Sternberg, as soon as a vessel flying a yellow flag anchors. When transports or large liners arrive, as well as in the case of suspected vessels, the quarantine officer, four junior medical officers and a female inspector, go aboard. Temperatures or glandular examinations, or both, are taken of the personnel of vessels from plague or yellow fever ports; freight manifests are examined, baggage of steerage is looked at for certificates of medical officers, the general condition of the vessel as to mechanical cleanliness gone into, and a careful examination made of the source of food and water supply, especially if from cholera ports; the report of the vessel's surgeon, the bill of health and anything else pertinent to the inquiry. In addition to this careful inspection of every person and thing for quarantine, the quarantine officer is charged with the medical inspection of aliens for the Immigration Bureau of the Department of Commerce and Labor, and, as neither this bureau nor the transportation companies provide any other place, this inspection is done aboard ship.

Opened April 29, 1891, the first vessel treated was the *China,* which arrived on December 20th, 1891, with smallpox on board. No sanitary inspection or disinfection of vessels was done by the Federal authorities, these being made by the city quarantine officer who was appointed by the Governor. Neither the State nor the city had any quarantine buildings or modern appliances, and the station was used as the national and local quarantine by the desire of the State and local boards of health.

In 1892 the fumigating steamer *George M. Sternberg* was completed. In 1895 the Legislature of the State passed a joint resolution requesting the Federal government to assume entire control of the maritime quarantine service at the port of San Francisco, meaning the inspection of vessels in addition to the quarantine functions already performed. The Chamber of Commerce of San Francisco passed similar resolutions, and the steamer *George M. Sternberg* was put into commission as boarding steamer.

May 11, 1897, the duty of medical inspection of immigrants was assumed, and after a disagreeable controversy between the local quarantine officer and the commanding officer of this station, the latter was appointed by the President by virtue of the power given him by Section 3, of the Act of February 15, 1893, quarantine officer at the port of San Francisco, California. The following officers have been in command of the station:

Surgeons Preston H. Bailhache, until June 13, 1891; W. P. McIntosh, June 13, 1891, until May 2, 1892; D. A. Carmichael, May 2, 1892, until April 16, 1894; Passed Assistant Surgeons, J. H. Oakley, April 16, 1894, until June 8, 1894; C. T. Peckham, June 8, 1894, until March 2, 1896; M. J. Roseman, March 2, 1896, until December 6, 1898; Surgeon Brooks, December 6, 1898, until June 17, 1899; J. J. Kinyoun, June 17, 1899, until May 3, 1901; D. A. Carmichael, May 3, 1901, until January 2, 1902, when the writer became quarantine officer at the port of San Francisco.

A FEW ILLUSTRATIVE CASES OF DIPHTHERIA.*

By FRED GRANT BURROWS, A. M., M. D., San Francisco.

THE Board of Health of San Francisco has kept complete records of the cases of diphtheria that have occurred in that city since 1896. These records show that the disease is increasing; and that the greatest number of cases recorded in any one year occurred during the last fiscal year, 1901-1902. During that year there were 1357 cases with 204 deaths, or a death rate of 15%. During the six years covered by these records there were 4297 cases of diphtheria and 704 deaths, a death rate of 16.38%.

It is well known that both the morbidity and the mortality from diphtheria are universally greater during the cold winter months than during the summer. Under like conditions of treatment we should therefore expect more favorable results in this climate than in the severe climate of that part of the United States east of the Rocky Mountains. We have failed, however, to achieve as good results. The death rate from diphtheria is now greater in San Francisco than it is in New York, Boston or Chicago. Not only is this true, but the number of deaths is increasing here and decreasing there. There were more than three times as many deaths from diphtheria in San Francisco during the fiscal year 1901-1902 as there were during the year 1896-1897. Whereas the number of deaths from the same disease in the combined population of the five largest cities of the United States was considerably less in 1901 than it was in 1896. During 1901 there was one death from diphtheria in San Francisco for every 1680 of the living; and in the five cities above referred to there was only one death from the same disease for every 2,128 of the living. †

Statistics from all the large hospitals of Europe and the United States prove beyond any possibility of doubt that the death rate from diphtheria properly treated with antitoxin is very much less than it is from that disease treated without antitoxin.

