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DIVISION OF HEALTH

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FROM THE

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Typhoid Fever - 1975

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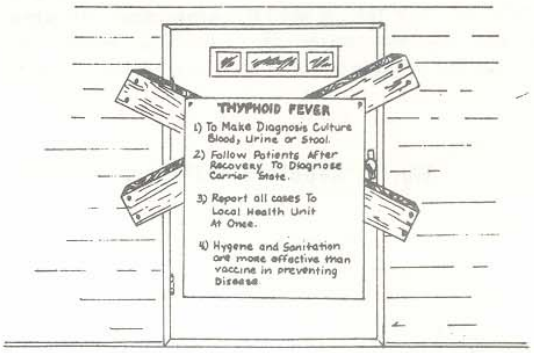

Typhoid fever, although an uncommon disease in Louisiana in recent years, continues to exist in our state and is still a public health concern. This review begins with a presentation of a recent health department investigation of a case and then discusses current thoughts on the diagnosis, treatment, carrier state, and vaccine for typhoid fever.

CASE REPORT

On March 29, 1975, a 10 year old female developed fever of 104°F, headache, nausea, and vomiting. During the next two weeks, despite medication (tetracycline), she continued to be ill with fever, chills, sweats, nausea, vomiting, headache, cough, constipation, and a faint erythematous rash on her abdomen. A stool specimen taken April 14, 1975, grew Salmonella typhi, phage type E_1. Over the ensuing three weeks, treated only with bed rest and fluids, she recovered. Stool specimens taken in June and again in August did not grow Salmonella typhi.

An investigation by the local health unit began April 17, 1975, to determine the source of infection (a standard procedure in this state once a new case of typhoid fever has been identified). The young girl gave no history of known typhoid exposure, of previous typhoid vaccination, or of exposure to poor sanitary conditions. A home visit by a state sanitary inspector substantiated her story. No one in her immediate family reported a current illness or a past history of typhoid fever. Stool specimens from them taken on three separate occasion were negative for Salmonella typhi. The parish health nurse then had cultured all people who prepared food for the patient during the month prior to her illness; to obtain the specimens she visited the child's school and each of the child's relatives who lived in the parish (a total of 60 people were cultured). On May 28, 1975, stools from two relatives were reported positive for S. typhi, phage type E_1. Both relatives were part of the same household and both had helped to prepare a meal for the patient on March 14, 1975, when the patient stayed overnight at their home. Both relatives denied any recent illness. One was an elderly woman, the grandmother of the patient's sister-in-law; the other was a young woman of college age, a granddaughter of the elderly woman. The elderly woman reported that in 1934 a son and a daughter had typhoid fever but that she remained well. The younger carrier said that in her infancy she was hospitalized for one week with a high fever of unknown etiology and that her sister, at age two years, was hospitalized for one month with typhoid fever.

This investigation took six weeks and over 150

stool cultures and innumerable trips around the parish to locate all potential sources. It is to the public health nurse's credit that since June, 1975, the newly discovered carriers have ceased food preparation and that no new cases of typhoid fever have been reported from the parish.

Before World War I typhoid fever was epidemic and devastating throughout the nation; its mortality rate in larger cities ranged from 100 to 180 per 100,000 population and it ranked ninth among causes of death.[1] However, during this century the incidence of the disease in the U.S.A. has been declining, in marked contrast to the trend of other human salmonelloses (See Figure 1).[2] Since 1966 there have been less than 400 cases reported per year in the U.S.A.[3] Louisiana statistics reflect a similar decline (See Table 1); since 1968 we have had no more than 10 cases reported in any one year. The control of typhoid fever has succeeded because of improvements in methods of sewage disposal, protection of water supplies, pasteurization of selected foods, and exclusion of typhoid carriers from food handling professions (three to five percent of all people infected with the organism continue to carry it after recovery).[2,4]

