mit the necessary power; but by pivoting the whole frame of the dynamo in trunnions very smooth driving is obtained, with comparatively small tension in the belt and little strain on the spindle. Our illustration shows a hole cut in the floor, into which is placed a fixed cradle having bearings for the trunnions, T. The dynamo, D, is mounted in the cradle, C, hanging from these trunnions, and slots are provided in the cradle for shifting the dynamo in a direction at right angles to the axis of the trunnions, so that the weight can be more or less overhung on the pulley side, by which means the initial tension of the belt is adjusted. The dynamo is not placed plumb under the center of the fly wheel, but to one side of the center line, so that the tight side of the belt forms a greater angle with the horizontal than the slack side. By suitably selecting the distance from the center line, the component of the two tensions in the belt can be made to pass vertically upward through the center of the pulley, thus relieving the bearing of the weight of the pulley and of that of a portion of the armature. An extra heavy pulley is recommended for this type of gear, and if it should be found impossible to put sufficient weight in the pulley, the dynamo can be shifted on its cradle in the manner above alluded to, so as to bring additional weight upon the belt. A gear of this description has been running for some time with perfect success in the laboratory of the City and Guilds Central Institute.—*Industries.*

MICROSCOPICAL RESEARCHES INTO THE CAUSE, ORIGIN, AND PROPAGATION OF DIPHTHERIA.

By Dr. CARL BUNSEN, Galesville, Wisconsin.

YEARS have passed since I began to study the microscopical causes of diseases, and my thoughts have been engaged ever since in finding the cause of diphtheria. We know of many instances where a loving mother, by giving the parting kiss to the little darling just breathing its last upon earth, has taken the deadly contagion into her own system, thus innocently helping to spread a destructive epidemic.

When we examine the throat of a child that has died of diphtheria, we are at a loss to account for the cause, as we do not find anything except perhaps little, shining, transparent dots in the muscular layers below the epithelium.

We find, further, that the epithelium of the throat has been partly destroyed. Again, in other places where the epithelium has not been destroyed, the epithelial cells are transformed into a leathery, tough-looking mass, that is closely dotted with the same kind of globular bodies, scarcely perceptible through a powerful microscope.

Microscopy teaches us that these are the microbes or micrococci of a parasite; but with all our advanced knowledge, we are not able to produce any propagation of these microbes. We shall see if we can find the reason for this.

If a child dies of diphtheria croup, and we examine the bronchial tubes and the trachea, we find the same closed, but not with a tough mucus, but with the same mass of opaque, epithelial cells, which often reach into the smaller tubes, so as to prevent respiration, thus producing asphyxia.

It is not my purpose to describe how the diphtheria dead look after death, as the reader can find this in books on pathology, which will nevertheless give him no sure microscopical cause.

I, therefore, concluded to make my experiments (the description of which will follow) upon living animals. Vivisection is the only means to study the causes of diseases. By examining a cadaver, we may learn what destructive changes have occurred, but we are in most cases ignorant as to the causes of the same. These little microscopical causes that have produced these destructive effects usually fall to pieces as soon as the supply of blood and animal heat ceases, and leave perhaps only their immature microbes behind. If we consider that diphtheria is physiologically and pathologically closely connected with the so-called *specific fevers*, we may learn from the observations of the microscopical anatomy of the local lesion that this lesion is chiefly connected with infiltration of the affected tissue with a granular substance, which, by closer inspection, is found to consist of microbes.

How these microbes get there is a matter of speculation, as these minute bodies can be detected in all stages of disease and in all affected tissues.

We find them in the smallest and most superficial spots. We find them deposited in millions in the tissues, and we notice, further, that the greater the quantity of micrococci present, the greater will be the toxic affection. Diphtheria always commences with a severe catarrhus affection of the larynx, fauces, and, afterward, of the nares, and it was this affection which guided me in my examinations. This catarrh may last sometimes for weeks before we find that diphtheria has taken possession of the patient. Let us, therefore, study the nature of catarrh. We find in nature different kinds of cells that produce catarrh, and we must classify these cells as ferments. We shall only study the nature of two kinds, which will represent all the rest.

