In the group of severe eclampsia there was one set of twins and two babies whose outcome was not recorded. Twenty-four babies in this group were lost and sixteen left the hospital alive, a fetal mortality of 60 per cent. Even more striking is the fetal loss in the cases in which the maternal systolic blood pressure went over 200 mm. of mercury, whether they had any of the other stigmata of severe eclampsia or not. There were twenty-three such cases with fifteen fetal deaths, six live babies and two unrecored, a fetal mortality of over 71 per cent.

**TREATMENT**

In the treatment of these cases, I have not followed slavishly a routine but have been guided by certain principles. Mine consist of four: (1) stopping the convulsions, (2) good nursing care with emphasis on rest, (3) promoting kidney activity, and (4) digitalis. Magnesium sulfate intravenously has been remarkably efficient in stopping convulsions. My initial dose is 20 cc. of a 10 per cent solution. Frequently I have given a second dose of 15 cc. and occasionally a third dose of 15 cc. In a few cases I have used 7½ grains (0.5 Gm.) of sodium amytal intravenously. I prefer the magnesium sulfate to the sodium amytal, because the former wakes the patient up when it stops the convulsions and the latter puts her to sleep. In such an event it is hard then to know whether the patient is comatose or simply drugged. Nevertheless, sodium amytal is a drug to have around, because occasionally one drug will control the convulsions when the other will not.

Under the head of good nursing care comes the avoidance of external stimuli, such as bright light, noises and jarring the bed. The patient should be kept on her side to lessen the chance of aspirating vomitus and other fluids in the mouth. The tongue should be protected during the clonic stage of a convulsion, and the nurse should be prepared to give artificial respiration if it should be necessary. Too much emphasis cannot be placed on rest. For this reason I am opposed to colonic irrigations, gastric lavage and purgatives. These patients are desperately ill and need all the rest they can get.

I feel very much better when the patient is putting out a good quantity of urine. Usually I rely on water or cream of tartar lemonade to promote kidney activity. The best way to give fluids to an eclamptic patient is by the stomach. If the patient is not awake enough to drink, one can slip a nasal tube into the stomach and pour in a pint of fluid every eight hours. When there is anuria or marked oliguria I resort to dextrose intravenously, varying the strength according to whether there is much or little edema present.

Digitalis has a definite place in the treatment of eclampsia. I give half a cat unit dose as soon as possible after I have given the magnesium sulfate or sodium amytal. I have never seen edema of the lungs when digitalis has been given. In this connection the recent report of Ware and Noblin ¹ is especially interesting, as a large part of my series is composed of patients treated at St. Philip’s and Memorial Hospital immediately prior to the time covered in their report. In 1931 and 1932 when they used morphine, bromide and chloral their mortality was 25.53 per cent. In 1933, 1934 and 1935 they used magnesium sulfate, dextrose and digitalis and their mortality dropped to 6.88 per cent. Dr. Ware tells me that in 1935 and 1936 he treated thirty-seven eclamptic patients with only one maternal death. In other words, at the Medical College of Virginia Hospitals I was treating eclampsia with magnesium sulfate, rest and digitalis, with a maternal mortality of 5 per cent. The treatment was then changed to morphone, bromides and chloral and colonic irrigations and the mortality rose to 25 per cent. The treatment was then changed back to practically the same as before and the mortality dropped to practically the same figure.

I have said nothing about delivery or terminating pregnancy, for the reason that I believe it has no place in the treatment of eclampsia. The patients who go into labor should be treated as conservatively as possible, which means in most cases episiotomy and low forceps under local anesthesia. When antepartum eclampsia is relieved and the patient is putting out a good quantity of urine, then comes up the question of terminating pregnancy. Unless one has exceptionally good control of the patient, it is unwise to let the patient leave the hospital undelivered. No eclamptic or recent eclamptic patient should have a general anesthetic.

