NEUROMYELITIS IN MUMPS

BY

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The occurrence of nervous complications in mumps points to the fact that the mumps virus possesses potential or even primarily—according to Dopter (1910)—neurotropic propensities. The experimental work of Gordon (1927), in which meningitis was produced in monkeys by intracerebral injection of a filtrate of saliva from mumps patients, lends support to this view.

Systematic lumbar puncture in cases of mumps by French physicians has shown that a meningeal reaction in the form of a lymphocytosis is almost constant. Definite meningeal symptoms are much less frequent, Dopter reporting 9.8% of cases with meningeal symptoms out of a total of 1,705. Typical meningitis is even more rare, Roux recording three examples among 274 cases of mumps. Meningo-encephalitis may also occur, and Howard (1919) reports three cases in which the encephalitis occurred without parotitis during a mumps epidemic.

The above complications seem to occur at the height of the attack of mumps or shortly before or after it. The rarest complication, and one which appears to have a more delayed onset, is neuritis, or neuromyelitis. This may be localized or widespread. The third, sixth, seventh, tenth, and twelfth cranial nerves have all been reported affected, the commonest being the seventh—possibly through mechanical causes rather than virus infection in some cases. Paralysis of the serratus magnus has been reported by Harris and Bethell (1938). Lightwood (1946) recorded a case of a flaccid paresis of the right lower limb occurring eight days after the onset of parotitis. While these cases are localized, a quadriplegia may develop, and five cases of this are reviewed by Collens and Rabinowitz (1928), their own case being of a man of 29 who developed a flaccid quadriplegia three weeks after the onset of epidemic parotitis, with complete bilateral facial paralysis, loss of the deep reflexes of the limbs and of superficial reflexes, and loss of position and vibration sense in the extremities. The cerebrospinal fluid was normal. Recovery was complete in four months. The four other cases were similar in the development of a quadriplegia at intervals varying from 7 to 21 days after the onset of parotitis. Position sense was lost in the case reported by Pitres and Marchand, and there was associated paralysis of both sixth nerves and left facial and right hypoglossal weakness in a case reported by Revilliod. Recovery was complete in all these cases, though Ford (1937) mentions fatalities due to paralysis of the diaphragm. The following provides another example of the quadriplegic type.

Case Report

A woman aged 37 developed bilateral parotid mumps on May 29, 1946, but ignored the attack so far as was possible, continuing with her housework. A week later she developed pains in the back between the shoulder-blades, which gradually became more severe until she was confined to bed. Two weeks after the onset of parotitis there was a gradually increasing weakness of arms, legs, and trunk until she was unable to sit or even move in bed. At the same time diplopia occurred, but there was no loss of sphincter control. The pain in the back continued all this time, but she had no headache. A week later, on June 19, the patient was admitted to Jersey General Hospital. On examination at this time she was apyrexial and mentally alert though very depressed. She was unable to move her position in bed; the tongue was dry and furred, and the parotid swellings had subsided. The fundi were normal; there was weakness of the third nerve bilaterally, paralysis of the left sixth nerve, complete paralysis of the right facial nerve, and weakness of the right accessory nerve. Cervical rigidity was marked. There was flaccid weakness of both arms with absent biceps jerks and weak triceps and supinator jerks. Position and vibration sense were impaired but other sensation was normal. Abdominal reflexes were absent. There was a paresis of both legs, greater in the left than in the right, with a positive Kernig sign, absent knee- and ankle-jerks, and absent plantar responses. As in the arms, position and vibration sense were impaired. Lumbar puncture was performed and a clear fluid, under 80 mm. pressure, was withdrawn. The Queckenstedt response was normal. The protein content was

20 mg. per 100 ml., there were 5 cells per c.mm., the W.R. and Kahn test were negative, and there was no change in the Lange test.