First, if an invasion of common, ordinary catarrh cells has begun, we find that the mucous membrane becomes covered in a few hours with millions of minute, round, globular bodies, which are fastened to the underlying epithelial cells by two minute, hook-like appendages or rootlets, being covered at the same time with mucus, which protects the cells against the influence of saliva. When we examine these little, round bodies on a *living* subject, we see that the inside of these cells contains a clear blastema. After a while, we see that upon the surface of these cells there appears a nucleus, and, just before bursting, we discover from three to five nucleoli.

These little cells will, during their growth and propagation, absorb the nitrogen and water at the expense of the mucus, which, in consequence of this process of fermentation, will be soon transformed, and can then no longer protect the little fermenting bodies against the ptyalin of the saliva, which soon puts a stop to their life, and the catarrh ceases, and the invaded parts become normal again. In those places which the saliva is not able to penetrate, the process of fermentation ceases on account of exhaustion, and the little cells will die.

Entirely different in its action is a cell of a catarrh which has its origin in the disease we call gonorrhea, the so-called syphilitic or gonorrheic catarrh. In the first stage of life we cannot microscopically discover a marked difference between the common catarrh cell and that of a gonorrheic origin. Their outer form is the same. Both kinds of cells are round, globular bodies, which are fastened between the rims and the walls of the underlying epithelial cells by two minute

about these catarrh cells. We shall return to them later.

I only wish to observe that each of these microbes will soon follow the same process of propagation, and we must not wonder that, in a couple of days, millions of these filaments are crossing and recrossing the whole mucous surface of the larynx, fauces, and nares. But these are not the only parts that will be infected. They will reach down into the pharynx and esophagus;

FIG. 1.—COMMON CATARRH CELLS.—a, ripe cells. b, ripe cell after the integument is rent, nucleus with nucleoli sitting on top of the blastema. c, ripe cell with nucleus bursted. The nucleoli are scattered in the neighborhood. d, ripe cell showing the harpoon-like antennae by which it is fastened in the rim of the wall of the epithelial cells.

FIG. 2.—GONORRHEIC CATARRH CELLS.—a, ripe cells. b, ripe cells with the integument rent, showing the nucleus surrounded and protected by the blastema. Inside of the nucleus are the double (identical) nucleoli visible. c, ripe cell, with nucleoli penetrating the nucleus and blastema. The center of the sporoides is protected by the surrounding nucleus and blastema. d, the protected inner end or root of the parasite, showing the forming micrococci or microbes. This root is never exposed until the process of growing and propagation ceases and when the integument falls to pieces.

FIG. 3.—a, parasite with parts of five nearly full-grown arms, full of nearly ripe microbes and micrococci, the roots still protected by nucleus and blastema of the mother cell. b, end part of an arm of a parasite. c, arms in the state of growing. Both b and c represent the parasite in an immature condition.

little hook-like appendages, which adhere so firmly in their places that they usually remain in their fastening places, and tear from the cell body if we try to remove these cells for the purpose of examination and propagation. When we look at these transparent bodies uninterruptedly (that is, during the life of the subject upon which they grow and propagate), we shall soon detect that the nucleus of these latter cells is suspended and surrounded by the blastema, and that just before bursting, double nucleoli can be seen, usually three to five appearing in one nucleus. As soon as the outer integument is rent, we notice that the blastema is forming a protecting cover over the nucleus, which resists the influence of the ptyalin of the saliva, which latter ferment does not produce any change in such kind of blastema. If we make our examinations with a powerful microscope, and during the lifetime of the animal, where a constant and fresh supply of lymph and blood is circulating for the support of the epithelial cells, we shall soon discover that the double nucleoli begin to lengthen at one end and to penetrate through the nucleus and blastema. Being fed on the liquid remains of the mother cell, it simply follows that the lengthened nucleoli are of the same nature.