Epidemics in the U.S.A. can be predicted to occur on occasion due to the presence of carriers, "mistakes in sanitation," and existence in developing nations of infectious pools.[1,5] In Louisiana the last large epidemic occurred in 1967 when fourteen cases were reported in a large family residing in northwestern Louisiana; the source was never found. Thirty-one cases resulted in 1960 from the eating of chicken salad sandwiches at a wedding reception prepared by a carrier.[6] Most cases on file in our state since 1960 can be traced to a carrier preparing food. Notable exceptions occurred in 1963 when four cases were associated with swimming in water polluted by a broken sewer line and in 1974 when a laboratory employee accidentally contaminated herself from a culture plate of the organism.

S. typhi (known in years past as Bacillus typhosus, Ebertrella typhosa, and Salmonella

Fig. 1. Reported incidence of human salmonellosis in the United States.

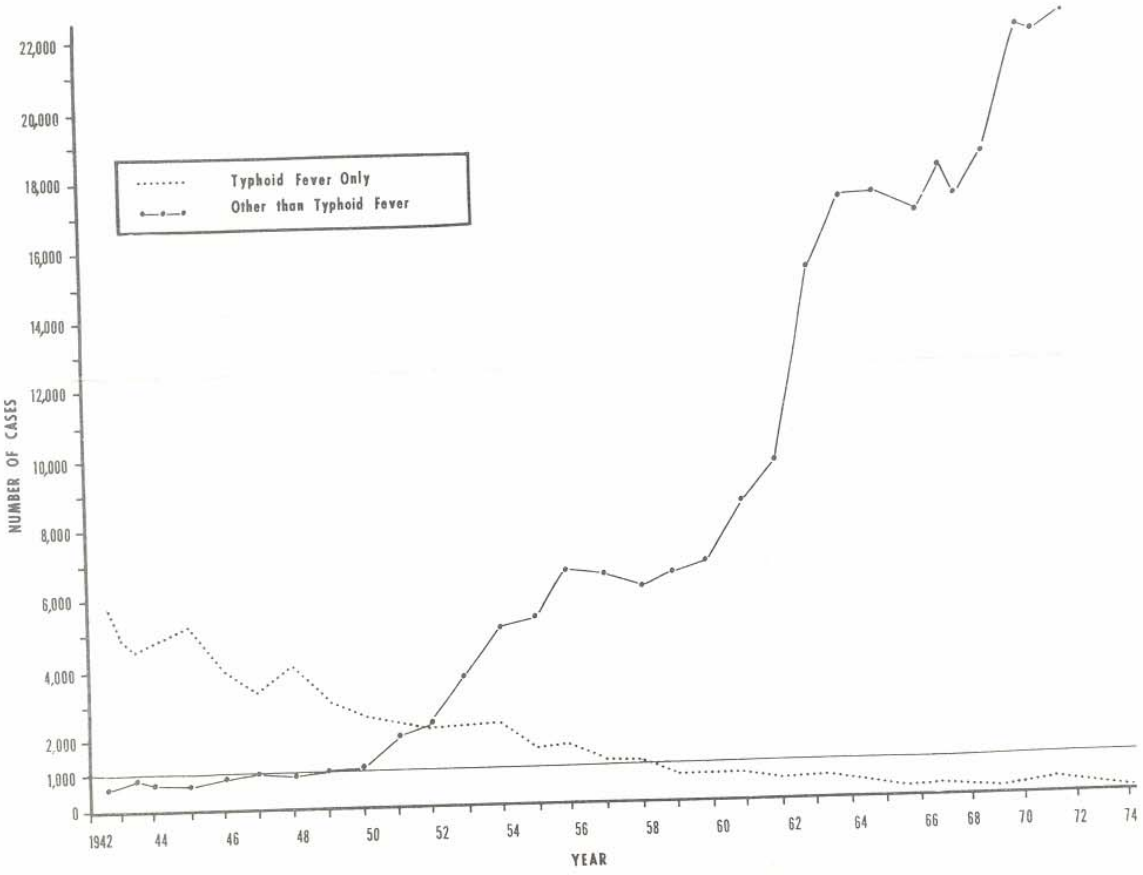

Table 1: Reported Cases and Carriers of Typhoid Fever in Louisiana for Selected Years

