Q Fever
Q Fever Survey in Southwest Texas *

Texas State Health Department, Austin, Tex.,
AND
City Health Department, San Antonio, Tex.

AFTER the occurrence of the March, 1946, outbreak of Q fever at Amarillo, Tex., the Texas State Health Department continued to search, particularly by laboratory procedures, for other cases. Because of the relative scarcity of ticks and apparently negative findings on cattle sera in this area it seemed probable that more promising areas for investigation of Q fever in Texas could be found. Coxiella burnetii had been demonstrated in pools of ticks collected in southeast Texas in 1937 and later at Camp Bullis near San Antonio. Evidence of this infection in packinghouse workers at Fort Worth has also been reported.

In February, 1948, a survey of packinghouse workers at San Antonio, Tex., was begun. It was planned to search for cases of Q fever in several communities in southwest Texas. A search of raw milk samples for C. burnetii, the causative agent of Q fever, was also begun.

In much of southwest Texas, the people are to a considerable extent of Latin-American descent, and the Negro population is low. Ranching and diverse farming activities provide occupations for many, and toward the Rio Grande Valley the climate is subtropical and suitable for winter gardening. In the lower Rio Grande Valley citrus orchards are abundant.

Methods of Investigation—Through the cooperation of several local physicians the names of persons who had undergone recent febrile attacks were obtained. Cases of "virus pneumonia" especially were sought. Blood samples were taken by local physicians, or persons were visited, interviewed, and blood samples were obtained by us. Upper respiratory infection constituted the main group of illnesses to be differentiated from Q fever in the spring, while typhus more often was considered in the summer.

The complement-fixation test was of great help in the recognition of Q fever. For the most part the complement-fixation antigen was prepared from the Italian (Henzerling) strain of C. burnetii, but the Nine Mile antigen was also utilized on some occasions. Survey bloods from slaughter-house workers and food handlers, as well as milk samples, were obtained in connection with routine tests. All sera were examined in a "screen" test, with a single tube at a 1:10 or 1:20 dilution. All positive tests were repeated quantitatively. The titer was expressed as the highest dilution at which 3 or 4 fixation was seen. Blood clots and milk samples to be

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tested for *C. burnetii* were stored in duplicate in the frozen state, so that all positive tests could be repeated. Mice, hamsters, or guinea pigs were used for isolation of *C. burnetii*.

**Investigation of a Small Outbreak of Q fever in Packinghouse Workers at San Antonio**—Almost immediately as a result of the survey in packinghouse workers at San Antonio, a serum showing a very high titer for Q fever was encountered. Upon inquiry the recent occurrence of "virus pneumonia" in workers on the killing floor of one of the major packinghouses at San Antonio was revealed. Three cases, all showing unusually high Q fever titers readily were located. (Cases 3, 4, and 6 in Tables 1 and 3). Case 3 was the general foreman on the killing floor and had become ill December 17. Case 4 was supervisor of the tankage department and had become ill December 18. Case 6 worked in the beef cooler and had become ill December 25. Other members of their families had not been ill. Since the dates of onset were grouped closely it appeared probable that there had been a common source of exposure, and that some of the livestock butchered had been the source. In view of reports on the incubation period in Q fever it appeared that exposures were in late November or early December, 1947. Approximately 80 persons were employed on the killing floor and conceivably a somewhat larger number could have been exposed. During the probable exposure time there had been days when several butchers were absent. None of those ill in December could recall removing ticks from themselves. Hogs, sheep, goats, and cattle had been butchered in late November and early December. Sheep and goat carcasses also passed through the "beef cooler." Since hogs are not handled in the "beef cooler" they were probably not involved. The subsequent serologic survey revealed only one more butcher (case 5 in Tables 1 and 3) who evidently was exposed and became ill December 23. Perhaps others with low titers, as shown in Table 2, were exposed in late November or early December, and had inapparent infections in December, 1947. The low attack rate suggested that the source of infection was not widely disseminated, or perhaps that some workers had been exposed in previous years.