†The combined population of New York, Chicago, Philadelphia, St. Louis and Boston in 1900 was 7,556,586; there were 3555 deaths from diphtheria in those cities in 1901. San Francisco had a population of 342,782 in 1900 and there were 204 deaths from diphtheria in that city during the fiscal year 1901-1902.

*Read at the Thirty-third Annual Meeting of the State Society, Santa Barbara, April 21-23, 1903.

The hospital death rate before antitoxin was used was over 45%. It has now been reduced in most large hospitals to less than 15%. It is known that diphtheria is caused by a specific organism which during its life processes eliminates a virulent toxin. This toxin is taken up by the blood and distributed throughout the body, where it produces poisonous and destructive effects. It is also known that this toxin is non-selective in action and may poison any tissue or destroy any organ; and that the general symptoms of the disease are due to the virulence of the toxin. Laboratory tests and clinical observations have proven that antitoxin has the power of completely neutralizing this toxin; but in order to save the tissues of the body from the destructive action of the toxin it must be neutralized early. In the treatment of diphtheria, therefore, the first aim of the physician should be to neutralize this toxin at the earliest possible moment. Abundant clinical experience has shown that antitoxin never produces harmful effects, and that it may be given in any amount. A single dose may be 6,000 units or 8,000 units. The paralyses, heart complications and albuminuria sometimes seen in those cases recovering after the use of antitoxin are not due to the antitoxin, but to the toxin that was not neutralized before it had time to injure the tissues.

I believe that under ideal condition it would be possible to save 99% of patients with uncomplicated diphtheria. The present contagious diseases department of the Boston City Hospital was opened in 1895. During the first five years of the existence of that department, more than 7,000 patients with diphtheria were treated in the wards, and more than 100 nurses, physicians and employes of the department contracted the disease. The infections of these hospital attaches were usually severe, but they all recovered, and they all recovered because they were treated at the very beginning of the disease with sufficient antitoxin. One hundred per cent of that series of more than 100 cases were saved. In the same hospital, in a much smaller department, 1,421 diphtheria patients were treated during the four years immediately preceding the advent of antitoxin, and two physicians, two nurses, and one orderly died from the disease contracted from these patients. Let me state these facts in another way. In a given hospital during a period of four years, only 1,421 cases of diphtheria were treated, and five members of the hospital staff died from the disease contracted from these patients. During another period of five years more than 7,000 cases of diphtheria were treated; and although many members of the staff contracted the disease, not one of them died. The only essential difference in the treatment of the members of the staff during these two periods was in the use of antitoxin during the second period.

Our knowledge of the effects of antitoxin and the proper methods of using it has been a matter of growth. The history of several attacks of diphtheria from which an orderly in the hospital above referred to suffered, will illustrate this point and seems convincing evidence.

Case 1, W. J. C.—An orderly in the diphtheretic wards. His first attack occurred Jan. 1899. He was given 2,000 unit doses antitoxin and received 37,000 units in 120 hours. He was sick in the hospital thirty days, but made a good recovery. His second attack occurred March, 1900. He was given 4,000 unit doses antitoxin and received 16,000 units in 48 hours. He was in the hospital four days, and made a good recovery. His third attack occurred in July, 1900. He was given two 5,000 unit doses early and was not really sick at all. He had learned from experience to recognize his symptoms early, and we had learned from experience to give antitoxin early and in large doses.

The history of an attack of diphtheria from which one of the house physicians of the above hospital suffered shows the results that may be obtained by early and vigorous use of antitoxin.