Year	Cases	Carriers *
1941	410	IU **
1945	168	IU
1950	134	IU
1955	94	142
1960	58	158
1965	12	147
1970	9	124
1971	6	IU
1972	7	IU
1973	6	IU
1974	10	120

* Number of carriers represents cummulative figures for year specified.
** Information unavailable.

typhosa)[1] is a motile, short, gram negative rod which is destroyed by heat or exposure to sunlight. It does withstand freezing or some degree of drying and can remain alive for months in soil, water, or feces. Man is its sole host, though it can be transmitted by a variety of vehicles (e.g. canned foods, shellfish, water . . .).[1,7]

S. typhi almost always enters the body through the mouth and rapidly gains access to the blood through lymphatics in the bowel. The bacteremia is quickly terminated as organisms are sequestered from the blood by the phagocytic reticuloendothelial cells in liver, spleen, bone marrow, and lymph nodes. For the next seven to ten days the organisms multiply intracellularly. Clinical manifestations become evident as the reticuloendothelial cells rupture releasing organisms and endotoxin into the blood. During this phase of bacteremia infection of the G.I. tract re-occurs, leading to multiplication of organisms in the biliary and intestinal tracts. Proliferation of large mononuclear cells derived from the reticuloendothelial tissue is the most prominent feature of the pathology of typhoid fever. Involvement of lymphoid tissue leads to necrosis and ulceration. Occasionally a large vessel is eroded producing fatal hemorrhage or the bowel wall is eroded leading to perforation and peritonitis. Organisms generally disappear from the blood during the second week of illness, about the time antibodies appear, though symptoms usually progress until the end of the third week of illness.

The clinical features begin with a prodromal stage of malaise, headache, and anorexia. Occasionally sore throat, dry mouth, restlessness, abdominal pains, diarrhea, or cough are present. After a gradual onset, a remittent fever appears that progressively worsens. By the second week of illness the patient usually complains of constipation, vague abdominal pain (most commonly right lower quadrant), nonproductive cough, and severe headache.[8] Signs of the illness include lethargy, delirium, evidence of bronchitis, a pulse that is slower than expected for the degree of fever, slight abdominal tenderness, a palpable soft spleen, and blanching maculopapular skin lesions ("rose colored spots") that are two to five millimeters in diameter, located on the upper abdomen or anterior chest, and last only a few days.[8] Patients without complications begin to improve by the third or fourth week. Complications occur in a low percentage of those treated adequately. The most serious one is intestinal perforation, usually in the lower ileum, which develops in about one percent of cases; severe intestinal hemorrhage occurs in about two percent of patients, although positive test for blood in feces can appear in 10 to 20 percent of cases. Other complications include osteomyelitis, thrombophlebitis (especially of the femoral vein), abortion of a pregnancy (especially during the first trimester), cholecystitis, meningitis, alopecia, pneumonia, and localized infection in any organ.[4] A high degree of resistance generally follows recovery.[9] Asymptomatic infection does occur.[9,7] Relapses occur in eight to ten percent of patients not treated with antibiotics and in 15 to 20 percent of those that are treated.[4]

Differential diagnosis must include many systemic diseases as not one symptom or sign is pathognomonic for typhoid fever. The list includes acute bronchitis, brucellosis, disseminated tuberculosis, influenza, malaria, Mycoplasma pneumoniae infection, salmonellosis, shigellosis, and viral pneumonia. Even malignancies such as Hodgkins disease can be confused for typhoid fever.[4]