The patient was treated in the usual way for polyneuritis, including the injection of 50 mg. aneurine hydrochloride daily. The right facial nerve had fully recovered in seven days and the other affected cranial nerves in fourteen days. In five weeks she was able to walk, and in eight weeks returned home, with absent knee- and ankle-jerks as the only residual signs.

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CONSERVATIVE TREATMENT OF ACUTE PERFORATED PEPTIC ULCER*

BY

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The history of the treatment of acute perforated peptic ulcer shows a gradual trend towards conservatism. Four distinct phases in treatment during the last twenty-five years can be recognized.

Stage 1.—Closure of perforation. Thorough peritoneal toilet. Flushing and mopping out of all exudate and foreign bodies. Multiple drainage-tubes.

Stage 2.—Closure of perforation. Limited cleansing and mopping of peritoneum. Removal of obvious foreign bodies. Single supra-pubic drainage for twenty-four hours.

Stage 3.—Closure of perforation. Aspiration of fluid only if obscuring site of ulcer. No drainage.

Stage 4.—Conservative non-operative treatment.

It is a very short but adventurous step from Stage 3 to Stage 4. For many years I had deliberately made no effort to remove the beer and carrots from the peritoneum because the task seemed hopeless, yet not one case of subphrenic abscess resulted. It was pneumonia and cardio-renal failure which were responsible for the fatalities that did occur.

Three further observations seemed to point the way towards greater conservatism. First, all surgeons must have felt on many occasions that surgical intervention had done more harm than good. It was no uncommon experience to find the perforation already sealed off. Operation then merely breaks down the adhesions and attempts to repeat what Nature has already effected. Secondly, during the course of the last 400 gastrectomies for chronic peptic ulcer we have several times noted the presence of sheets of adhesions which indicated that a spontaneous closure of an acute perforation must have occurred in the past. Finally, I can remember more than one obstinate patient who, although threatened with the serious consequences of refusing operation, has nevertheless survived and remained well.

In spite of these reminders that spontaneous recovery can occur it was not until Bedford-Turner (1945) reported a series of six cases treated conservatively that I had the courage to follow his lead. For twelve months I treated every case of acute perforated ulcer conservatively. There was no selection of favourable cases, but unfortunately the series is small. Taken in conjunction with many similar series all over the country, it did provide encouraging evidence in support of a trial for non-operative treatment.

Technique

The technique of treatment was as Bedford-Turner described:

Intravenous morphine, repeated as often as necessary to give complete relief from pain. Stomach wash-out with large-bore tube if a recent meal has been taken. Intermittent suction half-hourly through a Ryle tube passed via the nose. No drinks allowed for

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thirty-six hours, fluid intake being maintained by intravenous saline (and sulphonamides). Frequent mouth-washes are given.

For the earlier cases Fowler's position was maintained, but Spalding (1946) has shown that this position is not always desirable, and we are now observing the effects of allowing patients to remain in any position they find comfortable.

On admission each patient presented the typical picture of a case of acute perforation. With the exception of Cases 5 and 14 pain has been relieved within one hour, and thereafter no patient complained of pain apart from movement. Deep breathing continued to cause pain under the right costal margin, but it is important to encourage occasional forced respiration to avoid basal congestion. Temperatures were usually sub-normal on admission, frequently rising to between 100° and 104° F. (37.8° and 40° C.) within the next twenty-four hours. A similar rise in the pulse rate to between 110 and 120 occurred in most cases. Thereafter the temperature fell gradually by lysis, becoming normal between the sixth and eighth days. No anxiety was felt in spite of the rise, because the general appearance of the patient remained tranquil. Provided adequate fluid intake was maintained intravenously the tongue became moist and urinary output was satisfactory.

The abdomen remained boardlike for twelve to eighteen hours, after which the muscles gradually relaxed, first on the left side, then over the right iliac fossa, and finally over the right upper abdomen, leaving a slowly diminishing area of guarding and tenderness over the right hypochondrium for six or seven days. Persistent pain in this area is suggestive of the development of a subphrenic abscess, which occurred in Case 10 and required drainage on the tenth day.