**SUMMARY**

A series of 129 consecutive cases of eclampsia have been treated with an uncorrected maternal mortality of 4.65 per cent. The guiding principles in the treatment of these cases have been (1) stopping the convulsions with intravenous magnesium sulfate or sodium amytal, (2) the greatest possible amount of rest, (3) promoting kidney activity with fluids by mouth or dextrose solution intravenously, and (4) adequate dosage of digitalis.

116 East Franklin Street.

**ACTIVE IMMUNIZATION OF TUBERCULOUS CHILDREN AGAINST WHOOPING COUGH WITH SAUER’S VACCINE**

MORRIS SIEGEL, M.D.
AND
ESTHER W. GOLDBERGER, M.D.
NEW YORK

Shortly after Sauer’s early reports of his success in the prevention of whooping cough with doses of 80 billion bacilli of an unwashed vaccine prepared from freshly isolated strains of Haemophilus pertussis, it was decided to test the effectiveness of the vaccine at Sea View Hospital under adequate control conditions. At Sea View Hospital tuberculous persons of all ages are admitted from hospitals and homes of New York City and hospitalized for the duration of their illness. In the pediatrics service there are almost 200 patients under 16 years of age with tuberculosis or suspected of having tuberculosis. The average duration of hospitalization for the tuberculous children is about two years. Newly admitted children are kept in the pediatrics admitting ward for several weeks and are then transferred to one of the other wards. For those over 6 years of age there is a separate ward for boys and girls. They intermingle in the hospital school.

For the children under 6 years of age there is one ward for infants, boys under 3 years and girls under 6 years of age, and another ward for boys between the ages of 3 and 6 years. These younger children remain in their respective wards throughout the day. They are in

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¹ J. Ware, H. H., Jr., and Noblin, Frances E.: South. M. J. 20: 153 (Feb.) 1937.

From the Pediatrics Service of Dr. Bela Schick, Sea View Hospital, Staten Island.
intimate contact with one another but do not mingle with children in other wards. Those with positive sputum are separated from the others in each ward. In addition there is a ward for those with bone tuberculosis and a small ward for normal infants vaccinated with BCG. Visitors are allowed daily between 3 and 5 p.m. Children are not permitted to visit the wards.

Since several cases of whooping cough occurred among our younger children in 1931 and in 1934, we felt that there might be another outbreak in two or three years. This would give us the opportunity to test the effectiveness of Sauer's vaccine under known conditions of prolonged and intense exposure during the catarrhal stages of the disease before the diagnosis is made and when the disease is most contagious, and to compare the outcome among vaccinated and non-vaccinated children living under similar conditions of exposure.

Unfortunately it is difficult to obtain comparable conditions in respect to exposure among vaccinated and control children in studies among children in the general population. In such studies it is particularly difficult to evaluate the degree of exposure among children under investigation unless familial exposure occurs. When there is no familial exposure, it is possible to follow up large groups of children for long periods of time without finding a single undoubted exposure. In cases in which extrafamilial exposure is reported, it is often difficult to determine the degree of the exposure or to be certain that the disease was in its communicable phase when contact took place, even though whooping occurred at the time. It is common knowledge that the infectivity of whooping cough diminishes with the duration of the disease and that a paroxysmal cough can be observed long after the disease has ceased to be infectious. Because of this, reports of extrafamilial exposure not only lose much of their significance as tests of the effectiveness of the vaccine but also can be highly misleading.

Since the degree of exposure varies considerably among the children in a study, and since many children never develop recognizable symptoms of whooping cough, it is obviously essential to choose systematically during the periods of vaccination an adequate number of unvaccinated children as controls if the truth is to be learned of the value of vaccines in whooping cough prophylaxis. This need for adequate controls in the study of whooping cough prophylaxis is well illustrated by the reports of from fifteen to twenty years ago. At that time, when there were no controls, protection was reported in from 90 to 100 per cent of the children to whom doses varying from 50 million to "large" doses of 3 1/2 billion bacilli were administered. When controls were used, the vaccine was found to have little or no prophylactic value.