**Serologic Survey of Q Fever in Slaughter-house Workers at San An-

### Table 1

**Epidemiological and Clinical Findings With Twelve Cases of Q Fever**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Occupation</th>
<th>Date of Onset of Illness</th>
<th>Duration of Fever (Days)</th>
<th>Symptoms at Onset</th>
<th>Cough</th>
<th>Expectoration</th>
<th>X-ray</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>Butcher</td>
<td>11-10-47</td>
<td>10</td>
<td>Headache, malaise</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>Butcher</td>
<td>11-24-47</td>
<td>4</td>
<td>Headache, nausea</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>40</td>
<td>Butcher</td>
<td>12-17-47</td>
<td>10</td>
<td>Malaise</td>
<td>++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>35</td>
<td>Butcher</td>
<td>12-18-47</td>
<td>8</td>
<td>Headache, malaise</td>
<td>++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>Butcher</td>
<td>12-23-47</td>
<td>3</td>
<td>Malaise</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>19</td>
<td>Butcher</td>
<td>12-25-47</td>
<td>6</td>
<td>Headache, chills</td>
<td>++</td>
<td></td>
<td>Atypical pneumonia</td>
</tr>
<tr>
<td>7</td>
<td>28</td>
<td>Postal clerk</td>
<td>3-3-48</td>
<td>12</td>
<td>Headache, nausea</td>
<td>++</td>
<td></td>
<td>Atypical pneumonia</td>
</tr>
<tr>
<td>8</td>
<td>78</td>
<td>Rancher</td>
<td>4-1-48</td>
<td>21</td>
<td>Malaise</td>
<td>++</td>
<td>++</td>
<td>Atypical pneumonia</td>
</tr>
<tr>
<td>9</td>
<td>28</td>
<td>Construction worker</td>
<td>5-2-48</td>
<td>5</td>
<td>Headache, malaise</td>
<td>++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>40</td>
<td>Machinist</td>
<td>9-6-48</td>
<td>9</td>
<td>Malaise, nausea</td>
<td>+</td>
<td></td>
<td>Pneumonitis, or atypical pneumonia</td>
</tr>
<tr>
<td>11</td>
<td>19</td>
<td>Laborer</td>
<td>9-20-48</td>
<td>7</td>
<td>Headache, malaise</td>
<td>+</td>
<td></td>
<td>Pneumonitis</td>
</tr>
<tr>
<td>12</td>
<td>31</td>
<td>Housewife *</td>
<td>10-1-48</td>
<td>12</td>
<td>Malaise</td>
<td>+</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* All others were males.
**Q Fever**

**Table 2**

*Q Fever Complement-Fixation Titters With Sera from Packinghouse Workers at San Antonio, Texas*

<table>
<thead>
<tr>
<th>Packinghouse</th>
<th>No. Sera Tested</th>
<th>No. Sera Negative</th>
<th>1:10</th>
<th>1:20</th>
<th>1:40</th>
<th>1:80</th>
<th>1:160</th>
<th>1:320</th>
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<td>1</td>
<td>190</td>
<td>182</td>
<td>5</td>
<td>4</td>
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<td></td>
<td></td>
<td>1*</td>
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<tr>
<td>2</td>
<td>182</td>
<td>172</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>140</td>
<td>140</td>
<td>2</td>
<td>5†</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>96</td>
<td>94</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>94</td>
<td>92</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>88</td>
<td>81</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>79</td>
<td>73</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>71</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>9</td>
<td>62</td>
<td>59</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>10</td>
<td>59</td>
<td>58</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>52</td>
<td>49</td>
<td>1</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>49</td>
<td>46</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Others</td>
<td>86</td>
<td>79</td>
<td></td>
<td></td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Totals</td>
<td>1,256</td>
<td>1,195</td>
<td>19</td>
<td>16</td>
<td>15</td>
<td>4</td>
<td>1</td>
<td>6</td>
</tr>
</tbody>
</table>

* December 17-25, 1947, outbreak
† November, 1947, cases

**Antonio**—The majority of sera were from persons working on or about the killing floors. Practically every slaughter-house in San Antonio was included in the survey. There was a great majority of men in various age groups and mostly with Latin-American names.

Approximately 5 per cent of these sera had a titer of 1:10 or more, and approximately 2.8 per cent had a titer of 1:20 or more, as shown in Table 2.