Results

Of the 14 cases 11 were discharged symptom-free, but all are too recent for comment on results. Details of the cases will be seen from Table I. All patients were males, twelve having duodenal ulcer, one a gastric ulcer, and one a stomal ulcer following previous gastro-enterostomy.

TABLE I.—Details of Cases

Case	Age	Previous History	Hours Perforated	Hours Since Meal	Remarks	X-ray	Result (July, 1946)	Time Since Perforation
1	62	2 weeks	5	6	Refused operation	D.U.	No symptoms	13 months
2	50	10 years	4	4	Gastrectomy 3 weeks later	D.U.	"	13 "
3	40	20 "	3	3	Gastrectomy 2 weeks later	S.U.	"	13 "
4	25	2 "	2	5	"	D.U.	"	12 "
5	42	2 weeks	7	7	Pneumonia	P.M. : D.U.	Died	"
6	54	15 years	1	2	"	D.U.	No symptoms	8 "
7	36	5 "	1	5	"	D.U.	"	5 "
8	45	20 "	5	7	Subphrenic abscess	D.U.	"	4 "
9	32	17 "	16	3	"	D.U.	"	2 "
10	64	20 "	5	5	"	D.U.	Pain +	1 month
11	42	3 "	6	7	"	"	No symptoms	1 "
12	29	5 "	5	5	"	"	"	1 "
13	50	15 "	4	5	Pneumonia	P.M. : D.U.	Died	2 weeks
14	59	25 "	4	6	Subphrenic abscess; uraemia	P.M. : G.U.	"	1 week

Five cases call for special comment:

Case 3.—This patient was known to have a stomal ulcer following gastro-enterostomy, for which he had refused operation one year previously. He was seen in consultation three hours after acute perforation, and routine conservative treatment was started. He responded satisfactorily, and two weeks later a very extensive five-sixths gastrectomy was performed. Evidence of recent peritonitis was obvious, oedematous filmy adhesions covering the left upper abdomen. He is symptom-free now, and a fractional test meal shows no free acid.

Case 5.—This patient was admitted seven hours after acute perforation. His clinical condition was very poor, marked cyanosis being a most noticeable feature; pulse 90, temperature subnormal, tongue dry, herpes labialis present. He was put on routine treatment, but four hours later his colour remained poor. At this point I lost my nerve and decided to operate. As soon as the spinal anaesthetic became effective his breathing became tranquil and his colour improved. On opening the abdomen much turbid fluid was seen, and an enormous duodenal perforation was found completely sealed off by recent fibrin and by adhesions to the adjacent liver. These adhesions were broken down and the ulcer closed by omental plug. During the next four days his abdomen remained soft and was not tender, but his general condition deteriorated, and he died on the fourth day. Post-mortem examination confirmed the presence of pneumonia, and there was every indication that the peritonitis was clearing satisfactorily.

This case taught us two lessons: (1) Enough morphine must be given to enable the patient to breathe without pain if bronchopneumonia is to be avoided. (2) Even an enormous perforation is capable of spontaneous closure if it is left alone.

Case 8.—This patient was admitted five hours after perforation. He responded satisfactorily for three days, but on the fourth day he complained of increased pain under the right ribs, and x-ray examination suggested a subphrenic abscess, which was drained through the twelfth rib on the tenth day. This delayed his convalescence for two weeks.

Case 13.—I had seen this man one year previously and decided that, owing to severe chronic bronchitis, surgical treatment was not justified. When he was readmitted with an acute perforation conservative treatment seemed indicated.

Case 14.—Post-mortem examination disclosed a large gastric ulcer partly adherent to the pancreas. There was a subphrenic and subhepatic abscess, into which the gastric contents continued to leak. The patient died from uraemia on the fifth day.

Of the three unsuccessful cases operation might have proved successful in Case 14. Delayed operation undoubtedly contributed to failure in Case 5, while in Case 13 the patient would probably have died from chest trouble if he had been given an anaesthetic.