THE DEFINITIVE DIAGNOSIS IS MADE BY CULTURING. S. typhi in the untreated patient can be isolated from the blood in 80 to 90 percent of patients during the first week of illness and in 50 percent at the end of the third week.[10,11] It can be cultured from the "rose colored spots," bone marrow, or urine; about 25 percent of patients will have positive urine cultures during days 21 through 28 after onset. The highest yield of positive results, however, is

obtained from stool cultures. The organism can be found in feces at any stage of illness, with best results obtained after the first week of illness.[8,10] In patients who have received some prior form of antibiotic therapy but are still febrile and suspected to have typhoid fever, culturing can be helpful and is indicated. A recent study emphasized not only the value of culturing blood, urine, and feces in these patients (40 percent positive yield) but also the strikingly important value of culturing aspirates from rose spots (63 percent yield) and bone marrow (90 percent yield).[12] For the ill traveler returning from an endemic area, this type of culturing may be essential to making the diagnosis, especially if the patient has treated himself with over-the-counter drugs.[12,13]

Once isolated the organism can be phage typed. Phage typing is an epidemiologic tool and is not of therapeutic significance. The technique subdivides the species by phage sensitivities and supplies more definitive information needed to link carriers with cases. Currently there are over 100 internationally recognized S. typhi phage types.[14]

SEROLOGY IS NO LONGER ACCEPTED AS CONFIRMATORY EVIDENCE OF INFECTION IN MOST INSTANCES.[15] Widal's reactions are the names given to the serological tests for typhoid fever. The tests are either tube dilution or slide tests that measure agglutinating antibodies against O, H, and Vi Salmonella typhi antigens. They usually comprise part of the serology performed when the physician orders febrile agglutinins.

The O, or somatic, antigens are prepared from S. typhi cell wall material; the H, or flagellae, antigens from S. typhi flagellae; and the Vi, or virulence factor, antigens from a so-called thermolabile material that surrounds the bacterial cell wall. The problems with S. typhi serological testing are numerous and substantiate without any doubts why this technique should not be used to make the diagnosis of typhoid fever when culturing facilities are available.

PROBLEMS WITH SEROLOGICAL TESTING

(1) Cross Reactivity: S. typhi belongs to the serogroup D of salmonellae species [the 1400+ types of salmonellae are initially separated into serogroups by the agglutinating pattern of their O antigens (See Table 2)]. Because of the similarity of O antigens, salmonellae other than S. typhi belonging to group D can precipitate a rise in the O agglutinins of a Widal test.[16] Moreover, due to the occasional presence of an S. typhi O antigen in another serogroup (e.g., XII is common to some organisms in serogroups A, B, and D) cross reactions may occur with an organism of a different serogroup.[10,15,17] In Louisiana the most common salmonella causing a rise in the typhoid O titer is probably S. enteritidis.

Table 2: Antigenic Formulae of Important Salmonellae from Four Groups

Group	Species	O Antigen	H Antigen
A	Paratyphi A	I, II, XII	a
B	Paratyphi B	I, IV, V, XII	b, 1, 2
	Derby	I, IV, XII	f, g
	Typhimurium	I, IV, V, XII	i, 1, 2, 3
C	Newport	VI, VIII	e, h, 1, 2, 3
	Infantis	VI, VII	c, 1, 5
	Choleraesuis	VI, VII	r, 1, 5
D	Panama	I, VII, IX	l, v, 1, 5
	Typhi	IX, XII	d
	Enteritidis	I, IX, XII	g, m

(2) Non-Specific Anamnestic Response: In individuals who have already been sensitized to the typhoid bacillus by having the infection itself or having been vaccinated with typhoid vaccine in the past, titer rises may occur as a result of the non-specific immune apparatus stimulated by other infectious diseases. Among these diseases, brucellosis and urinary tract infections with Enterobacteriaceae are the most important. Also, totally non-cross-reactive infections such as streptococcal endocarditis may provoke impressive rises.[10]

(3) Laboratory Standardization: Reagents used in Widal's reactions are frequently poorly standardized or test methods may vary from laboratory to laboratory. Moreover, the tube dilution technique is considered less subject to error than the slide test and should be, but frequently is not, used to confirm any positive reactions in the slide test.[15]