Similarly, no complete prophylactic value was reported by Madsen in 1925 and 1933 in epidemics in the Faroe Islands in which doses of 22 billion bacilli were employed. However, Madsen stressed the fact that the disease was milder and of shorter duration among the vaccinated and that the mortality in the vaccinated group was "one sixteenth of that in the non-vaccinated group." Sauer's reports since 1933 as to the prophylactic value of 80 billion bacilli seem convincing, except for the fact that he does not fully discuss his results in an adequate number of control children. The same criticism holds for the reports by others who have thus far published favorable results with his vaccine. This is unfortunate and detracts from the value of their work. Of significance in this respect is not only the experience of twenty years ago, as already mentioned, but particularly the recently published work of Duull, Shibley, and McClelland. These investigators found that their whooping cough vaccine prepared of recently isolated strains and administered in a dose of 80 billion bacilli had virtually no prophylactic value, for they had almost as many occurrences among their vaccinated children three months or more after vaccination as among their control children. Their impression was that the disease tended to be milder among the vaccinated children.

In our study at Sea View Hospital we limited ourselves to a small group of children who were known to have had no past history of whooping cough. Approximately half of this group were given the vaccine and the remainder were considered as unvaccinated controls. The vaccinations were begun in August 1934. The children received Sauer's authorized commercial vaccine in the recommended dose of 80 billion bacilli administered subcutaneously. The required dose of 8 cc. was given in three successive weeks in divided amounts of 2, 3 and 3 cc. as advised by Sauer. The vaccine was sent to us on the suggestion of Dr. Sauer. It had to be assumed that this vaccine authorized by Sauer fulfilled all the requirements established by him.

In an effort to maintain an approximately equal number of vaccinated children and unvaccinated controls of similar ages in each ward at all times, suitable children were selected for vaccination at varying intervals between August 1934 and April 1936, shown in table 1. About two thirds of the vaccinated children were under 6 years of age; the average age for the entire group at the time of vaccination was 3.1 years. There were eighty-two children designated as controls. Their average age was 2.7 years.

| Table 1.—Time of Vaccination |
|-----------------------------|---------|
| Children                     |
| August 1934                  | 30      |
| March 1935                   | 20      |
| July 1935                    | 20      |
| December 1935                | 14      |
| April 1936                   | 8       |
| Total                        | 101     |


Vaccinations were not given after April 1936, because a
outbreak of whooping cough occurred during the
late spring and early summer. The first case was
diagnosed May 15 in a child, aged 3½ years, who was
admitted May 1. The last case was recognized August
19. The duration of the epidemic period was about
three and a half months. During this period there were
twenty-seven occurrences of whooping cough in an
exposed group of sixty-four children (42.1 per cent),
6 years of age or less. The high attack rate was
expected, for the children were largely runabouts in
close contact with one another during the day and
exposed during the catarrhal stage when the disease is
most communicable. Isolation was instituted in indi-
vidual cases when symptoms suggestive of whooping
cough were noticed. In all, thirty-three children were
isolated. The characteristic whoop and lymphocytosis
developed in twenty-seven children. These were con-
sidered as undoubted cases of whooping cough. Five
other children had lymphocytosis and cough but no
whoop. Since cough plate cultures were not made, the
cases were considered as probable whooping cough.
The remaining child had a cough which was associated
with an exacerbation of his tuberculosis. Since there
was no change in the lymphocytic ratio, he was not
considered as having whooping cough.

<table>
<thead>
<tr>
<th>Table 2—Age Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in Years</td>
</tr>
<tr>
<td>---------------</td>
</tr>
<tr>
<td>0-1</td>
</tr>
<tr>
<td>2-4</td>
</tr>
<tr>
<td>5-9</td>
</tr>
<tr>
<td>10-14</td>
</tr>
<tr>
<td>15-19</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

For purposes of study, a grouping of the sixty-four
exposed children showed that there were sixteen chil-
dren with a past history of whooping cough, twelve
children with a questionable history of whooping cough,
and thirty-six children with no past occurrence of
whooping cough.

Only the thirty-six children who were known to have
had no past history of whooping cough were included in
our study. The remaining twenty-eight children
were excluded from our study because they had had
whooping cough or their past history of whooping
cough was either questionable or unobtainable.