This small series of cases provides evidence which shows that, if the perforation is small and spillage of gastric contents is minimal, spontaneous closure of the ulcer can occur, with subsequent resolution of peritonitis. It is precisely in this group of patients that operative mortality is minimal. More extended experience is necessary before we can decide whether the risks involved in conservative treatment will justify further trial. At the moment simple closure without drainage remains our standard treatment, but for doubtful cases where clinical signs indicate a leaking ulcer conservative treatment does seem reasonable.

Late Effects of Acute Perforation

Illingworth (1946) has shown that the longer the history before perforation and the sooner symptoms recur after operation, the worse is the prognosis, and that within five years 50%

of patients experience severe relapse. It will be of great interest to compare the late results in a group of patients after conservative treatment with a similar group treated by operation.

Effect of Previous Operation on Subsequent Gastrectomy

Table II shows that previous operation for acute perforation has no effects on the mortality of a subsequent gastrectomy. Provided the ulcer is simple the mortality remains 1.1%. If the ulcer has developed a chronic perforation the risk of gastrectomy is increased sixfold, and here again previous operation for acute perforation does not add to the danger of gastrectomy, the mortality being 6.6%.

TABLE II.—Effect of Previous Operation for Acute Perforation on Mortality of Gastrectomy for Duodenal Ulcer

	Cases	Deaths	Mortality
Simple D.U.	171	2	1.1%
Simple D.U. and acute perforation	23	0	
Chronic perforated D.U.	77	5	
Chronic perforation and acute perforation	15	1	6.6%
Total	286	8	2.8%

Simple D.U. includes all types of duodenal ulcers which have not penetrated all layers of duodenum. Ten were associated with previous gastro-enterostomy.

Most duodenal ulcers are kissing ulcers—one anterior and one posterior—and it is difficult to understand why the posterior ulcer penetrates so much more deeply and more often than the anterior. Although we had 92 chronic perforating posterior duodenal ulcers they were associated with previous acute anterior perforations on only 15 occasions. In every case of acute perforation simple closure had been performed.

Youdin has advocated gastrectomy for acute perforations, and his mortality is 6.0%, but in this country such radical treatment is considered unjustifiable. I have done it only twice, for patients who had each perforated on two previous occasions, and I can confirm that their post-operative condition compared favourably with that of patients by their side who had merely had a closure of an acute perforation.

Effect of Gastrectomy on the Incidence of Acute Perforation

In years past it was no uncommon experience for patients to perforate while in a medical ward under treatment or while on the waiting list for surgical treatment. One man recently insisted on a full six-weeks course of medical treatment, although his pain was not relieved by rest. He perforated in the early hours of the morning on which his gastrectomy had been planned. These experiences have taught us to advise urgent gastrectomy for all patients whose pain is not relieved by two weeks' adequate medical treatment, and to treat as cases requiring priority admission those who are considered suitable for gastrectomy. By this means we have undoubtedly anticipated acute perforation on several occasions.

One woman, aged 21, with a five-year history of severe recurrent pain, must have perforated during the induction of anaesthesia. On raising the liver a neat round hole was seen punched out of the duodenum. There were no limiting adhesions and no effort at localization. One good cough and her gastric contents would have spilled.

Another patient, a woman of 33, showed a neat pyramid of recently formed red oedematous fibrin lying on the duodenum. When this was wiped away a small perforation was found underneath.

In many other cases oedematous adhesions and massive induration were indications of the imminent perforation of the ulcer which lay underneath, for when the specimen has been examined after removal the ulcer base has been seen to consist only of peritoneum. In many of these cases active surgical treatment forestalled inevitable acute perforation.