Case 2, Dr. A.—On duty in the diphtheria wards. On Dec. 5, 1899 his throat felt slightly sore. The next morning it was a little worse and there was a small patch of diphtheretic membrane on one tonsil. A diagnosis of diptheria was made and 4,000 units antitoxin given. At noon the patch was thicker and larger and the dose of antitoxin was repeated. By 6 P.M. the membrane had not begun to disappear and another 4,000 units of antitoxin was given. The throat was again examined at 11 P. M. and the false membrane did not seem to have grown larger. It was therefore thought that no more antitoxin would be needed. The next morning, however, it was found that the patch had enlarged and thickened; there was also a patch on the other tonsil and a film on the uvula. It was decided to give more antitoxin, and 4,000 units were given at 8 A. M. At noon of that day (the second of the disease) the throat looked much as it had in the morning, so another 4,000 units of antitoxin were given. By evening the throat seemed to be clearing. We were not willing to take any chances, so gave 4,000 units more. The patient had thus received 24,000 units in 36 hours. On the following day the throat cleared rapidly, became entirely clear on the fourth day of the disease, and the patient made a rapid and uneventful recovery. His temperature was never above 100°; his urine never contained albumen; his heart was not effected; and there was no post-diphtheretic paralysis.

This patient was a physician who had a severe infection contracted from his diphtheretic patients. In spite of large initial doses of antitoxin the disease spread rapidly, but was quickly controlled when sufficient antitoxin had been given to neutralize the toxin and render the conditions unfavorable for further growth and development of the diphtheria bacilli. There were no complications because the toxin was neutralized early, before it had time to affect the tissues. There were no severe constitutional disturbances

because the toxin was neutralized as it was produced. The patient was absent from duty only thirteen days, and was in the ward as a patient only ten days.

Another patient seen in private practice illustrates the advantages of early antitoxin treatment

Case 3—A child between two and three years old was seen in consultation in the evening of Dec. 8, 1902. The patient was first seen by the family physician at 11 P. M. the day before, and the history was that he had then been croupy about 24 hours; that is he had been croupy 48 hours when I saw him. I was called to perform intubation as the child had considerable dyspnea and was rapidly growing worse. He had already been given 3,000 units antitoxin. An examination showed that there was no nasal nor fauceal diphtheria, but considerable laryngeal stenosis, with paroxysms of coughing during which the dyspnea and cyanosis became marked. Phonation was still possible and the dyspnea was not extreme. Considering the dangers of accidents to an intubed patient situated as he was, it was decided to give more antitoxin and if possible avoid the operation. We gave 3,000 units then and visited him again at midnight prepared to operate if necessary. We found however that he was considerably better and had been sleeping quietly. He was again given 3,000 units of antitoxin and the expectant treatment continued. I did not see him again, but his physician reported him much better the next morning. In the afternoon, however, returning stenosis demanded more antitoxin and 2,000 units were given. From that time he made a rapid recovery. There is no doubt in the mind of this patient's physician or myself that with less vigorous antitoxin treatment, intubation with all its dangers and responsibilities would have been necessary.

Only those who have had actual experience with such cases can appreciate the difficulties of properly caring for intubation patients outside of a hospital. San Francisco, unfortunately, has no hospital properly equipped for their treatment.

Another patient from the hospital series illustrates what may sometimes be accomplished in the treatment of late and neglected cases:

Case 4, E. L.—A slender, frail young man, 18 years old, was admitted to the Boston City Hospital Dec. 8, 1899. He had been ill with diphtheria six days and had had no antitoxin. He was delirious and greatly prostrated. His pulse was soft, irregular and intermittent. His heart was dilated and there was a well marked systotic murmur heard over the greater part of the precordia. A thick diphtheretic membrane completely covered the tonsils, uvula, and soft palate, and extended onto the hard palate. There was an abundant dark, glairy, serous discharge from the nose, and diphtheretic membrane could be seen extending to the margins of both nostrils. The cervical glands were greatly enlarged, and the diphtheretic odor was so marked that a diagnosis could have been made in the dark. The patient seemed in an exceedingly critical condition, and it was not thought possible for him to live until morning. Up to this time 4,000 unit doses were the largest ever given in the hospital and, so far as I knew, the largest ever given anywhere. As this was a desperate case—such as was never known to recover without antitoxin—and as my experience with large doses of antitoxin had led me to believe thoroughly in such treatment, we gave him 6,000 units as an initial dose. He began to improve and was given 6,000 units every four hours until he had received 84,000 units—all in 78 hours. At the time of admission to the hospital he was delirious and refused to swallow. He was therefore fed and stimulated by rectum. He expelled his enemata, and it was necessary to compel him to retain them by applying an anal pad in such a way as to render this impossible. This method of alimentation had to be continued for three days. On the seventh day in the hospital, or the thirteenth day of the disease, the patient suffered from one of those attacks of extreme weakness or collapse so frequently seen in severe cases of diphtheria. Vigorous subcutaneous stimulation revived him and he continued to improve. At the time of admission to the hospital his urine contained albumen; but this disappeared in a couple of weeks and the patient was discharged well after 35 days in the hospital. I have not seen a patient more seriously ill from any disease, recover. His history seems to justify the opinion that it is never too late to give antitoxin, and that large doses should be given in severe cases. It also demonstrates the well-established fact that the cardiac disturbances and albuminuria are due to the toxin of diphtheria, for they were present before antitoxin was given.

The two following cases are of unusual interest and illustrate several points:

Case 5, N. C.—On Jan. 29, 1902, I was called to the home of a physician to see his child, a boy of five years of age. I was told that the child had been croupy three days, and had grown steadily worse. I found him suffering from marked laryneal stenosis. There was great dyspnea, considerable cyanosis and profuse perspiration. The child was aphonic, and had frequent attacks of coughing, the cough being brassy and paroxysmal in character. Respiratory effects caused retraction of the substernal and supraclavical spaces, and marked dilitation of the alae of the nose. In short, the respiratory efforts presented an exaggerated picture of those of an athlete after severe and prolonged exertion. No diphtheretic membrane could be seen in the throat or nose, and there was no nasal obstruction. As diphtheria laryngitis is the only disease of childhood leading to a gradual stenosis, a diagnosis of diphtheria was made, and as suffocation was imminent the child was intubed. The insertion of the tube afforded instant and complete relief. The breathing became easy, all the above symptoms disappeared and the child went to sleep. As he lay sleeping he seemed in perfect health, but the sad experience of the many physicians who treated diphtheria before 1894 warned us that without further treatment he would probably soon awake to the agonies of suffocation, and that his chances of ultimate recovery were less than thirteen in a hundred. The subsequent history of the patient proved these chances to have been pactically nil. We therefore began antitoxin treatment and gave him his first dose at 3 P. M. He received 3,000 units at 3 P. M., 3,000 units at 9 P. M., 3,000 units at 2 A. M. the next day, and 3,000 units at 8 A. M. At 9:45 A. M. the second day, Jan. 30th., his laryngeal tube became accluded and had to be removed. As he had difficulty in breathing without the tube it was again inserted, but gave no relief, so was at once removed, after which a membranous cast of the upper part of the trachea was coughed up. The patient then breathed freely without the tube and no more antitoxin was given until the evening, when returning dyspnea showed that the diphtheretic process was extending. It also showed that we had stopped giving antitoxin too soon. We now resumed the antitoxin treatment, and 3,000 units were given at 7:30 P. M. As the patient did not im-

prove, the dose was repeated at 11 P. M. and at 3 A. M. Jan. 31. By 6:30 A. M. the dyspnea became so great that the tube was again inserted. The insertion of the tube loosened more diphtheretic membrane from the air passages, which was coughed up. When the tube was again inserted the breathing was unimpeded. This second cast was very extensive, including part of the trachea, both primary bronchi and many secondary bronchi. On one side of the cast ten branches from as many small bronchi can be seen. This membrane showed that the inflammatory process had extended into the very center of the lungs. It also warned us that still more antitoxin was needed. He had already received seven 3,000 unit doses of antitoxin, which had caused the false membrane covering the mucosa of the air passages to be thrown off. We knew the mucosa of the air passages was inflamed. We knew that this inflamed mucosa was infected with thousands of the bacilli of diphtheria. We also knew that the conditions in this inflamed mucosa of the air passages was favorable for the growth and multiplication of the bacilli.