(4) Supression of Response: Early treatment of typhoid fever can decrease the serologic response.[15]

(5) Previous Immunization or Infection: High titers are sometimes seen in people with previous typhoid infection or immunization.[15]

(6) Interpretation of the O Titer: The most that can be said about the O titer is that in a previously unvaccinated patient, a four-fold rise in the O titer or a titer greater than 1:80 on a single specimen in the second or third week of illness suggests S. typhi infection or an infection with an antigenically related salmonella species.[15,18] Once elevated, the O titer tends to remain elevated for months.

(7) Interpretation of the H Titer: The H antigens

are distributed among the salmonellae species without regard to somatic groups (See Table 2). This titer, hence, is also very non-specific. Once elevated it remains so indefinitely.[15]

(8) Interpretation of the Vi Titer: The capsular antigen, Vi, is seen only in the S. typhi species; moreover, antibodies against it are not produced following immunization. The Vi titer usually rises late in the illness and disappears upon recovery unless the patient becomes a typhoid carrier. An agglutination titer of 1:5 is usually significant. The Vi test has been used to detect chronic carriers; however, studies have yielded false positives in six to eight percent of those tested and false negatives in 25 to 30 percent of known carriers. Also the test is technically difficult and not suited for performance by most routine hospital laboratories.[10,19]

Paratyphoid serology deserves brief mention to emphasize that the results of its serology are equally non-specific, often "positive", due to cross-reactivity with other salmonellae groups, and never adequate to make a definitive diagnosis. Only culturing should be used to establish this agent as the etiology of an illness.[15]

Since 1948, there has been a specific therapeutic agent, chloramphenicol. Utilizing good supportive care and appropriate dosage of chloramphenicol the mortality rate has been reduced from 15 percent during the mid-1940's to less than two percent, and now most fatalities occur in infants, the elderly, and those suffering from malnutrition or general disability. Chloramphenicol is given orally in doses of 50 mgms. per kg. per day in four divided doses until temperature is normal; thereafter the dose may be reduced to 30 mgms. per kg. per day. Therapy should continue for a total of two weeks realizing that response to treatment is slow, with temperature usually subsiding five days after the beginning of therapy. Chloramphenicol does not alter the three to five percent incidence of chronic carriers after illness.[4]

Alternatives to chloramphenicol exist. Ampicillin can be used but response appears slower and the drug needs to be given parenterally.[4,9] Amoxicillin given orally (100 mgms. per kg. daily in four divided doses for 10 to 14 days) has recently been shown as effective as I.V. ampicillin and chloramphenicol in curing the disease.[20,21]

A recent development in the epidemiology and treatment of typhoid fever has been the emergence of chloramphenicol-resistant strains. The strains have been noticed world-wide since 1972 (most reports have been from Southeast Asia and Mexico) and have been responsible for at least one major epidemic in Mexico during 1972 (for 10,000+ cases, all S. typhi organisms isolated were resistant to chloramphenicol and 97 percent were of the same phage type). Most infections with this strain have eventually cleared with large amounts of ampicillin or trimethoprim-sulfamethoxazole,[1,22-24] but 25 percent have remained carriers of the organism. An R-factor has been shown to be responsible for much of the spread of this resistance pattern.[23,24]

Patients may continue to shed organisms for several weeks or months after therapy. Relapses, especially after treatment with antibiotics, are common. These people are called convalescent carriers. If shedding of organisms continues beyond one year after illness the patient is defined to be a chronic carrier.[25] The health department adds to this definition anyone not presently ill and without a history of typhoid fever during the past year who, on two separate examinations done not less than 48 hours apart, are found to have typhoid bacilli in stool or urine.