The individuals with Q fever antibodies mainly were butchers and were scattered among several plants. There was no clear evidence that any particular job on the killing floor was most hazardous. There was some evidence that those few who worked exclusively with hogs were found less often to be reactors. However, it is doubtful that any epidemiological significance could be attached to this observation, because of lack of definite information on workers’ activities in relation to exposures.

In the five packinghouses where the higher titers were found, the men were interviewed and absentee records were reviewed for dates of any illnesses during 1947 or 1948. While the results were impossible to evaluate in some instances, it seemed evident enough that two butchers (cases 1 and 2) employed by a second large packinghouse had Q fever in November, 1947. There seemed to be a good possibility that cases 1 and 2 were exposed during the course of their work, perhaps on the same occasion, late in October or early in November, 1947, when both men assisted with butchering cattle, sheep, goats, and hogs. There was no evidence that any others of the 50 men on the killing floor, or others who conceivably were exposed, had been ill in November, 1947.

**Serologic Survey of Q Fever in Food Handlers at San Antonio**—Five hundred and sixty-five sera from food handlers at San Antonio were examined by the “screen” test for Q fever. This group was similar to the slaughter-house workers’ group except for differences in occupation. The group comprised a great preponderance of males of various age groups mainly with Latin-American names. Five hundred fifty-six of these sera were not reactive in the 1:10 serum dilution. Seven showed 4+ at 1:10 and two at 1:20 serum dilution.

**Recognition of Scattered Cases of Q Fever in Southwest Texas**—Table 1 lists the clinical cases of Q fever which have been found by preliminary investigations in San Antonio and other widely scattered communities in southwest.
Texas. The diagnosis of Q fever in each case was based on clinical and confirmatory laboratory findings. The typical clinical history was the same as that described by others.\textsuperscript{2, 5, 10} The onset was acute with fever, headache, chills or chilly sensations, and body aches or pains. Chest discomfort was common, and cough was frequently complained of but was not a prominent symptom. Physical examination usually revealed little of note. There was no rash. Chest plates frequently revealed an atypical pneumonic process. The leucocyte count tended to be normal or only slightly elevated. The febrile period usually persisted 1 to 2 weeks and convalescence was prolonged especially in older patients. No deaths occurred.

When the patient was seen in the acute phase of illness, blood was obtained both for antibody test and animal inoculation. Another blood specimen was sought late in the illness or after recovery for possible rise in antibody titer. It was believed that diagnosis was most firmly established by demonstration of a marked rise in antibody titer or recovery of \textit{C. burnetii}. Some patients had recovered when the history notes and blood specimens were obtained, so that the criteria for recognition of Q fever rested on typical clinical history and demonstration of complement-fixing antibodies in high titer, as shown in Table 3.

Since the patients were contacted mainly after the acute phase of illness, the isolation of \textit{C. burnetii} was successful only with case 7. This strain appeared to be typical of the Q fever organism. It caused an enlarged spleen in which the characteristic organisms were demonstrable. Mice developed complement-fixing antibodies in high titers, both with antigens prepared from American (Nine Mile) and Italian (Henzerling) strains of \textit{C. burnetii}.

\textbf{Epidemiologic Findings on Scattered Cases of Q Fever in Southwest Texas—From Table 1 it is seen that the age of confirmed clinical cases varied from 19 to 78 years. With the exception of case 12 all were men. However, this is of doubtful significance because of the possibility of poor sampling. The cases occurred from November, 1947, to October, 1948, and were still occurring. It seems likely that Q fever has been present for some time. No seasonal in-}