If we can keep the animal that we use for purposes of experiment alive for a few hours, and we follow the growth of this medusa (for I am inclined to call it by this name), we shall soon see that these filaments are lengthening rapidly with a jerking motion. In the course of about three hours, we shall see that these filaments stop growing, and that they grow larger around, and soon we may notice that minute light gray dots appear inside of the now transparent integument, which continue to grow larger and larger until the integument bursts. With my micrometer I have calculated that there are sometimes from 200 to 300 little dots in one integument. After the integument has fallen to pieces, the little microbes will pierce the

but it seems that, below the epiglottis, they do not prosper, as these filaments do not grow much and their propagation is much retarded and disturbed. Their *nidus* ceases below the epiglottis. The reader may wonder why I have been so very particular in describing the cause and progress of this latter catarrh. I shall simply state here that diphtheria and this gonorrheic catarrh are closely connected, that diphtheria is only the *second state* of this catarrh, as I am well able to prove through my experiments on live rats. I know that the medical profession will attack me on this simple statement, but I am prepared to defend what I am asserting and writing. I have not referred to any books, and I openly confess that I have stud-

Piece of an epithelium covered with gonorrheic catarrh cells in the state of growing and propagation.

little hooklets in the wall of the epithelial cells and begin life on their own responsibility. Having been originated and fed on a poisonous mother germ, we can easily comprehend why such a catarrh will never stop; and having transformed the mucus into a thin corroding fluid that is unable to protect the underlying epithelial cells, the parasites will then attack the epithelium, and will destroy the same. So much, then,

2420 Diam.

2420 Diam.

The same as Fig. IV.

ously avoided reading any publication on diphtheria, as I do not wish to argue with books and references. It is only the facts that the eye can see that I can accept, and not theories. I am well able to show and prove that which I have seen.

Let me now relate how I came to the idea to attribute the cause of diphtheria to a gonorrheic origin.

The first case of the disease that I saw here, in America, originated from a young man, who came to my office suffering with gonorrhea. There were no cases

of diphtheria in our neighborhood. But after I had had him ten days under my treatment, his four smaller brothers and two sisters, also his mother, were infected through him with a bad catarrh. The mother and one little boy (the latter sleeping with the sick young man) became blind through a blennorrhoea, and the whole house came down with diphtheria, after having had this catarrhus affection from eight to twelve days after first infection; but not alone that, the young man visited five other families, which he also infected with the bad catarrh, and which was followed, after an incubation of eight days, with diphtheria. But this is no proof, and I may openly confess that this was only a thought or theory—that is to suppose a connection between diseases apparently so different.

So following the usual book treatment recommended for diphtheria, I had the same success that the other physicians had, namely, that most of the patients died.

Not being satisfied with the result of my treatment, I inquired and tried to find the cause through *post-mortem* examination, but with *no result*. Thus I came back to the idea of finding the cause of this constant catarrhus affection preceding every attack of diphtheria, which I considered a most essential step toward solving the problem. Here, in Wisconsin, we had formerly two regular epidemics every year—one in the spring, when our logging camps discharged their men and they returned to their homes.

Sometimes the diphtheria croup broke out in the camp, and they had to be closed on account of the scourge before the cutting of the logs was finished.

The second epidemic appeared in the fall, after the strolling harvest hands had left our vicinity. But this was more of a sporadic character, and has ceased entirely since self-binders have come into use. I have kept strict watch of these sporadic cases, and these latter ones are those which have helped me to get the first hint as to the cause. And further, I undertook a journey to the logging camps near Neillsville, and examined the inmates of each carefully, especially those where I found men suffering with a suspicious catarrh. In seven camps I found some cases, and could in each place trace without difficulty the origin to one or two men, who had come into the camp with a gonorrhoea. I predicted an outbreak of diphtheria in every one of these seven camps, and had the satisfaction of seeing five of them closed within three weeks on account of diphtheria. From the inmates of the other two camps I received intelligence that shortly after they reached their homes, after being dismissed, their families came down with diphtheria. The men had slept in the rooms with the persons infected with gonorrhoea and a severe catarrh, and had breathed the same atmosphere, used the same towels, etc., and carried the germs of the deadly foe, diphtheria, to their innocent families. I know that this idea will probably be attacked, but I can prove what I am writing. I have for years never lost a case of diphtheria if I was called before the croup had set in, and I can give the names of prominent physicians who will vouch for the truth of this statement. I shall relate one more case to support my position among the many cases that I could recount, and which would be pertinent illustrations. I was called seventeen miles from my residence to a family sick with the scourge. A few days later I was called to some families living seven miles farther, but in an entirely different direction from the first family, and I was called in on my way back to two more families living in two different valleys.

