2	5	2	0	0	0
Nevada	0	-------	-------	-------	-------	-------	0	-------	-------	0	-------	-------
PACIFIC												
Washington	0	4	3	12	-------	-------	4	6	7	0	0	0
Oregon	0	3	3	16	15	11	15	7	8	0	0	0
California [2]	12	14	15	14	19	16	75	47	47	0	1	1
Total	451	307	609	830	468	525	778	668	668	25	28	28
39 weeks [5]	9, 581	10, 350	16, 979	603, 973	172, 013	154, 152	835, 423	232, 468	272, 221	1, 600	1, 319	2, 337

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended September 27, 1941, and comparison with corresponding week of 1940 and 5-year median—Continued

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Sept. 27, 1941	Sept. 28, 1940		Sept. 27, 1941	Sept. 28, 1940		Sept. 27, 1941	Sept. 28, 1940		Sept. 27, 1941	Sept. 28, 1940	
NEW ENG.												
Maine	5	1	1	6	0	4	0	0	0	0	0	2
New Hampshire	0	0	0	1	1	3	0	0	0	0	0	0
Vermont	0	0	0	2	4	4	0	0	0	1	0	0
Massachusetts	15	7	4	68	28	40	0	0	0	1	0	2
Rhode Island	1	0	0	2	3	3	0	0	0	0	0	0
Connecticut	12	1	3	13	17	17	0	0	0	2	0	2
MID. ATL.												
New York	115	21	21	[1] 93	[1] 92	[1] 125	0	0	0	31	16	25
New Jersey	29	3	3	31	38	38	0	0	0	9	5	10
Pennsylvania	66	13	13	53	95	110	0	0	0	16	20	20
E. NO. CEN.												
Ohio	42	46	27	81	93	106	0	0	0	12	12	24
Indiana	10	44	7	7	22	68	0	0	0	2	7	8
Illinois	31	65	65	65	104	107	0	2	1	13	21	29
Michigan [2]	26	72	44	55	62	88	1	1	1	4	5	5
Wisconsin	8	40	8	52	61	68	0	0	0	2	1	2
W. NO. CEN.												
Minnesota	16	25	25	20	38	37	0	1	1	0	4	4
Iowa	0	101	16	26	18	25	0	0	2	0	6	3
Missouri	4	28	2	19	16	25	0	0	0	20	14	14
North Dakota	0	0	0	1	10	10	0	3	3	0	0	1
South Dakota	2	8	1	4	11	8	0	0	0	0	0	2
Nebraska	8	7	3	12	3	12	0	0	0	0	1	1
Kansas	2	45	4	44	28	56	0	0	0	3	5	5
SO. ATL.												
Delaware	4	0	0	10	2	2	0	0	0	0	1	1
Maryland [2]	15	1	2	23	17	17	0	0	0	17	8	8
Dist. of Col	3	1	1	6	11	6	0	0	0	4	1	1
Virginia [3 4]	8	24	3	25	18	23	0	0	0	18	15	19
West Virginia [2 5]	4	64	2	45	20	35	0	0	0	17	19	15
North Carolina [4]	10	2	2	46	81	81	0	0	0	9	6	10
South Carolina [3]	11	0	0	18	6	8	0	0	0	14	8	14
Georgia [3]	17	0	1	26	27	23	0	0	0	4	17	14
Florida [3]	9	2	0	7	2	2	0	0	0	4	4	4
E. SO. CEN.												
Kentucky	6	13	2	47	35	52	0	0	0	14	15	24
Tennessee [3]	39	4	4	43	60	44	0	0	0	12	14	11
Alabama [3]	35	0	1	27	26	26	0	0	1	10	17	6
Mississippi [2 3]	3	1	1	9	14	13	0	0	0	6	3	7
W. SO. CEN.												
Arkansas	1	1	1	1	11	11	0	0	0	4	18	16
Louisiana [3]	4	12	1	1	10	5	0	0	0	5	25	22
Oklahoma	2	8	3	12	18	14	0	0	0	4	12	12
Texas [2 3]	5	3	3	14	20	24	0	0	1	22	48	34
MOUNTAIN												
Montana	0	6	1	7	20	20	0	0	0	1	0	3
Idaho	1	6	1	7	10	9	0	0	0	2	5	4
Wyoming	0	6	1	1	1	3	0	0	0	0	2	1
Colorado	1	0	9	21	11	18	0	0	1	0	1	10
New Mexico	0	2	2	1	0	4	0	1	0	6	6	10
Arizona	0	0	0	1	2	2	0	0	0	0	1	1
Utah [2]	3	0	0	3	4	7	0	0	0	1	1	0
Nevada	0	----	----	0	----	----	0	----	----	0	----	----
PACIFIC												
Washington	4	15	5	21	18	18	0	0	2	4	2	4
Oregon	5	0	3	6	10	10	0	0	1	7	8	4
California [3]	10	13	18	59	72	93	0	1	1	7	9	12
Total	592	711	469	1,142	1,270	1,487	1	9	20	308	383	428
39 weeks [1]	6,393	6,363	4,899	96,661	124,176	144,157	1,214	2,020	8,284	6,472	7,441	10,440