In addition to specimens collected during the acute phase of illness for confirmation of diagnosis, each case should be requested to submit for culturing, starting one month after cessation of antibiotic therapy, a series of three stool specimens and three urine specimens once a month for three months and then each three months for the remainder of a 12-month period. Since the bacilli may be intermittently excreted in the carrier, a single negative culture does not rule out carriage of the organism.[10] If during this period the patient submits two consecutive series of specimens that are negative for S. typhi, culturing is discontinued and the patient is removed from state surveillance. If at the end of 12 months, the patient continues to excrete typhoid bacilli, the patient is added to the official state typhoid carrier list and must uphold state health regulations for chronic typhoid carriers.

Restrictions[25] placed on chronic carriers are to protect the community. These measures are explained to the carrier and immediate contacts by local parish health officials and are enforced by the Division of Health. These restrictions are as follows;

(1) That I will not prepare or handle any food to be eaten by others.

(2) That all members of my household will be vaccinated against typhoid fever every three years.

(3) That I will have provided at my home a sanitary method of excreta disposal, approved by the parish health unit, and will dispose of my excreta only by such a method.

(4) That I will wash my hands thoroughly with soap and water after each bowel movement and urination.

(5) That I will not in any way assist with milking or in the handling of milk or milk

utensils.

(6) That before changing my occupation or residence in the state I will notify a parish health official of such anticipated change of occupation or residence.

Once a carrier is identified, the health department will keep a confidential record on the location of the carrier and his/her phage type. A carrier can be released from the above obligations if the following requirements can be demonstrated:[25]

(1) Fecal Carriers. If the person has been determined to be a chronic fecal carrier, he/she shall not be released until four consecutive series of 3 stool specimens (1 specimen per day for 3 days) collected at not less than 3 month intervals are negative for typhoid baccilli. Stool specimens for release of carriers should be fluid and obtained after administration of a saline laxative. Also, in order to insure the authenticity of specimens, two 5-grain capsules of lycopodium spores should be swallowed by the carrier in the presence of the Local Health Officer or his representative, one day previous to starting the collection of each series of specimens. (Refer to Technique for Collection and Shipment of Specimens for Laboratory Examination Manual for instruction on the use of lycopodium.)[26] If a cholecystectomy has been performed for the purpose of curing a fecal carrier, the requirements for release are the same.

(2) Urinary Carriers. If the person has been determined to be a chronic urinary carrier, he/she shall not be released until four consecutive series of 3 urine specimens (1 specimen per day for 3 days) collected at not less than 3 month intervals have been found negative for typhoid bacilli.

(3) Specimens. No specimens for release of typhoid carriers shall be submitted from persons who have received specific drug therapy within the preceding month.

There is no accepted treatment for alleviation of the carrier state.[4] Cholecystectomy has been reported to terminate the chronic enteric carrier state in about 85 percent of cases.[4] Ampicillin in a total dose of 6 grams per day given orally every six hours for six weeks combined with probenecid may cure the problem in carriers without gallstones and with normal gallbladder function as indicated by cholecystogram.[4,27] Penicillin G in doses of 12 million units or more daily combined with probenecid for two weeks has also been reported successful at times.[4,28,29] Chloramphenicol has failed to be effective in carriers.[30] Other treatments, such as kanamycin,[28] and vaccine with antibiotics,[31] have had an occasional success. More recently, treatment with trimethoprim-sulfamethoxazole, giving two tablets daily for three months, has had discouraging results.[32,33]

The currently available acetone-killed typhoid vaccine appears to provide some protection to those exposed; studies conducted by the World Health Organization between 1954 and 1966 showed it was superior to all other available preparations.[7,34,35] Nevertheless, the vaccine was not ideal. In exposure doses of 10^5 organisms (a typical exposure dose in waterborne spread of the disease), only about 67 percent of those vaccinated three to five years earlier were protected. The vaccine consistantly failed to protect those exposed to heavier concentrations (doses that are consistant with food-borne spread). Also, illness in a vaccinated person was not different in morbidity than in an unvaccinated host.[7]

People given vaccination should be told that their best chance of preventing disease is not due to vaccination, but rather to good hygiene and sanitation practice.[36] Vaccine can help prevent disease in immediate contacts of carriers if the carrier limits the dose of the inoculum by the practice of good hygiene. In this regard, both education and vaccine appear indicated for any household with a carrier.