We determined if possible to neutralize the toxin that would be produced in their growth. Therefore we gave more antitoxin. We gave 3,000 units at 6:30 A. M., 3,000 units at 10:30 A. M., 3,000 units at 2:30 P. M., 3,000 units at 5:30 P. M., and 3,000 units at 9:30 P. M. That is to say, although he had already received 21,000 units, this five-year old child was given 18,000 more units of antitoxin in eighteen hours. At 4:30 P. M. of that same day, Jan. 31st., the laryngeal tube again became accluded and had to be removed, after which a piece of false membrane, evidently from part of the trachea, was coughed up. The patient then breathed easily and went without the tube until 7:30 A. M. the next day, Feb. 1st., when it was again needed, this time on account of swelling and edema of the larynx. The tube was now worn until 9:30 P. M. of the same day, when it had to be removed on account of partial acclusion with dry mucus. The patient then went without the tube for fifteen hours, when it was again demanded, at 12:30 noon, Feb. 2d., for the last time. It was finally removed at 7:45 P. M. Feb. 4th., or a little more than six days after it was first removed.

The last dose of antitoxin I have told you of was given at 9:30 P. M. Jan. 31st., by which time the patient had received 36,000 units. For reasons above given we were afraid to stop there and continued the treatment. On Feb. 1st. the patient was given four more doses of 3,000 units each at 2:15 A. M., 8 A. M., 12 noon, and 3 P. M. That is, we gave antitoxin until the character of the pulse, respiration and bronchial secretions indicated that no more was needed. To recapitulate. This five-year-old child was given 48,000 units antitoxin in 72 hours. He was intubed six times, and the tube was removed with the extractor three times; each of these nine operations was imperatively demanded. During this entire illness the temperature was never above 100, and was for the greater part of the time normal. The pulse was usually about 120, but during the night of Jan. 30th., when the diphtheretic process was so rapidly extending into the lungs, it was in the neighborhood of 140 and was an important indication for more antitoxin. The urine contained a slight trace of albumen for one or two days only. During the time the laryngeal tube was worn, the patient was not allowed to swallow food nor drink, but was fed by esophageal tube and by rectum. He was fed partially by rectum because the esophageal tube caused some vomiting. All rectal feedings were retained and assimilated. There were absolutely no complications and the recovery was rapid and uninterrupted.

This child had been critically ill. He had been in imminent danger of suffocation and in imminent danger from the virulent toxin of diphtheria. If there is any other disease of childhood that may cause a similar inflammatory condition of the air passages and lungs from which the patient will recover, I have yet to learn of that disease. If one patient ever recovered from such a diphtheretic infection without the use of antitoxin, his history has not been published.