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended September 27, 1941, and comparison with corresponding week of 1940—Continued

Division and State	Whooping cough	
	Week ended	
	Sept. 27, 1941	Sept. 28, 1940
NEW ENG.		
Maine	35	6
New Hampshire	2	0
Vermont	7	9
Massachusetts	109	104
Rhode Island	33	2
Connecticut	31	49
MID. ATL.		
New York	343	224
New Jersey	159	118
Pennsylvania	173	353
E. NO. CEN.		
Ohio	269	160
Indiana	19	21
Illinois	185	111
Michigan [2]	319	243
Wisconsin	265	110
W. NO. CEN.		
Minnesota	70	38
Iowa	21	3
Missouri	18	25
North Dakota	33	26
South Dakota	14	2
Nebraska	24	4
Kansas	50	41
SO. ATL.		
Delaware	3	15
Maryland [2]	36	65
Dist. of Col	24	2
Virginia [3] [4]	45	48
West Virginia [2] [5]	25	37
North Carolina [4]	103	99
SO. ATL.—continued		
South Carolina [3]	94	18
Georgia [3]	10	10
Florida [3]	24	5
E. SO. CEN.		
Kentucky	79	79
Tennessee [3]	20	37
Alabama [3]	14	27
Mississippi [2] [3]	----	----
W. SO. CEN.		
Arkansas	20	6
Louisiana [3]	7	3
Oklahoma	14	14
Texas [2] [3]	93	117
MOUNTAIN		
Montana	3	3
Idaho	6	2
Wyoming	6	1
Colorado	62	13
New Mexico	24	18
Arizona	13	12
Utah [3]	19	20
Nevada	1	----
PACIFIC		
Washington	50	36
Oregon	19	6
California [3]	202	269
Total	3,195	2,611
39 weeks [5]	165,957	122,903

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Typhus fever, week ended Sept. 27, 1941, 93 cases as follows: Virginia, 1; South Carolina, 5; Georgia, 36; Florida, 4; Tennessee, 1; Alabama, 9; Mississippi, 3; Louisiana, 5; Texas, 27; California, 2.
[4] Rocky Mountain spotted fever, week ended Sept. 27, 1941, 4 cases as follows: Virginia, 3; North Carolina, 1.
[5] Figures for West Virginia are for the current week instead of a week earlier as has been the case previously. Figures for the week ended Sept. 20 are as follows: Diphtheria, 4; measles, 9; poliomyelitis, 2; scarlet fever, 24; typhoid fever, 9; whooping cough, 24.