\begin{table}
\centering
\begin{tabular}{|l|c|c|c|c|c|c|}
\hline
\textbf{Case No.} & \textbf{Date of Onset of Illness} & \textbf{Bleeding} & \textbf{Complement-Fixation Titers} \\
& & \textbf{Dates 1948} & \textbf{1:20} & \textbf{1:40} & \textbf{1:80} & \textbf{1:160} & \textbf{1:320} & \textbf{1:640} \\
\hline
1 & 11-10-47 & 4-5-48 & 4 & 4 & 4 & 4 & 3 & - \\
2 & 11-24-47 & 4-5-48 & 4 & 4 & 4 & 3 & - & - \\
3 & 12-17-47 & 2-25-48 & 4 & 4 & 4 & 4 & 4 & 4 \\
5 & 12-3-47 & 3-19-48 & 4 & 4 & 4 & 4 & - & - \\
6 & 12-25-47 & 2-10-48 & 4 & 4 & 4 & 4 & 4 & 4 \\
7 & 3-3-48 & 3-11-48 * & - & - & - & - & - & - \\
8 & & 3-18-48 & 4 & 4 & 4 & - & - & - \\
9 & & 4-2-48 & 4 & 4 & 4 & 4 & 4 & 4 \\
10 & & 8-30-48 & 4 & 4 & 4 & - & - & - \\
11 & 4-1-48 & 4-24-48 & 4 & 4 & 4 & 4 & 4 & 4 \\
15 & 10-4-48 & 10-7-48 & 4 & 4 & 4 & 4 & 4 & 4 \\
\hline
\end{tabular}
\caption{Laboratory Findings With Twelve Cases of Q Fever in Southwest Texas, 1947-1948}
\end{table}

\* \textit{C. burnetii} was recovered from the clot.
cidence is as yet evident. The cases are listed in Tables 1 and 3 in chronological order.

While cases 1 through 6 were butchers, no common epidemiologic factor in cases 7 through 12 is as yet evident. Three cases claimed they drank only pasteurized milk. It is interesting that case 7 claimed he drank only pasteurized milk, but it was learned that the cafe which served the coffee that he and his co-workers at the local postoffice drank several times daily served raw cream with the coffee. Some worked with livestock while others neither worked with, nor lived very near livestock. Working with goats and sheep rather than cattle seemed to be of probable significance in case 8. The possibility of infection from tick bite as a common factor is difficult to rule out, but, with one exception, none could recall recent tick bite before onset of illness. The location of these cases over a wide area is of particular interest.

Demonstration of Complement-fixing Antibodies in Sera from Goats and Sheep—The possibility that case 8 had acquired infection on the W. C. ranch seemed excellent. This man had been working daily with large numbers of goats and frequently with the much smaller flock of sheep on the W. C. ranch. He could not recall seeing any ticks around the sheep or goats. There was a lone cow and her calf on the ranch. Blood specimens were collected from the cow and calf, from three sheep, and approximately in equal numbers from goats raised on the W. C. ranch and from a few truck loads brought to the ranch the previous fall from two other distant ranches. Sera from the cow and calf were negative. It should be mentioned that results of tests on sera from cattle elsewhere have been difficult to interpret since titers found so far have been consistently very low. One of three sera from sheep and eight of fifteen sera from goats, including bearers of each of the three brands, showed the presence of antibodies. Each of the five sera from goats raised on the W. C. ranch showed a titer, ranging from 1:10 to 1:160.

The Demonstration of C. burnetii in Raw Milk and Its Possible Significance in Epidemiology of Q Fever—Raw milk samples were received on ice from several communities in southwest Texas usually in connection with routine tests according to A.P.H.A. Standard Methods. Duplicate tubes on each sample were stored in a deep freeze box until the result of the "screen" test for Q fever on a pool of samples became known. Six to fifteen (with an average of ten) samples from as many dairies were included in a pool. Routine samples were picked up on delivery routes or collected from bulk milk either at the dairy or the processing plant before pasteurization. Specimens collected by us were taken at the dairy either from individual strippings or from pools of small strings of cows. One ml. of milk was inoculated intraperitoneally in each of four mice, or occasionally 2–5 ml. of milk was inoculated in a hamster or guinea pig. In some few instances small doses of penicillin were given. One or two mice were sacrificed at 10 days and their spleens were saved in a deep freeze box. The remainder were bled at 21 or 30 days for complement-fixation test. Passages were not attempted with 10 day spleens as a rule unless a mouse for 21 or 30 day bleeding was not available or showed a titer. Serial bleedings were attempted on hamsters and guinea pigs. Two of seventy tests on pools gave unequivocal positive results. Likewise, unequivocal results were obtained on one sample each from the components of the two positive pools and involved two widely separated dairies in southwest Texas.