Several potential oral vaccines are currently being evaluated. In Europe, killed bacteria in the form of keratinized tablets are being given in some countries. Studies in America have yet to demonstrate a superior protective value to these vaccines.[7] Oral preparations incorporating an attenuated strain of S. typhi show the greatest promise; however, additional studies are needed.[18,7] Currently no oral preparations are recommended for use in this country.

Routine vaccination is no longer recommended in the United States. Education about good hygiene is one's best defense against typhoid. Selective immunizations are indicated only in (a) prolonged exposure to a known carrier, as would occur with continual household contact, (b) community or institutional outbreaks of typhoid fever, and (c) foreign travel to endemic areas for those residing in rural areas for extended periods.[36] The vaccine is not indicated for children attending summer camps or for those in areas where flooding has occurred.[37]

Initial vaccination should be (a) for children less than 10 years, 0.25 ml. subcutaneously on two occasions, separated by four weeks or more, and (b) for adults, 0.5 ml. subcutaneously on two occasions, separated by four weeks or more. Booster doses should be given only when exposure is continued or repeated and then only every three

years. A single booster injection of 0.5 ml. is sufficient.[36]

The vaccine is contraindicated during acute severe illness, in patients receiving steroids and in those people exposed to infectious disease for which no satisfactory antimicrobial therapy is available.

Adverse reactions are common, especially inflammation about the injection site 6-24 hours after injection. Systemic reactions include fever, malaise, headaches, and nausea. Post vaccinal neurological disorders have been reported but are very rare. Most reactions subside dramatically 2-3 days after injection.

It is inappropriate to utilize available resources during times of disaster to administer typhoid vaccine.[37] The arguments against typhoid immunization during disasters include:

(1) Evidence of typhoid fever following natural disasters in the United States or any other area of the world in recent decades has been conspicuous by its absence. This is true even in endemic areas.

(2) The limited protection against illness obtainable by vaccine is not realized in individuals previously not vaccinated until after the second injection, given some weeks after initial vaccination. Thus, vaccination in these people will not provide immediate protection.

(3) People who have been vaccinated may develop a false sense of security and neglect fundamental rules of hygiene.

(4) The administration of vaccine requires concerted efforts of many individuals, whose services during a disaster might be allocated to more productive efforts.

(5) It is inappropriate to subject a large population to the considerable temporary morbidity associated with vaccination during a period of considerable stress, especially inappropriate because the immunization is of suspect value.

This review has been offered as an up-date on the current status of typhoid fever. It is hoped that all physicians will continue to report typhoid fever cases promptly to their local parish health unit.

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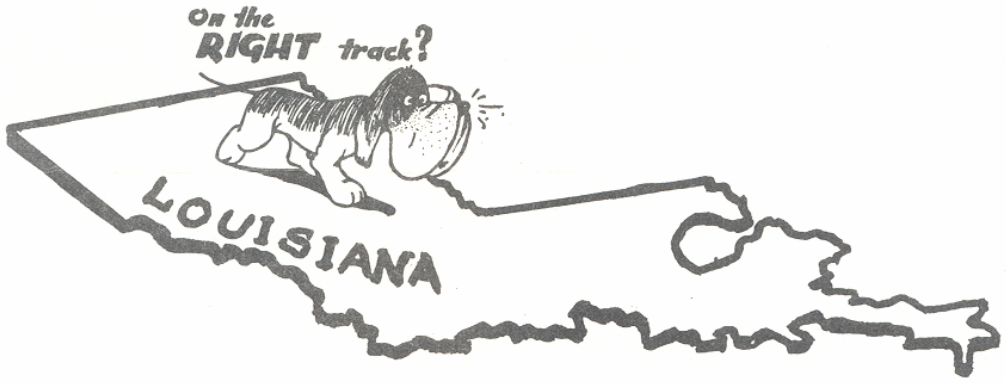