WEEKLY REPORTS FROM CITIES

City reports for week ended September 13, 1941

This table lists the reports from 133 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.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Maine:											
Portland	0	------	0	1	0	1	0	0	0	1	21
New Hampshire:											
Concord	0	------	0	0	0	0	0	0	0	0	6
Manchester	0	------	0	0	1	4	0	0	0	0	26
Nashua	0	------	0	0	0	0	0	0	0	0	7
Vermont:											
Burlington	0	------	0	0	0	0	0	0	0	0	9
Rutland	0	------	0	0	0	0	0	0	0	0	5
Massachusetts:											
Boston	0	------	0	1	2	10	0	11	0	26	180
Fall River	3	------	0	2	1	2	0	0	0	10	25
Springfield	0	------	0	1	0	4	0	0	2	7	32
Worcester	0	------	0	2	2	2	0	0	0	18	31
Rhode Island:											
Pawtucket	0	------	0	0	0	1	0	0	0	0	14
Providence	0	------	0	9	0	3	0	1	0	43	55
Connecticut:											
Bridgeport	0	------	0	1	2	4	0	0	0	5	26
Hartford	0	------	0	0	2	2	0	0	0	0	38
New Haven	0	------	0	2	1	0	0	0	0	13	44
New York:											
Buffalo	0	------	0	1	6	5	0	5	0	8	106
New York	6	------	1	18	23	30	0	58	21	194	1,211
Rochester	0	------	1	0	2	0	0	1	0	3	48
Syracuse	0	------	0	2	2	0	0	0	0	27	47
New Jersey:											
Camden	0	------	0	1	0	1	0	1	0	3	37
Newark	0	1	0	2	0	9	0	5	0	72	82
Trenton	0	------	0	0	1	1	0	1	2	1	32
Pennsylvania:											
Philadelphia	1	2	0	3	15	7	0	21	3	45	415
Pittsburgh	0	------	0	2	10	3	0	2	0	34	145
Reading	0	------	0	0	0	0	0	0	0	2	23
Scranton	0	------	--------	0	------	0	0	------	0	2	--------
Ohio:											
Cincinnati	0	------	0	1	2	12	0	13	0	6	116
Cleveland	1	6	0	1	6	7	0	7	0	60	170
Columbus	0	------	0	2	2	4	0	1	0	4	73
Toledo	0	------	0	0	4	2	0	5	1	24	74
Indiana:											
Anderson	0	------	0	0	1	0	0	1	0	0	3
Fort Wayne	0	------	0	0	1	1	0	1	0	0	25
Indianapolis	0	------	0	0	6	2	0	1	0	3	80
Muncie	0	------	0	0	0	0	0	0	0	2	9
South Bend	0	------	0	1	0	0	0	0	0	0	13
Illinois:											
Alton	0	------	0	0	1	0	0	0	0	0	6
Chicago	6	1	0	4	7	17	0	23	1	120	546
Elgin	0	------	0	0	2	0	0	0	0	2	6
Moline	0	------	0	0	0	0	0	0	0	2	16
Springfield	0	------	0	0	4	0	0	0	0	0	19
Michigan:											
Detroit	1	------	0	3	8	18	0	7	0	105	216
Flint	0	------	0	0	3	0	0	0	0	0	21
Grand Rapids	0	------	0	1	0	1	0	0	0	10	35
Wisconsin:											
Kenosha	0	------	0	0	0	0	0	0	0	0	4
Milwaukee	0	------	0	7	0	0	0	3	0	107	93
Racine	0	------	0	2	0	1	0	0	0	6	7
Superior	0	------	0	1	0	1	0	0	0	10	12
Minnesota:											
Duluth	0	------	0	0	0	0	0	1	0	5	24
Minneapolis	0	------	1	2	0	1	0	3	0	11	102
St. Paul	1	------	0	0	2	3	0	0	0	14	50
Iowa:											
Cedar Rapids	0	------	--------	1	------	0	0	------	0	0	--------
Davenport	0	------	--------	0	------	0	0	------	0	0	--------
Des Moines	0	------	0	0	0	2	0	0	0	0	34
Sioux City	0	------	--------	0	------	1	0	------	0	2	--------
Waterloo	0	------	--------	1	------	1	0	------	0	2	--------