There were seventeen vaccinated and nineteen control
children among the thirty-six exposed children under
consideration. There were fourteen boys and three
girls in the vaccinated group and twelve boys and seven
girls in the control group. The ages varied from 1 to 6
years. The average age for the vaccinated children at
the time of vaccination was 2.8 years. At the onset of
the epidemic the average age was 4.2 years for the
vaccinated and 2.3 years for the nonvaccinated children.
The age distribution in both groups at this time is given
in table 2. The majority of the control children were
less than 3 years of age, while the majority of the
vaccinated children were 3 years of age or older.

The nature of the tuberculous lesion was quite
similar in the two groups. There were eleven vac-
cinated and twelve control children with primary
pulmonary tuberculosis, three vaccinated and two
control children with hilar lymph node tuberculosis, one
vaccinated and two control children with destructive
pulmonary tuberculosis and one child in each group
with tuberculous pleurisy with effusion. In addition,
one control child had miliary tuberculosis, and another
had Still's disease, and one vaccinated child was a
normal infant who had received BCG vaccination about
six months prior to the whooping cough vaccination.

There were five characteristic cases of whooping
cough among the seventeen exposed vaccinated
children (29 per cent) and ten among the nineteen
exposed control children (53 per cent). In all of these
fifteen occurrences a paroxysmal cough, whoop and
lymphocytosis were present.

As already mentioned, five other children in this
group of thirty-six had a lymphocytosis and cough but
no whoop. In the absence of cough plate cultures it
was thought permissible to consider them as probable
cases of whooping cough. They probably had an
abortive form of the disease, which is not at all uncom-
mon. Four of these children were vaccinated (24 per
cent) and one was a control child (5 per cent). If
characteristic and probable cases of whooping cough are
combined, there were nine cases among the seventeen
vaccinated children (53 per cent) and eleven among
the nineteen control children (58 per cent). The
evitable data relative to these twenty cases are shown
in table 3.

The five vaccinated children in whom characteristic
symptoms of whooping cough developed were from 2 to
3+½ years of age at the time of vaccination. Their
average age at the time of onset of whooping cough was
4 years, as compared with 2.5 years in the
control group. The time interval between the com-
pletion of vaccination and the onset of symptoms of
whooping cough was three months in one case, twelve
months in three cases and sixteen months in another
case. The types of tuberculous lesions in these five
cases were primary pulmonary tuberculosis (three
cases), hilar lymph node tuberculosis (one case) and
destructive pulmonary lesion (one case). The attack
of whooping cough seemed to have no apparent ill effect
on the pulmonary tuberculosis in any of these cases.

The ten nonvaccinated control children in whom
characteristic symptoms of whooping cough developed
varied in age from 1 to 4 years; their average age at
the onset of symptoms was 2.5 years. The types of
tuberculous lesions in these ten cases were primary pul-
monary tuberculosis (six cases), destructive pulmonary
lesion (two cases), hilar lymph node involvement
(one case) and miliary tuberculosis with extensive
pulmonary tuberculosis (one case). The attack of
whooping cough seemed to have no apparent ill effect
on the pulmonary tuberculosis in any of these control
cases except for one case (table 3, case 6), in which
the development of whooping cough might have hast-
ened the death of the child.

Among the five children who have been grouped
separately as probably having whooping cough, there
were four vaccinated children and one control child.
Three of the four vaccinated children were less than
2 years old at the time of vaccination. All the vaccina-
tions were given in December 1935, about seven months
before the onset of symptoms.

An accurate evaluation of the severity of whooping
cough in the group of children with characteristic
symptoms is quite difficult. The disease was mild in
most of the children. It was of moderate severity in
one vaccinated child (case 1) and in several control
children (cases 8, 9, 10 and 11). One control child
(case 6) died of miliary tuberculosis and extensive

9. Since the totals are small, the calculation of percentage is only a rough estimate.
pulmonary tuberculosis, which antedated the onset of whooping cough. In view of his extensive tuberculosis it was impossible to determine accurately the severity of the whooping cough or the effect on the tuberculous process.