They informed me that they had neither visited nor seen each other for months, most of them being total strangers to each other; but that a young man, whose parents lived in the vicinity, had called on them (he being a great horse trader). I followed up his track, and found that he had been in every place a week or ten days before the diphtheria broke out. I had the satisfaction to see Mr. M. coming a few days later into my office, suffering with a severe gonorrhoea. Upon examination of his throat, I found it in a state of high catarrhus inflammation; he told me upon inquiry that about five days after the gonorrhoea had set in, he began to have trouble with his throat, and that he had

ments on live rats for the purpose of studying the nature of the disease germ. I am a great admirer of Louis Pasteur, and have adopted his method of investigation.

It is said that it is difficult to inoculate an animal with disease germs, and that diphtheria germs have never been successfully inoculated.

This is true; for every disease germ has its own *nidus* in which it propagates, and it would be a nonsensical task to inoculate diphtheria, even if we could get microbes without lacerating them; for whenever we try to remove a diphtheria germ, that is, a cell originating from a gonorrhoeic catarrh, we shall readily observe that the two hook-like antennæ with which it is fastened in the rim and walls of the epithelial cells will remain in their place, and the outer integument will become lacerated. Such a cell will *never grow*, and further, if we should try to remove an entire medusa,

Fig VIII

3250 Diam.

Gap in the epithelium filled with parasites forcing their arms below the epithelium.

and if we should lacerate any part of the filament, it will at the same moment lose its liquid contents, and the integument will disappear from our sight.

I have never been able to get either a *cell* or a *medusa* entire so as to mount it. I have never seen diphtheria inoculated in the common way able to propagate. We have to consider from the beginning that the cause of diphtheria has to be studied during life, for, as I stated, the germs will fall to pieces as soon as the bodily warmth and the supply of blood and lymph ceases.

I began, therefore, to feed thirteen rats with soft bread and butter seasoned with the sputum and nasal exudation of a child dying with the scourge. These animals were separated and carefully fed upon soft food. I also brushed direct from the dying child's mouth the saliva into the open mouth of some of the rats, depositing it as far back as possible. As I had already kept the animals for weeks in a very cool place and got their bodily warmth to about 99° or 100° Fahr., I concluded that the disease germs would find a congenial soil for propagation. I will state that I infected no animal which showed a higher or a lower degree of temperature, and I watched carefully that the warmth of the infected animals remained at 99° or 100° Fahr. In the course of four days the rats seemed to lose their appetite, and would eat only sparingly of the food. After the lapse of three more days some of them showed symptoms of a severe catarrhus affection, and I noticed that the rats were running around with regular snout noses. Then I concluded to begin my investigations. I selected one specimen that I considered well advanced. I put the rat under the influence of chloroform, and with a sharp knife I carefully opened the esophagus and pharynx as far as the epiglottis. This operation I had tried on dozens of rats previously, so as to become well accustomed to the operation. Having fastened the separated parts with small, sharp-pointed tenacula to the outside of the clamp with which I held the rat securely and firmly in its place, I brought my microscope, a large Nachez instrument, into position and directed a reflector to the cut, so as to illuminate the exposed parts. First, I directed my instrument to a place just below the epiglottis, and I saw a fine film, composed of thousands of fine silky threads of light gray color, most of them in a jerking motion, crossing and recrossing each other in every direction, some of them lying apparently motionless. My attention was directed especially toward these motionless threads, which by closer inspection appeared to become dotted lengthways with minute dots. After a while I noticed that the thread had swollen, and suddenly the integument had burst, leaving only the little gray dots in their former place.