City reports for week ended September 13, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Missouri:											
Kansas City	0	------	0	1	3	1	0	4	0	4	83
St. Joseph	0	------	0	0	0	0	0	0	0	0	18
St. Louis	0	------	0	2	8	1	0	5	1	13	172
North Dakota:											
Fargo	0	------	0	0	0	0	0	0	0	1	3
Grand Forks	0	------	------	0	------	0	0	------	0	1	------
Minot	0	------	0	0	0	1	0	0	0	0	10
South Dakota:											
Aberdeen	0	------	------	0	------	2	0	------	0	4	------
Sioux Falls	0	------	0	0	0	0	0	0	0	0	8
Nebraska:											
Lincoln	0	------	------	1	------	3	0	------	0	1	------
Omaha	0	------	0	1	1	0	0	0	0	3	41
Kansas:											
Lawrence	0	------	0	0	0	0	0	0	0	6	10
Topeka	0	------	0	0	0	2	0	0	0	8	8
Wichita	0	------	0	1	2	0	0	0	0	2	13
Delaware:											
Wilmington	0	------	0	0	0	5	0	0	0	1	23
Maryland:											
Baltimore	0	1	0	6	9	5	0	8	1	31	218
Cumberland	0	------	0	0	0	0	0	0	0	0	9
Frederick	0	------	0	0	0	0	0	0	0	0	4
District of Columbia:											
Washington	1	------	0	0	6	11	0	10	0	24	152
Virginia:											
Lynchburg	1	------	0	0	0	0	0	0	0	0	9
Norfolk	0	------	0	0	0	0	0	0	1	2	34
Richmond	0	------	0	2	2	1	0	0	1	0	51
Roanoke	0	------	0	0	0	0	0	0	1	0	19
West Virginia:											
Charleston	0	------	0	0	1	0	0	0	0	0	23
Huntington	0	------	------	0	------	0	0	------	1	0	------
Wheeling	0	------	0	0	2	0	0	1	0	2	15
North Carolina:											
Gastonia	0	------	------	0	------	0	0	------	0	0	------
Raleigh	1	------	0	0	0	0	0	0	0	8	6
Wilmington	0	------	0	0	1	1	0	0	0	16	14
Winston-Salem	1	------	0	0	2	0	0	1	1	0	19
South Carolina:											
Charleston	0	1	0	0	0	0	0	0	0	0	17
Florence	0	------	------	0	------	0	0	------	0	0	------
Greenville	1	------	0	0	0	1	0	0	0	0	11
Georgia:											
Atlanta	1	------	0	1	0	0	1	4	0	0	62
Brunswick	0	------	0	0	0	0	0	0	0	0	2
Savannah	1	------	0	0	1	1	0	0	0	0	22
Florida:											
Miami	0	2	0	1	0	0	0	1	0	0	34
Tampa	0	------	0	0	1	0	0	0	0	1	25
Kentucky:											
Ashland	0	------	0	1	1	1	0	0	1	1	9
Covington	0	------	0	0	1	1	0	0	0	0	12
Lexington	0	------	0	0	0	1	0	1	0	1	11
Louisville	1	------	0	2	4	2	0	2	1	18	66
Tennessee:											
Knoxville	0	------	0	0	0	0	0	0	0	0	15
Memphis	0	------	0	0	1	2	0	4	0	6	90
Nashville	0	------	0	0	4	0	0	4	0	11	42
Alabama:											
Birmingham	0	1	0	1	1	2	0	2	3	6	51
Mobile	0	------	1	0	0	0	0	2	0	0	29
Montgomery	1	------	------	0	------	0	0	------	0	0	------
Arkansas:											
Fort Smith	0	------	------	0	------	0	0	------	0	0	------
Little Rock	0	2	0	0	1	0	0	1	0	0	22
Louisiana:											
Lake Charles	0	------	0	0	0	0	0	0	0	2	11
New Orleans	3	3	2	0	8	1	0	15	5	0	155
Shreveport	0	------	0	0	0	1	0	2	3	1	36
Oklahoma:											
Oklahoma City	0	------	0	0	3	0	0	1	0	0	50
Tulsa	0	------	0	0	1	0	0	1	1	2	16