DISCUSSION

A small outbreak of Q fever in butchers employed by one of the larger
San Antonio slaughter-houses was encountered as a result of a survey. This outbreak was explosive and occurred in December, 1947. While it seemed evident that the infections were acquired in the course of work, it was impossible to assess the relative importance of cattle, sheep, and goats as the probable source of infection. Since three of the four men ill showed evidence of pneumonitis or atypical pneumonia, the possibility of infection from inhalation was suggested. The low attack rate suggested that exposure was limited or that some workers were immune. The relatively low attack rate perhaps was more typical of the picture in Australia \(^1\) than the outbreaks at Amarillo, Tex., in March, 1946,\(^1\) and Chicago, Ill., in August, 1946,\(^1\)

Results of a survey in slaughter-house workers at San Antonio suggested that the December outbreak of Q fever perhaps was not unique but merely was the most recent. In another slaughter-house two cases had occurred in November, 1947. It seemed probable that Q fever had been occurring for quite some time, as was found at Fort Worth, Tex.\(^8\) Many of those having antibodies for Q fever lacked a history of illness particularly suggestive of Q fever and it seemed likely that several had undergone mild or inapparent attacks. Many of these butchers have followed this occupation for years and perhaps had Q fever antibodies in past years which in some instances had become negative.\(^5\) The percentage of sera showing antibodies was significantly greater in slaughter-house workers than in food handlers at San Antonio.

The list of cases of Q fever shown in Table 1 is undoubtedly only a very small fraction of the total number of cases which have occurred in southwest Texas. Most of these persons were patients of a few physicians who were especially curious, or the diagnosis was made more or less accidentally. Undoubtedly physicians will become increasingly aware of the possibility of Q fever and will make increasing use of laboratory facilities to aid in the diagnosis of this disease.

Frequent failure to obtain a history of intimate contact with livestock was somewhat confusing, however, many persons perhaps would not recall occasional intimate contacts with livestock. It seems probable that case 8 had acquired the infection through working with goats or sheep rather than cattle. The finding of \(C. \text{burnetii}\) in raw milk confirms the report of Huebner and others,\(^12\) in California, and certainly suggests the possibility of infection through ingestion of contaminated milk. The possibility of survival of \(C. \text{burnetii}\) in improperly pasteurized milk has not as yet been investigated.

**SUMMARY**

1. Six cases of Q fever which occurred among butchers in two slaughter-houses at San Antonio, Tex., in November-December, 1947, were found as a result of a survey.

2. Approximately 5 per cent of slaughter-house workers' sera at San Antonio, Tex., showed complement-fixing antibodies for Q fever. Approximately 1.6 per cent of food handlers' sera at San Antonio showed complement-fixing antibodies.

3. Six cases of Q fever were found in widely scattered communities in southwest Texas.

4. \(C. \text{burnetii}\) was identified in raw milk samples from two dairies in southwest Texas, but the significance of this finding was not determined.

5. Complement-fixing antibodies were found in sera from sheep and goats on a ranch where a case of Q fever occurred.

**REFERENCES**


7. Communication from Dr. R. R. Parker, Hamilton, Mont.


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Frontal Attack on Cerebral Palsy

The first national conference on cerebral palsy was held in New York January 6-11. Attended by 1,500 persons, it explored the medical and social aspects of the ailment and laid the groundwork for a national program similar to those for tuberculosis, infantile paralysis, heart disease, and cancer. Among the facts brought out by the conference are that there are about half a million victims of the crippling disease in the country, and that 75 per cent of them can be rehabilitated and taught to gain the use of their muscles.

The preliminary framework for a National Foundation for Cerebral Palsy has been established through Leonard Goldenson, Vice-President of Paramount Pictures and father of a five year old sufferer from cerebral palsy. Three of the other founders, including Albert Felmet, Jr., President of the New York State Cerebral Palsy Association, are parents of cerebral palsied children.

The program for which the foundation plans to work includes federal aid for adequate special educational training for the handicapped; Congressional appropriations for improved local public health services; expansion of state school health activities and crippled children's services; formation of a national research center to work on causes and management of cerebral palsy; federal, state, and local aid for professional training to combat the affliction; and the expansion of citizen associations in the field.

The foundation has temporary offices in the New York Academy of Medicine Building, 2 East 103rd St., New York City. Arthur Larschan, first Vice-President of the New York Group, is serving as its temporary chairman.