But just at the most interesting point, my rat expired, and three minutes later the jerking motion of the filaments had ceased, and they began to disappear, and a few minutes later there was nothing more left but a gum-like substance (probably protoplasm) in which the shining little dots were visiblenow and then. Opening the mouth by cutting through the masseter, and laying the pharynx open, I could not see a trace of diphtheria, that is, of the identical white superficial false membranes, and I considered myself upon the wrong track. But in order to be sure of that, I took the second rat, chloroformed it and fastened it securely. Then I took more pains in preventing loss of blood, and made my incision of the esophagus not so far below as in the case of the first rat, and then I made a cross cut below the epiglottis, so as to get a good view. This time I did not sever any of the larger branches of the jugularis and carotid, and the animal breathed slowly, and whenever it showed symptoms of reviving, I gave it a little more of the anæsthetic, so as to keep it under the *first* influence.

My microscope was now directed to a place back of the epiglottis, but I saw nothing but filaments and dots. I took, therefore, a glass of 3,250 diameters and directed it at the dots, which became visible as round, globular cells, securely fastened to the underlying epithelial cells by two little harpoon-like rootlets, which

had always fastened between the walls of two epithelial cells. Keeping the instrument directed on them, I saw that these parasitical cells began to grow, and after a while I could discern a nucleus suspended in and surrounded by a blastema. In a short time I had the satisfaction to see these globes ripening, as there appeared inside the nucleus from four to five *double* nucleoli, that is, nucleoli with *two* ends.

I did not have to wait long, when one of these globes burst, and I perceived that the blastema protected the nucleus, and at the same time the double nucleoli lengthened at one end and perforated the nucleus, but their other ends I could see joining into a kind of a knob, resembling a rose.

The parasite now began to come forth from under the blastema, constantly spreading its arms with a jerking motion, the whole looking like a medusa. Having watched these little arms continually growing, I could plainly see that all the filaments that I perceived in the first rat were nothing but the arms of the same kind of parasites. But in moving my instrument all over the exposed pharynx, I could not see any white superficial false membranes, which I was most anxious to find. But no trace of them was to be seen.

Disgusted, I was about ready to kill the animal, when, by accident, I looked sideways where the epithelial cells had been separated at the time I opened the esophagus, and there I perceived a white rim at the cut. As I had no idea as to the origin of this white seam, I directed my instrument on the same.

Then I could plainly see that the filaments of the parasite had grown over the cut, and were already reaching below the epithelial cells, and were spreading with great rapidity. The death of the rat put a stop to my further investigation, but one thought struck me. Is it possible that the filaments will gradually weaken the epithelial layers through their constant growth, so as to force a gap in the close-fitting cells, from which they will penetrate and spread under the cells, thus absorbing and appropriating their nutritious supply for their own use? Is this, therefore, the cause of the white spots? Then I took five rats, chloroformed them, and scraped and scratched with the point of a small knife the fauces and also the roots of their tongues, some deep enough so as to draw blood, and of others I only lacerated the epithelium lightly. After this operation I removed them to their former compartments, where they soon became as lively as ever.

Six hours later I brought one specimen under the influence of chloroform, and opened its pharynx, as before described.

When my instrument was directed upon the lacerated places, I found that where the scratches were only light, there was a greater accumulation of parasitical cells, but no white superficial false membranes, and that where the incisions were deep enough, so as to separate the epithelial cells, the filament had already penetrated through the artificial gap below, and that the parasites were propagating and absorbing the nutritious supply and spreading in every direction under the epithelial cells, which became weaker and weaker, until they twined into an opaque, shriveled substance, a *secure cover for the parasite*.

I opened two more rats, and saw the same phenomenon, for wherever the lacerations were deep enough, so as to destroy the cohesion of the cell walls, the parasites had forced their filaments and the identical white superficial false membranes were formed, as described above. In places where the laceration was only slight, the great accumulation of parasitical cells soon weakened the cohesion of the epithelial cells, which soon gave way to the penetrating filaments, and I could plainly see that from these places the plaques (superficial false membranes) spread farther and farther. Being now satisfied as to how the diphtheria plaques (superficial false membranes) formed, I left the two other rats to their fate. One of them died fifty-two hours after laceration, and the other fifty-nine hours after.