City reports for week ended September 13, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Texas:											
Dallas	5	------	0	3	1	1	0	1	1	1	66
Fort Worth	0	------	0	0	2	1	0	0	2	1	27
Galveston	0	------	0	0	0	0	0	0	0	0	13
Houston	3	------	0	0	7	1	0	3	1	0	95
San Antonio	1	1	0	0	2	2	0	3	0	4	68
Montana:											
Billings	0	------	0	0	0	0	0	0	0	0	14
Great Falls	0	------	0	1	1	0	0	0	0	0	8
Helena	0	------	0	0	0	0	0	0	0	2	4
Missoula	0	------	0	0	2	1	0	0	0	5	8
Idaho:											
Boise	0	------	0	0	0	0	0	0	0	0	6
Colorado:											
Colorado Springs	0	------	0	0	0	2	0	0	1	3	6
Denver	7	9	0	1	2	1	0	3	0	41	83
Pueblo	0	------	0	0	1	2	0	0	0	7	11
New Mexico:											
Albuquerque	0	------	0	0	0	0	0	2	0	0	14
Arizona:											
Phoenix	0	11	--------	0	-------	1	0	-------	0	2	--------
Utah:											
Salt Lake City	0	------	0	1	0	1	0	0	0	8	31
Washington:											
Seattle	0	------	0	0	2	1	0	2	0	26	100
Spokane	0	------	0	0	2	5	0	0	1	2	27
Tacoma	0	------	0	0	2	0	0	1	0	4	29
Oregon:											
Portland	1	------	0	1	0	0	0	2	0	2	77
Salem	0	------	--------	0	-------	0	0	-------	0	0	--------
California:											
Los Angeles	6	5	0	6	2	4	0	12	0	32	367
Sacramento	1	------	0	0	4	0	0	2	0	1	29
San Francisco	1	1	0	2	6	4	0	3	0	8	158

State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths	
New Hampshire:			
Nashua	0	0	1
Massachusetts:			
Boston	0	0	7
Springfield	0	0	1
Connecticut:			
Bridgeport	0	0	3
New York:			
Buffalo	0	0	4
New York	2	0	53
Rochester	0	0	6
Syracuse	0	0	2
New Jersey:			
Camden	0	0	1
Newark	1	1	3
Pennsylvania:			
Philadelphia	1	0	15
Pittsburgh	0	0	1
Scranton	0	0	2
Ohio:			
Cleveland	0	0	19
Illinois:			
Chicago	0	0	12
Elgin	0	0	1
Michigan:			
Detroit	0	0	10
Grand Rapids	0	0	3
Minnesota:			
Duluth	0	0	5
Minneapolis	0	0	7
St. Paul	0	0	8
Maryland:			
Baltimore	0	0	5
District of Columbia:			
Washington	0	0	3
Virginia:			
Norfolk	0	0	1
Richmond	0	0	1
North Carolina:			
Gastonia	0	0	1
Georgia:			
Atlanta	0	0	2
Kentucky:			
Lexington	0	0	1
Louisville	0	0	5
Tennessee:			
Knoxville	0	0	1
Nashville	0	0	5
Alabama:			
Birmingham	1	0	2
Montgomery	0	0	1
Texas:			
Houston	0	0	1
Montana:			
Billings	0	0	1
Colorado:			
Colorado Springs	0	0	1
Utah:			
Salt Lake City	0	0	1
Washington:			
Seattle	0	0	3
Oregon:			
Portland	0	0	1
California:			
Los Angeles	0	0	2

Encephalitis, epidemic or lethargic.—Cases: Rochester, 1; Duluth, 1; Minneapolis, 1; St. Paul, 2; Cedar Rapids, 2; Sioux City, 2; Omaha, 1; Billings, 1. Deaths: Portland, Me., 1; New York, 2; Newark, 1; Cedar Rapids, 1; Fargo, 1; Mobile, 1; Great Falls, 1.

Pellagra.—Cases: Atlanta, 1; Savannah, 3; Montgomery, 1.

Typhus fever.—Cases: New York, 1; Charleston, S. C., 1; Atlanta, 1; Brunswick, 1; Savannah, 2; Miami, 1; New Orleans, 6; Dallas, 2; Houston, 4. Deaths: Nashville, 1.