Case 6, M. C.—The child whose history I have just read has a sister two years his senior. She was then seven years old. She was said to have had a cold for a week or two before I saw her brother, and the cold was thought peculiar inasmuch as the nasal discharge seemed to come from only one nostril. An examination showed a pale, delicate-looking child with a rapid pulse and a considerable dark, glairy, serous discharge from one nostril. Further examination showed that nasal cavity to contain a small piece of diphtheretic membrane. Cultures taken from the nose gave an abundant growth of diphtheria bacilli The patient was given 3,000 units antitoxin and made a good recovery. She had not been considered ill, but had had this nasal discharge for a week or ten days. There is no doubt that her brother contracted the disease from her. Her infection was a comparatively mild one of the nasal type, and she had been running about in a physician's family with the disease unrecognized. The association of these two cases emphasizes the importance of recognizing the mild cases, and especially the cases of nasal diphtheria. Although hers was, so far as it had gone, a mild case, her brother contracted a most malignant form of the disease from her. She might have recovered without treatment, but the disease might have lighted up at any time and proved fatal; or she might have continued with the nasal discharge for weeks or months and infected scores of other children. These two cases are of unusual importance, for aside from the fact that they are of medical interest the patients are the children of an eminent physician. Their father is an authority on some medical subjects, but he no longer treats children's diseases and some of the recent literature on diphtheria had escaped his attention. He said he had never seen a case of nasal diphtheria to recognize it as such, and he thought his boy had a bad cold and acute laryngitis.

I wish to emphasize the fact that nasal diphtheria is a common form of the disease. A single unrecognized case of nasal diphtheria may be the source of a severe epidemic. This is especially true among school children, and inmates of orphanages and children's homes. I also wish to emphasize the fact that the most severe form of the disease may be contracted from a so-called mild attack.

I wish further to emphasize the fact that progressive laryngeal stenosis in children is always caused by a diphtheretic inflammation of the larynx. So-called membranous croup is always diphtheria. Spasmodic croup or acute laryngitis is a disease of sudden onset. It is usually accompanied by considerable fever and the pulse is

not disproportionately accelerated. The symptom complex is entirely different.

I have presented a detailed account of these few cases of diphtheria hoping to show the importance of early and vigorous antitoxin treatment. I have also tried to show the importance of recognizing the nasal type of the disease. It is possible to have an infection of the anterior nasal cavity run a long and subacute course. Such patients are a menace to the health of the community, and should be promptly isolated and treated.

In the treatment of the disease, I have considered only the use of antitoxin. If the toxin is neutralized as it is produced, there remains but little to do. The value of alcoholic stimulation in severe and neglected cases is so universally recognized that further emphasis seems unnecessary. Local treatment I believe to be worse than useless. The exhibition of antiseptics and corrosives usually results in larger areas of excoriated mucosa, which are promptly invaded by the bacteria of the disease. Moreover, the tissues of an individual suffering from diphtheretic intoxication are not in a condition to withstand the strain put upon them by the resistive efforts usually put forth against local applications.

I have shown that the present death rate from diphtheria is greater in California than elsewhere. This high death rate cannot be attributed to our climate nor to the unsanitary condition of our cities. Our climate is mild; our cities are not overcrowded; and there is no extreme poverty in our midst.

It is our duty to lower this high death rate. We can do it by recognizing the disease early in each infected individual, isolating each patient and giving antitoxin freely. Let me beg you to accept this responsibility. Give antitoxin early. Give it in large doses. Repeat the doses often. Do not stop giving antitoxin until the toxin of the disease is completely neutralized.

DISCUSSION.