When I opened the first one, I found the trachea filled with the white plaques (superficial false membranes). Even the bronchial tubes were filled with them, and the animal had died of asphyxia.

Fig VII

3250 Diam

The same parasites magnified 3,250 diameters.

been infected with his disease in Muscatine, Iowa. As I was called too late to one of the latter families, they lost two children of diphtheria croup, which had set in before I was called.

Of the other seventeen patients I did not lose a single case. If we look over the reports of the armies in Europe, we may easily learn that, whenever a soldier is found out to be suffering with gonorrhoea, he is not allowed in a ward where there are wounded men, as, in spite of all precautions, these wounds will not heal on account of the so-called false membranes forming.

Diphtheria always follows an army, as the reports of the Franco-Prussian war show, and some of the most promising young men lost their lives in consequence of the infection.

But let me now direct your attention to the proofs which I am going to give you. I mean my experi-

Fig IX.

3250 Diam

Same parasites below the epithelium.

Closer examination revealed the fact that the inside membrane was pressed together through an accumulation of a gum-like substance, through which thousands of little microbes were visible. The other rat had died of the asphyxia; but as the animals had been deeply lacerated, I found the root of the tongue and a part of the larynx a mass of foul-smelling sores.

Having described to you truthfully the result of my experiments on live rats, infected through the exudation and sputum of a child in the last state of diphtheria, and having convinced myself concerning the nature of the identical germ, I came back to the first visible symptom, the catarrh, concerning the origin of which I was by no means satisfied.

I know by the great many cases of gonorrhoea which I have treated during my life time, that not every person needs to come down with a gonorrhoeic catarrh,

and, also, that persons exposed to gonorrhea are not always forced to take the infection.

The reason of that is easily explained, when we consider the nature of a contagion-germ, and by comparing the so called specific fevers, whose origin is only to be found in the invasion and propagation of their identical contagion-germs. Every contagion-germ has its *nidus* in which it may propagate. We do not find any germ of the disease which it represents in any other part of the body except in the *nidus*, where it finds the necessary congenial supply of nutriment for life and propagation.

If a person has been infected but once, it is not likely that he will have a repetition of the same kind of a contagious disease, as the first invasion usually uses up all possible nutrition and exhausts its reproduction completely. This is plainly to be seen in small-pox or scarlet fever, which, as a rule, are never repeated after the first invasion; but if this invasion has been incomplete, then the nutritious supply for the disease germs will be replenished, and there follows a second invasion, however, not severe.

Gonorrhea is usually stopped through medical interference before its cells have exhausted their supply of nutrition, and this is the cause of its repetition.

The knowledge of the causes of the specific fevers led me to these investigations as to the relation between gonorrhea and the diphtheria catarrh. It is a known fact that a gonorrheic person will not always get a nasal or pharyngeal catarrh, even if he should bring the escaping infectious discharges in contact with the tongue or upon the nasal mucosa. The reason for this is to be found in the lack of congenial nutrition.

Such persons are not carriers of diphtheria. But why is it that the contagium-germs of gonorrhea cannot produce diphtheria in another person? Why do diphtheria microbes produce no chancre?

These are questions that I shall be obliged to answer.

The first question is a difficult one to answer, but experiments upon live rats will easily explain it. I have never been able to produce in them any symptoms of catarrh, if I fed them on fresh gonorrheic discharges. Vivisection never showed any germs or filaments. To ascertain the cause of this, I determined to observe what persons were infected with the identical catarrh at the time of a gonorrhea, and the fact was revealed, namely, that persons who had already previously had one or more infections were free from catarrh, provided they had had the identical catarrh once at the time of a previous infection.