In two of the children who were vaccinated the disease was very mild (cases 2 and 3). The average duration of the whoop was twenty-one days among the vaccinated children, as compared with thirty-two and a half days among the controls.

The average number of days during which the whoop was considered severe was two and eight-tenths days among the vaccinated as compared with seven and a half days among the controls. The disease, therefore, seemed somewhat milder among the vaccinated children than it did among the controls. The age of the children might have been a factor here, for all the vaccinated children in this group were 3 years of age or older, while eight of the ten control children were less than 3 years of age.

Twenty-eight exposed children were excluded from the study because they had had a past history of whooping cough (sixteen children) or because their past history of whooping cough was doubtful or not obtainable (twelve children). Their ages varied from 1 to 6 years, the average being 4½ years. There were eleven occurrences of typical whooping cough among these twenty-eight children (39 per cent). Five occurrences were among the sixteen children (31 per cent) with a past history of whooping cough. We do not know whether these cases are recurrences, for we are often doubtful of the diagnosis of whooping cough in the past history of our tuberculous children, because the paroxysmal cough of tuberculosis is frequently mistaken for whooping cough. There were six occurrences among the twelve children (50 per cent) in whom the past history of whooping cough was doubtful. The disease was mild in most instances. There were no complications.

Furthermore, there were fourteen vaccinated and twenty-four nonvaccinated control children over 6 years of age in the hospital during the epidemic, but they were not exposed to the disease. There were obviously no occurrences in this group, and they can be omitted from our discussion.

In addition, seventy vaccinated children and thirty-nine nonvaccinated control children had been discharged from the hospital before the onset of the epidemic. We succeeded in locating forty-six vaccinated children and twenty-two nonvaccinated controls. Of these, there were no known exposures to whooping cough and only one occurrence. In a nonvaccinated child whooping cough developed in September 1936, at the age of 3½ years, six months after his discharge from the hospital. Little significance can be attributed to the absence of whooping cough in the discharged vaccinated group of children because there was no known exposure.