Owing to the shortage of beds during the war we were not able to give preliminary medical treatment to all, yet to our surprise we found that operation during the active phase, without preliminary medical treatment, does not carry any increased risk, and is in fact much easier than during a quiescent interval. Just as an acute appendix will separate easily by gentle finger dissection during an acute attack, but requires sharp dissection with scissors to divide firm dry adhesions during a quiescent interval, so it is with the duodenum. The line of cleavage is more easily defined even if a chronic posterior penetrating ulcer is present, and in not one case in which perforation seemed to be imminent has the patient died.

An acute perforation "out of the blue" cannot be anticipated, but an acute perforation following a long period of dyspepsia can and should be prevented.

It has often been said that a rise in the number of gastrectomies performed will be accompanied by a disastrous increase in the operative death rate. We have found that for the years 1941-3 the mortality of gastrectomy was 12.3%. The number of acute perforations operated on by all the surgeons in York averaged 36 a year, and the combined operative mortality for these cases was 20%. With increased experience the mortality of the gastrectomies I have performed has fallen to 3.6% for the last 330 cases. The number of acute perforations occurring each year has been reduced by 44%, and the operative mortality of those who did perforate has remained disappointingly high—22%. These figures indicate that not only has the mortality rate of gastrectomy been materially reduced but there has been a substantial drop in the total number of acute perforations.

It seems reasonable to infer that we have in fact anticipated acute perforations in a considerable number of "cold" gastrectomies, substituting an operation which carries a mortality rate of 3.6% for one which carries a 20% risk if the ulcer is allowed to perforate.

TABLE III.—Including all Types of Peptic Ulcer—Gastric, Duodenal, and Secondary

Case Nos.	Previous Acute Perforation	Chronic Perforation	Previous Acute Perforation and Subsequent Chronic Perforation
1-100	24	22	4
101-200	14	25	5
201-300	13	33	5
301-400	6	43	4
400	57	123	18

Table III shows the following points of interest: (1) The diminishing incidents of a history of acute perforation. In each succeeding 100 patients accepted for gastrectomy there has been a history of fewer acute perforations. (2) The marked increase in the number of chronic perforations among the last 200 cases. (3) The relatively small proportion of cases in which a chronic posterior perforation has been associated with a previous acute anterior perforation.

Prophylactic Gastrectomy

Patients who have perforated more than once are in special danger, and no one who has had a second perforation should be allowed to risk a third. For him prophylactic gastrectomy is indicated, and it must be no timid two-thirds removal, as the following cases indicate.

Case 350.—A man aged 48 gave a history of three acute perforations treated by suture. Three years after the third acute perforation a "gastrectomy" was done, which gave relief for eighteen months. When I first saw him there were four abdominal scars, each with a hernia. I operated for the fifth time, and found that a very timid two-thirds gastrectomy had been done, which had not prevented the formation of an enormous jejunal ulcer that had penetrated into the liver. It was possible to remove the greater part of the stomach remaining, together with the jejunum containing the ulcer, leaving only a small cuff of stomach for the anastomosis. This patient is now symptom-free, and since he has achlorhydria an ulcer is unlikely to re-form.

Case 101.—A man was admitted six hours after his third acute perforation. As his general condition was good I thought it would save him a fourth operation if I performed an emergency two-thirds gastrectomy. The operation presented no difficulty, and his convalescence was uneventful. Twelve months later he developed symptoms due to stomal ulcer. A test meal was high normal, and radiographs confirmed the presence of stomal ulcer. A very radical secondary gastrectomy has eliminated free acid, and he is now symptom-free. It would have been better had I treated the third acute perforation conservatively, and performed a more radical gastrectomy two weeks later.

These cases indicate the necessity for performing a very radical 90% gastrectomy for those who have had multiple acute perforations.

As a result of my experience during the past five years I have come to the conclusion that there is justification for the further trial of conservative treatment of acute perforated peptic ulcer. A more urgent radical treatment for chronic peptic ulcer causing severe symptoms which do not respond to medical treatment has on many occasions proved successful in anticipating acute perforation.