SELECTED REPORTABLE DISEASES

(By Place of Residence)

STATE AND PARISH TOTALS REPORTED MORBIDITY SEPTEMBER, 1975	ASEPTIC MENINGITIS	DIPHTHERIA	ENCEPHALITIS	ENCEPHALITIS, POST INFECTIOUS	HEPATITIS A AND UNSPECIFIED	HEPATITIS B	TUBERCULOSIS, PULMONARY	MENINGOCOCCAL INFECTIONS	PERTUSSIS	RABIES IN ANIMALS	RUBELLA*	SEVERE UNDERNUTRITION	SHIGELLOSIS	TYPHOID FEVER	OTHER SALMONELLOSIS	TETANUS	MEASLES	GONORRHEA	SYPHILIS, PRIMARY AND SECONDARY
TOTAL TO DATE 19 74	114	0	14	5	446	138	443	36	17	18	82	18	129	9	177	3	12	18806	477
TOTAL TO DATE 19 75	120	0	24	12	427	148	380	32	51	5	286	12	111	4	181	4	1	16996	393
TOTAL THIS MONTH	3	0	1	2	57	24	36	2	12	2	0	0	16	0	71	0	0	2102	58
ACADIA					3		2											11	
ALLEN																		6	
ASCENSION					1													2	1
ASSUMPTION																		2	
AVOYELLES																		13	
BEAUREGARD															2			2	
BIENVILLE															1			2	
BOSSIER							1			1								12	
CADDO					2	1	2	1					2		1			163	7
CALCASIEU					1	1	4		3				1		2			105	1
CALDWELL																			
CAMERON															2			3	
CATAHOULA																		2	
CLAIBORNE					1										1			4	
CONCORDIA																		13	
DESOTO																		10	
EAST BATON ROUGE								1							3			146	5
EAST CARROLL					1													19	
EAST FELICIANA																		2	
EVANGELINE																		3	1
FRANKLIN																		3	
GRANT					1										1			3	
IBERIA							1											9	
IBERVILLE																		9	1
JACKSON																		2	
JEFFERSON					7	4	2		1						2			80	3
JEFFERSON DAVIS																		15	
LAFAYETTE					1		2								2			36	
LAFOURCHE					1	2												19	
LASALLE					1														
LINCOLN							1											13	
LIVINGSTON																		2	
MADISON					2													10	
MOREHOUSE							1											27	
NATCHITOCHES															1			27	
ORLEANS	3				7	9	9		5				8		8			733	25
OUACHITA							1								1			97	5
PLAQUEMINES					2	1												4	
POINTE COUPEE																		2	1
RAPIDES					1		4								22			130	1
RED RIVER										1								4	
RICHLAND																		14	
SABINE																		6	
ST. BERNARD			1			2	1											10	
ST. CHARLES							1											17	
ST. HELENA																		15	
ST. JAMES																		3	
ST. JOHN									1									11	2
ST. LANDRY					4	3	1											47	1
ST. MARTIN					2		2								2			4	
ST. MARY					1	1	1		2									16	1
ST. TAMMANY					5										1			17	
TANGIPAHOA					1													19	2
TENSAS																		2	
TERREBONNE					1													2	1
UNION																		6	
VERMILION					4													5	
VERNON					2								5		18			80	
WASHINGTON				2														10	
WEBSTER					1										1			14	
WEST BATON ROUGE																		12	
WEST CARROLL					4													7	
WEST FELICIANA																		30	
WINN																		7	
OUT OF STATE																		3	

* Includes Rubella, Congenital Syndrome

From January 1 through September 30, 1975, the following cases were also reported: 4-Brucellosis; 1-Malaria (contracted outside the U.S.A.); 1-Rocky Mountain Spotted Fever