### Table 3.—Clinical Data Relative to Twenty Children with Characteristic and Probable Whooping Cough

<table>
<thead>
<tr>
<th>Case</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Type of Pulmonary Tuberculosis</th>
<th>Date of Vaccination</th>
<th>Date of Onset of Symptoms</th>
<th>Interval Between Vaccination and Onset (in Mo.)</th>
<th>Duration of Whoop (in Days)</th>
<th>Days Whoop Was Severe</th>
<th>Total White Count (Probable Cases of Whooping Cough)</th>
<th>Lympho¬cytosis, per Cent</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>C. A.</td>
<td>3</td>
<td>c</td>
<td>Primary</td>
<td>7/25</td>
<td>7/7/36</td>
<td>12</td>
<td>5</td>
<td>41,200</td>
<td>8</td>
<td>Died of tuberculosis</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>V. C.</td>
<td>3½</td>
<td>c</td>
<td>Primary</td>
<td>7/29/36</td>
<td>3</td>
<td>11</td>
<td>0</td>
<td>9,200</td>
<td>88</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>R. H.</td>
<td>4½</td>
<td>c</td>
<td>Hilar</td>
<td>7/30/36</td>
<td>3</td>
<td>50</td>
<td>0</td>
<td>Not examined</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>W. G.</td>
<td>4½</td>
<td>c</td>
<td>Destructive</td>
<td>7/30/36</td>
<td>12</td>
<td>20</td>
<td>0</td>
<td>Not examined</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>B. T.</td>
<td>5½</td>
<td>c</td>
<td>Primary</td>
<td>7/25/36</td>
<td>12</td>
<td>24</td>
<td>0</td>
<td>12,100</td>
<td>47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>J. P.</td>
<td>5½</td>
<td>c</td>
<td>Military</td>
<td>7/26/36</td>
<td>28</td>
<td>5</td>
<td>0</td>
<td>40,400</td>
<td>61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>J. B.</td>
<td>7½</td>
<td>c</td>
<td>Primary</td>
<td>7/26/36</td>
<td>18</td>
<td>4</td>
<td>12,600</td>
<td>61</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>A. F.</td>
<td>3½</td>
<td>c</td>
<td>Hilar</td>
<td>7/28/36</td>
<td>27</td>
<td>11</td>
<td>30,900</td>
<td>78</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>A. G.</td>
<td>7½</td>
<td>c</td>
<td>Primary</td>
<td>7/28/36</td>
<td>27</td>
<td>11</td>
<td>12,000</td>
<td>61</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>M. R.</td>
<td>9½</td>
<td>c</td>
<td>Primary</td>
<td>8/10/36</td>
<td>27</td>
<td>11</td>
<td>30,900</td>
<td>62</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>A. M.</td>
<td>9½</td>
<td>c</td>
<td>Primary</td>
<td>8/11/36</td>
<td>29</td>
<td>3</td>
<td>11,250</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>L. J.</td>
<td>11½</td>
<td>c</td>
<td>Primary</td>
<td>8/11/36</td>
<td>42</td>
<td>2</td>
<td>12,000</td>
<td>61</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>W. G.</td>
<td>6</td>
<td>c</td>
<td>Destructive</td>
<td>8/10/36</td>
<td>26</td>
<td>3</td>
<td>21,600</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>A. F.</td>
<td>4½</td>
<td>c</td>
<td>Destructive</td>
<td>7/25/36</td>
<td>24</td>
<td>2</td>
<td>28,000</td>
<td>56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>C. D.</td>
<td>1½</td>
<td>c</td>
<td>Primary</td>
<td>12/25</td>
<td>7</td>
<td>0</td>
<td>17,400</td>
<td>61</td>
<td>Coughed 14 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>J. C.</td>
<td>1½</td>
<td>c</td>
<td>Hilar</td>
<td>12/25</td>
<td>8</td>
<td>0</td>
<td>17,500</td>
<td>61</td>
<td>Coughed 11 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>E. C.</td>
<td>1½</td>
<td>c</td>
<td>Primary</td>
<td>12/25</td>
<td>7</td>
<td>0</td>
<td>14,400</td>
<td>67</td>
<td>Coughed 7 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>P. C.</td>
<td>1½</td>
<td>c</td>
<td>Primary</td>
<td>12/25</td>
<td>7</td>
<td>0</td>
<td>14,500</td>
<td>64</td>
<td>Duration of cough unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>R. G.</td>
<td>1½</td>
<td>c</td>
<td>Primary</td>
<td>8/23/36</td>
<td>0</td>
<td>0</td>
<td>16,450</td>
<td>50</td>
<td>Duration of cough unknown</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Refers to the age in years at the onset of symptoms; the numbers in parenthesis refer to the age at the time of vaccination.

**Comment**

The relatively high incidence of whooping cough among our vaccinated children exposed to whooping cough in the wards might be due to (1) the presence of active tuberculosis in the vaccinated children, (2) the presence of a particularly virulent micro-organism or (3) the ineffectiveness of the vaccine in whooping cough prophylaxis.

There is at present no evidence to support the view that the presence of tuberculosis per se diminishes the effectiveness of whooping cough vaccines. Since there are no simple, reliable tests of immunity to whooping cough, the relationship of tuberculosis to whooping cough immunization remains obscure. By means of serologic tests it was found that tuberculous children under our observation responded to the whooping cough vaccine much like normal children. The complement fixation test 10 was strongly positive one week after vaccination.

10. The serologic tests in these cases were performed by Dr. Manfred Weichsel of the Department of Bacteriology, New York University Medical College.
after completion of vaccination and remained positive in tests done one month after the last dose of vaccine. These results are comparable to those obtained in normal children and indicate that the vaccine readily stimulated the formation of specific antibodies in our tuberculous children. Furthermore, in our vaccinations against a disease such as diphtheria, the results of which can well be controlled by a simple skin test, we have had no difficulty in immunizing our tuberculous children with diphtheria toxin-antitoxin or with toxoid, as determined by the conversion of a Schick positive reactor to a Schick negative reactor.