Summary

There has been a gradual trend towards increased conservatism in the treatment of acute perforated peptic ulcer.

A small series of 14 unselected consecutive cases of acute perforation were treated conservatively—twelve duodenal ulcers, one gastric ulcer, and one stomal ulcer. Of the 11 survivors one developed severe symptoms within a month of his discharge.

The essentials of treatment are relief of pain by adequate doses of morphine, and prevention of further leakage by aspiration of stomach contents. Intravenous sulphathiazole was given to all.

Previous operation for acute perforation has no effect on the mortality of subsequent gastrectomy, which is 1.1% for duodenal ulcer that has not penetrated deeply. If a chronic perforation has occurred the mortality of gastrectomy is 6.6%. Only 15 out of 92 chronic perforating duodenal ulcers were associated with a previous acute perforation.

Acute perforation can be anticipated and prevented by more urgent surgical treatment of chronic peptic ulcer. Such cases need priority admission.

In York the incidence of acute perforation has fallen 44% during the last two and a half years, but the mortality of those who do perforate remains disappointingly high—22%.

A patient who has perforated more than once needs prophylactic gastrectomy. It must be very radical (at least 90%).

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INOCULATION EXPERIMENTS AGAINST TYPHUS IN AFGHANISTAN

BY

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Mild and severe typhus can be seen outside Kabul throughout the year. During the summer cases are only sporadic, but they become more frequent in the autumn; in winter the disease reaches its maximum incidence, with a further exacerbation when the snow begins to melt. There are no marked differences in clinical characteristics in different seasons of the year, but cases with severe haemorrhagic eruptions and, on the other hand, those without exanthemata diagnosable only by serological examinations, are observed. In the hospital of the central prison, Kabul, in one year out of 249 cases of typhus 213 persons recovered and 36 (14.4%) died.

When sera of doubtful typhoid or typhus cases are sent from the hospitals or the city to the Bacteriological Institute here, agglutination tests are done in six rows of tubes with six different types of microbes—i.e., *Salm. typhi*, *Salm. paratyphi* A and B, and *Proteus* OX 19, OX 2, OX K. One hundred and twenty-three sera positive for one, two, or three types of microbes gave Weil-Felix reactions (W.F.R.) for *Proteus* OX 19 (116 sera positive and 7 negative), for OX 2 (73 positive and 50 negative), and for OX K (33 positive and 90 negative). If the result of agglutination with OX 19 was negative, but positive with OX 2 or OX K or both, it was a sign that the sera were from cases occurring during the month of May or the autumn. Thus although the patients were suffering from typhus, their sera in 5.7% of cases gave a negative W.F.R. with OX 19 but positive results with one or both of the other types. The Weil-Felix test must be done here with all the three strains of *Proteus*, as doubt will be created in cases with negative serological results to one strain.

In May, 1937, severe typhus broke out in the region of Ayyak, but the physicians there thought it was plague because pasteurellae had been found in patients' sputa. The disease was very severe, but exanthemata were seen in only a few persons, on the medial side of the thigh. My assistant was sent with all necessary equipment, and after making bacteriological and serological examinations, and experimental inoculations of material brought from the patients, he established that they were not plague cases. The W.F.R. was done with nine types of *Proteus* from the Lister Institute, London, results being positive with the OX 2 type but negative with all the others. This and further observations led me to the conclusion that different strains of *Proteus* were necessary for the diagnosis of the typhus of this region. The literature to date does not show OX 2 of *Proteus* as an independent agglutinin but as a coagglutinin, while in the Institute of Bacteriology at Kabul it has been found that free OX 2 agglutinin exists in the serum of typhus patients.