Rates (annual basis) per 100,000 population for a group of 88 selected cities (population, 1940, 33,855,510)

Period	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet-fever cases	Smallpox cases	Tuberculosis deaths	Typhoid-fever cases	Whooping-cough cases
		Cases	Deaths							
Week ended Sept. 13, 1941	8.0	5.2	0.9	15.7	30.5	32.2	0.2	39.9	7.4	192.8
Average for week, 1936–40	12.3	6.1	1.9	23.2	43.0	45.8	0.3	49.8	10.4	169.1

PLAGUE INFECTION IN FLEAS AND GROUND SQUIRRELS IN SAN BERNARDINO AND SISKIYOU COUNTIES, CALIF.

Under date of September 17, 1941, Dr. Bertram P. Brown, State Director of Public Health of California, reported plague infection proved in a pool of 21 fleas and in a pool of approximately 500 lice, all from 1 golden mantled ground squirrel submitted to the laboratory on August 20 from San Bernardino National Forest, 1 mile south of the Osito Girls' Camp, San Bernardino County, Calif.

Under the same date Dr. Brown also reported plague infection proved in fleas and ground squirrels submitted to the laboratory from Siskiyou County on August 21 and 22, as follows: In the carcass of 1 squirrel, species unknown, found on a ranch 4 miles east and 1 mile north of Yereka; in 1 ground squirrel, *C. douglasii*, and in a pool of 187 fleas from 9 ground squirrels of the same species taken from property 2 miles north and 2½ miles west of Mount Shasta City; and in another pool of 189 fleas from 5 ground squirrels, *C. douglasii*, from a location ½ mile north of Mount Shasta City.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended August 23, 1941.—During the week ended August 23, 1941, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis	2			1	1	2	1	2	3	12
Chickenpox		7		7	24		9	9	7	63
Diphtheria		2	1	21	3	2			2	31
Dysentery				38						38
Influenza		1			4	5			1	11
Lethargic encephalitis						184	[1] 147	1	1	333
Measles			1	106	97	5	9	6	63	287
Mumps				29	23	7	10	5	5	79
Pneumonia		2			5				5	12
Poliomyelitis		1	56	2	12	120	8	12	5	216
Scarlet fever		4	1	64	54	5	7	3	9	147
Trachoma									1	1
Tuberculosis	3		12	9	42	3		1		70
Typhoid and paratyphoid fever			6	24	3			5	3	41
Whooping cough			6	174	100	7		1	23	311

[1] Encephalomyelitis.

DENMARK

Notifiable diseases—April–June 1941.—During the months of April, May, and June 1941, cases of certain notifiable diseases were reported in Denmark, as follows:

Disease	April	May	June	Disease	April	May	June
Cerebrospinal meningitis	29	23	11	Mumps	376	384	403
Chickenpox	1,650	1,647	1,454	Paratyphoid fever		12	6
Diphtheria	57	26	41	Poliomyelitis	2	3	
Dysentery	24	25	83	Puerperal fever	17	20	10
Epidemic encephalitis	4	2	4	Scarlet fever	472	534	467
Erysipelas	212	174	207	Syphilis	41	42	13
Gastroenteritis, infectious	2,248	2,400	2,646	Tetanus, neonatorum	5	3	1
German measles	5,242	6,020	4,293	Typhoid fever	2	2	
Gonorrhea	541	619	717	Undulant fever	44	39	49
Influenza	22,174	7,696	3,297	Weil's disease	1		1
Measles	3,862	4,313	5,160	Whooping cough	2,487	2,790	3,006

SWEDEN

Notifiable diseases—June 1941.—During the month of June 1941, cases of certain notifiable diseases were reported in Sweden as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	10	Poliomyelitis	17
Diphtheria	5	Scarlet fever	1,249
Dysentery	3	Syphilis	17
Gonorrhea	695	Typhoid fever	1
Paratyphoid fever	4	Undulant fever	6

SWITZERLAND

Communicable diseases—May 1941.—During the month of May 1941, cases of certain communicable diseases were reported in Switzerland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	23	Paratyphoid fever	3
Chickenpox	121	Poliomyelitis	5
Diphtheria and croup	99	Scarlet fever	270
Dysentery	2	Trachoma	1
German measles	311	Tuberculosis	316
Influenza	2	Typhoid fever	7
Measles	388	Undulant fever	9
Mumps	71	Whooping cough	216

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