Dr. Geo. L. Cole, Los Angeles—The cases which Dr. Burrows reported were exceedingly interesting. We should be guided somewhat by the symptoms and not alone by the pathologist's report. I wish also to emphasize the early use of antitoxin. My own faith in antitoxin and my ideas regarding the late use of it have changed within the last few years. There is no question about the efficacy of large doses; but after the 5th or 6th day I question whether we do get as much good as we expected. One very interesting thing with regard to the increased diphtheria rate in San Francisco, it has grown more rapidly in the last 3 or 4 years than it did for 5 years previously. This has been noticed in Los Angeles. In San Francisco you have been turning up streets and doing other city work. In Los Angeles several years ago, when growth was rapid and in that portion where there was constant grading and turning up of the earth, diphtheria was pretty abundant. In talking to Dr. Powers he said that after every very severe windstorm, lasting a couple of days, he had noticed there was an increase in the number of cases of diphtheria. The winds coming down from the mountains were followed with excessive diphtheria. If I were to take exception to any one thing, it would be that local treatment was worse than useless. I cannot think this. I agree with him that in small children where, to make local treatment you have a struggle with the child, this is true; but I do not believe when we consider the possibilities of our antitoxin in some cases that we are justified in leaving off all local treatment. Another thing has occurred to me recently which I wish to emphasize. We know that the manufacturers put up large batches of antitoxin and if it does not quite come up to the standard, and they are a little short, will they absolutely destroy this antitoxin? Is it not possible that this antitoxin may be insufficient in some way? Therefore it has occurred to me that instead of going back to the same antitoxin, I should get another lot. There is one more point with regard to the feeling that I know exists in the profession, that all cases of diphtheria can be cured. I believe there are some cases of diphtheria that will die in spite of all antitoxin. I knew of a case recently where a woman had lost her only child. Her heart was broken. She went into a distant city and someone asked her why she was dressed in black. She said she had lost a child with diphtheria. The physician told her there was no need for the child to have died with the proper doses of antitoxin. We should never say that all cases of diphtheria can be cured. Give a child very large doses of antitoxin under the best of circumstances and sooner or later there will be recorded cases which it was impossible to cure.

Dr. J. Henry Barbat, San Francisco—It is pretty gratifying to hear these papers on diphtheria and on antitoxin. In 1892 I read a paper on this subject and I stated my belief that we would shortly be treating diphtheria with antitoxin. There was no attention paid to it. Dr. Cole has not expressed the reason for the increased mortality rate of diphtheria in San Francisco. The reason is that the so-called family doctor, the man who has no time to attend to medical meetings, cannot read his journal, does not know anything new, hears somebody talk about antitoxin, and thinks he will try a little antitoxin; he gives 500 units to a child who needs 10,000 and the next day the child dies. They are the men who treat the large majority of sick children in San Francisco and possibly in Los Angeles. That is something we do not know how to get around. They will have to die out; I do not see any other remedy.

Dr. George H. Evans, San Francisco—It seems to me that there are two points of major importance. They are the recognition of diphtheria and the administration of sufficiently large doses of antitoxin to cure the disease. I have had some little experience in this disease for a period of 12 years and have had occasion to compile statistics from boards of health. I presented the report of a number of cases which I culled from S. F. Board of Health statistics. There were a number of cases in which the mortality rate was a little less than 13% and I also remember this fact that in every fatal case the patient had either been found moribund or antitoxin had been given in ridiculously small doses. One case was of the laryngeal type and only 500 units of antitoxin had been given. We must give this fearlessly. I do not agree with Dr. Maher in giving such small doses. Dr. Burrows will bear me out in this matter. As evidence of the fact that these small doses are insufficient I would

like to bring up an illustration. Last week I was called in to see a patient early in the evening. I found the child had been to Sunday school that morning, had complained of not being well for 2 or 3 days, had come home that day and had developed a high temperature with delirium, swelling of the neck, and for the first time complained of sore throat. I found a child of nine years of age with both tonsils and a portion of the anterior pillars of the fauces covered with a membrane. That child immediately received 3000 units of antitoxin. As the child was nauseated no other treatment was given. I saw the child the next morning, and while the dose had spread the membrane and had been sufficient to prevent death, the child was still markedly toxemic. I immediately administered 6000 units more and that afternoon convalescence had commenced. It is a difficult matter to get at the proper dose of this drug.

Dr. J. Maher, Oakland—If there is anything which Dr. Burrow's paper emphasizes in particular it is the importance of meeting the toxins with antitoxin in sufficient quantity to neutralize them. That is what his paper has proven beyond all other things. I have in my paper confined myself to established facts. When we get enough of such experiments as Dr. Burrow's, then we can establish them as facts. I go a long way in agreeing with him in regard to the dose. I do not mean to say when I mention 1500 units that we begin all cases with that amount. I was simply making reference to the broad range that we have in the different cases. I have used myself 2500 or 3000 units with very young children. In some cases I have used 5000 or 6000 at the first dose. I think that one generally accepted fact is that after the administration of antitoxin, local treatment should not be abandoned.