Blood Vaccine

In the winter of 1941 typhus was observed in and around Kabul. Prophylactic inoculation was possible only in Allied armies. In some institutions hygienic measures were taken to arrest the disease, and I inoculated with my vaccine those who had not yet been infected. The Government also permitted me to make experiments on the prisoners. The blood used in the preparation of the vaccine was taken in the second week from otherwise healthy patients who had rashes before the tenth day of the disease, and had high fever, positive Weil-Felix and negative Kahn reactions. It was put into sterile

bottles, defibrinated, and then filtered through sterile gauze into cylindrical bottles, after addition of 0.5% carbolic acid (10 ml. of 5% solution acid in isotonic saline to 100 ml. blood), and kept one hour at 56° C. in a waterbath. I sometimes used 0.5 ml. of 5% formaldehyde in isotonic saline to 100 ml. of blood instead of carbolic acid. After checking its sterility, aerobically and anaerobically, this vaccine was kept in a refrigerator and warmed to blood-heat before use. It was inoculated three times subcutaneously into the abdominal wall 5 to 10 cm. from the navel, not more than once a week, the first dose being 2 ml., the second and third doses 4 ml. Four or five hours after the inoculation the patient began to feel unwell; the area of inoculation was hypersensitive to the touch and bluish in colour; but after about four days these signs disappeared.

In the prison at Kabul in 1942, 196 persons were inoculated in the spring and 368 in the autumn; other people who feared an infection or who found a louse on their bodies also came to the Institute of Bacteriology, Kabul, and were inoculated with my vaccine. Among the 196 prisoners not one case of infection was noted for six months. Between the sixth and seventh months 9 persons caught typhus; in 7 the disease had an uneventful course, but 2 died. Of the latter one was old, the other was weak and suffered from consumption. Of the batch of 368, 13 caught the disease; 11 of these recovered and 2 died. As the vaccine could not be prepared for more than 80 persons at a time only those who so wished and who had not previously had typhus were inoculated. Although all the prisoners lived together, out of 13 cases only two feeble treated patients died; but in the months of December and January, when the disease was very serious among the untreated, of 110 cases 89 recovered and 21 died. The result established the prophylactic efficacy of my vaccine, and these experiments showed that it could protect susceptible persons against the disease for 5 to 6 months. This experiment was made as a gesture of good will at a time when no better vaccine was available in the prison and in cases where there was no other way of giving active immunity.

American (Cox Type) Vaccine

In 1943 it occurred to me to try American vaccine, obtained through the Legation, and experimental inoculations were given as follows: May 7, 124 persons in the detention houses, 195 persons in the general prison; June 12, 100 persons in the women's prison; July 31, 150 policemen and 353 police soldiers; in all, 922 persons. In the first week of September 691 soldiers of the First Division, and in the first week of October 851 soldiers of the Second Division—in all 1,542 persons—were inoculated (three inoculations, each of 1 ml. subcutaneously, at intervals of one week). Because the vaccine in the general prison was not sufficient, only those who consented, and among the soldiers only those recommended by the Surgeon Major, were treated with the preparation of the Bacteriological Institute. All the other persons living together with the inoculated remained under control. Among the inoculated persons in the detention houses and the women's prison not one case of typhus was recorded during the first six months. In the central prison, of the inoculated 2 fell ill but recovered; and of the uninoculated 15 caught the disease, 12 recovering and 3 dying. Among the inoculated policemen and armed police not one case was noted, but of the uninoculated 5 fell ill; 4 of these recovered and 1 died. In March, 1944, the American Legation again sent some vaccines, and 214 soldiers of the First Division, 237 of the Second Division, and 207 of the independent armed police troops—in all 658 persons—were inoculated. During one year after these inoculations not one case was noted among those protected, but among the unprotected in the First Division 23 caught the disease, 18 recovering and 5 dying; in the Second Division 11 fell ill, 10 recovering and 1 dying. Among the independent companies of the police soldiers 30 persons caught the disease; 27 of these recovered and 3 died. The result of these experiments—not one case occurring among the protected persons, but 9 deaths out of 64 unprotected—established the efficacy of the vaccine.

Summary

It has been established that in the typhus cases seen in and around Kabul, carrying a mortality of 14.4%, the sera taken from the