Another possibility in explanation of the failure of the vaccine is the presence of an unusually virulent micro-organism during the epidemic. There seems to be no evidence in support of this view. The disease was mild in most of the children. There were no complications and no deaths due to a severe attack of whooping cough.

It seems that the attack rate was high not so much because of the presence of tuberculosis or of an unusually virulent micro-organism but because of the high degree of exposure of incompletely protected children. This is seen in other acute infectious diseases. In measles, for example, Karellitz and Schick 11 pointed out the increased difficulty of protecting children under conditions of intensive exposure and the consequent need for larger doses of immune serum. Under the conditions of exposure that prevailed at the hospital, the authorized commercial vaccine in a dose of 80 billion bacilli was ineffective in preventing the development of symptoms. However, although the vaccine did not prevent the development of symptoms, it seemed to be of some value, because the symptoms were, on the whole, less severe and of shorter duration among the vaccinated children than among the nonvaccinated children used as controls.

In this discussion we have omitted the question of the duration of immunity after vaccination. In our opinion, this problem has not been adequately studied. It is important, because active immunity produced by bacterial vaccine is short lived. It is advised, for example, that typhoid vaccination be repeated every two years. The long lasting immunity that follows recovery from an attack of typhoid or whooping cough does not indicate that an immunity of equal duration will result after vaccination.

SUMMARY

From August 1934 to April 1935, 101 tuberculous children at Sea View Hospital were vaccinated with 80 billion bacilli of Sauer's authorized commercial vaccine.

In a group of seventeen vaccinated and nineteen nonvaccinated tuberculous children intimately exposed in the hospital during the catarrhal stage of whooping cough, characteristic whooping cough developed in five (29 per cent) of the vaccinated children and in ten (53 per cent) of the nonvaccinated controls, while lymphocytosis and cough, but no whoop, occurred in four (24 per cent) of the vaccinated children and in one (5 per cent) of the control children (table 4). There were eight children without symptoms in the vaccinated group (47 per cent) and an equal number in the control group (42 per cent).

CONCLUSION

1. Sauer’s authorized commercial vaccine, in a dose of 80 billion bacilli, did not seem to prevent the development of symptoms in a group of vaccinated tuberculous children exposed to whooping cough at Sea View Hospital.

2. The symptoms seemed, on the whole, to be milder and of shorter duration among the vaccinated children than among the nonvaccinated children used as controls.

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**NURTURING A NATIONAL NEUROSI**

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In this supposedly modern age of psychiatry it is indeed inconsistent and somewhat startling to find that the government of an enlightened country is foisting upon its citizens a state akin to a national neurasthenia. Reference is made to the manner in which the Veterans' Administration disposes of those individuals who come under its jurisdiction with ills that might be included under the heading of psychoneurosis.

We are uninformed whether or not a somewhat comparable situation developed, psychiatrically speaking, after previous campaigns involving this country. There is no reason to believe that such was not the case; but that the neuroses were encouraged and provided with such fertility in which to develop as they were after the World War is unlikely. It is probable that no precedent was set for the psychiatric confusion in which the Veterans' Administration now finds itself. The experience of other governments with this problem should not have been overlooked, for they had had three years of war behind them when the United States became involved, and their physicians had been stimulated thereby to numerous publications on such subjects as "war neuroses," "soldier's heart," "shell fright," and the like (see especially Hurst, 2 Mott 3 and Marr 4 and particularly the references contained in the first two works).

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1. It has been suggested that if men suffering from "shell shock" had been labeled originally "shell fright" and made to wear a badge there would be far fewer of them today. A physician with one of the Scottish regiments has stated that the medical men of this particular regiment did not recognize "shell shock," with the result that the incidence of men presenting themselves with the signs of "shell shock" was reduced to near zero. (Hurst, A. F.: The Psychology of the Special Senses and Their Functional Disorders, London, Oxford University Press, 1928."