Dr. Burrows—I lived 2 years in a diphtheria hospital in New York and we had 250 cases most of the time. I never had it and never took any antitoxin. I was a youngster and gave very large doses although many critics said that they would kill the patient. I do not believe in giving large doses to everybody. If one dose is sufficient, all right, otherwise give another. In ideal conditions in uncomplicated cases, 99% of patients can be saved. Patients do not get antitoxin early enough. Sometimes the onset is exceedingly quick. Not all of the increased death rate can be attributed to the practitioner. In the case reported, this physician's boy almost died. He was sick three days before he recognized it. I once had a case of a child of a physician and I lived 6 days and 6 nights under the same roof, treating and watching that child. The father objected to giving any more antitoxin than was necessary, and asked if it were necessary every time a dose was given. We should try to neutralize the toxin. As far as pseudo-diphtheria is concerned, I do not know anything about it. All cases which have membranes covering the throat I call diphtheria and I treat them as such. If there is a thick membrane this may contain millions of bacteria. The mucosa underneath may contain as many, but the swab does not remove them. In diphtheric inflammation you find that the chronic process extends for a considerable distance into the mucosa. As Dr. Barbat said, we often meet doctors who know nothing about antitoxin or its use.

The Colorado State Medical Society is considering the establishment of its own journal, to take the place of the annual volume of Transactions it has hitherto published. At its last meeting the publication in journal form was strongly advocated; and a committee has been appointed to submit plans and estimates for such a journal at the meeting of the Society to be held October 6 and 7, 1903.

ABDOMINAL DRAINAGE.*

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THE present paper is presented not because the writer has any new theories to advance on the subject of abdominal drainage, but because the general views and practice regarding it have changed so greatly in the past few years, that it seems well to submit it to you for consideration and discussion. It is my intention, and I consider it my duty, to contribute to the subject the results of my own experience, which if not great, has been varied and may add something to the weight of authority which is accumulating on this subject. There is not time to present anything like a complete history and review of the subject in the limits of the present paper, and I shall not burden you with statistics, though at some future time I may consider it my duty to do so.

As late as seven or eight years ago it was the custom to use drainage in the great majority of cases after intraabdominal operations, and the rule was, whenever in doubt, to use drainage. The general indications were: *First,* any soiling of the peritoneum from rupture of pus tubes or cysts; *Second,* oozing from raw surfaces; *Third,* after most cases of intestinal suture; *Fourth,* when there was persistent capillary bleeding or when secondary hemorrhage was feared, particularly when large pedicles were tied with the Staffordshire knot, then in vogue; *Fifth,* in cases of tubercular peritonitis; *Sixth,* in diffuse peritonitis, septic or otherwise, on general principles, as applied to any wound the lips of which were closed.

It was known that the peritoneum was capable of absorbing immense quantities of fluids, and that it was able to manage, and finally dispose of, considerable sized masses of aseptic substances; but it was not known, and still is not by many, that if its function and vitality be not interfered with, it is capable of managing and disposing of considerable quantities of septic fluid also, as has been repeatedly observed clinically and proved experimentally. Of course, the variety and virulence of the micro-organism has much to do with this. There are many cases of peritonitis that are rapidly fatal; but many more will recover if not drained than if they are. Of this, I shall speak more fully later on. I would like to consider these indications for drainage in order, and speak finally of its use when peritonitis is already more or less advanced.

First, let us consider those cases in which there has been actual soiling of more or less of the peritoneum by fluids from cysts, old pus tubes, etc. In the first place, the contents of these pus tubes is almost always sterile. For the past five years I have had cultures and cover glass preparations made of all such fluids, and while often micro-

*Read at the Thirty-third Annual Meeting of the State Society, Santa Barbara, April 21–23, 1903.