Experiments upon rats, which followed this observation, proved that only animals infected with the catarrhus exudations of the throat of gonorrheic persons showed by vivisection the identical double nucleoli and filaments, and that animals fed upon the exudations of gonorrhea (not catarrh) were devoid of the germs, and showed only new mucous formation and no filaments—no trace of the cells with the double nucleoli.

From this it follows that the germs must first propagate upon the epithelial cells of the throat of a person at the time of a gonorrhea, and must produce the identical catarrh before they can be transferred from person to person, and that persons whose gonorrheic catarrh stopped on account of an exhaustion of the necessary supply of nutrition will never come down with diphtheria a second time.

The same is the case with persons who are naturally devoid of the needed supply for the propagation of the germs. Consequently, the disease germ must find the necessities for life and propagation upon the epithelium, and diphtheria can only form after the cohesion of the epithelium cells has been destroyed through an overtaxation of their supplying power of nutrition.

If the filaments, as we have seen, have penetrated below the epithelial cells, and are spreading and absorbing all nutrition for themselves, the epithelium will die and will form a protection for the parasites which is not easily removed.

This is the second state of the invasion, and consequently we must call the appearance of the white plaques (superficial false membranes—the only true proof of diphtheria) the secondary state.

The second question, why diphtheria germs produce no chancre, needs no explanation, as the places where we inoculate the disease germs are not their *nidus*, and are devoid of the nutrition to supply their life.

Having stated as plainly as possible what I experienced through a series of experiments, I hope that others who are younger and more skilled than I am will follow up the suggestions that I have given, as we can only by personal investigation trace the subtle causes of diseases.

I have used no book for references, nor can I be satisfied with them, as an answer and objection to my experiments. In the solution of scientific questions, *proofs*, and not theories, are required.

It is needless to state that I continued, after having found the cause of diphtheria, my experiments to find the method of destroying it, and my treatment is based upon these latter experiments. The result of these experiments has been eminently satisfactory, as I have never lost a case of diphtheria where I was called in time, and in case the people strictly followed out my directions, and hundreds of people can and will testify to the truth of my statement.

[JOURNAL OF COMPARATIVE MEDICINE AND SURGERY.]

DANIEL E. SALMON, D.V.M.,

CHIEF OF THE BUREAU OF ANIMAL INDUSTRY.

WE present the portrait of Dr. D. E. Salmon, the chief of the United States Bureau of Animal Industry, who has been connected with the official veterinary work of this country since Congress first instituted investigations of contagious animal diseases, as provided by the appropriation of 1878.

Dr. Salmon was born in Morris County, New Jersey, July 23, 1850. He entered Cornell University at its opening in 1868, being a member of its first freshman class. Here he became acquainted with Prof. James Law, who had just come to America to fill the chair of veterinary science at this new institution. At that time there were few who realized the importance of veterinary instruction, or who could see any reason for including such studies among the branches taught by our higher institutions of learning.

It was the aim of Ezra Cornell, however, "to found an institution where any person could find instruction

in any study;" and with an admirable appreciation of the value of knowledge in regard to the diseases of animals, he determined that veterinary medicine and surgery should be well represented in the faculty of the university which was to bear his name.

Very early in his course at Cornell the subject of our sketch became impressed with the importance of the position and work which were soon to be assumed in this country by educated veterinarians. Even then the flocks and herds of the land had multiplied until their numbers were enormous. And many of our States were without a single veterinarian competent to diagnose the most important animal plagues, much less to give intelligent advice for their control.

Massachusetts had succeeded only two years before in ridding that State, after a six years' struggle, of an outbreak of pleuro-pneumonia which might have been stamped out in as many months. The investigations just made by Prof. Gamgee, under the direction of the United States Department of Agriculture, had shown the existence of contagious pleuro-pneumonia from Long Island, N. Y., on the north to the District of Columbia on the south. The most extensive outbreak of Texas fever which this country has ever known, and which resulted from the wide distribution of Southern cattle over the Northern States in 1868, was then in progress, and was attracting attention everywhere. Hog cholera, although considerably disseminated before the war, was lost sight of for a few years on account of the stirring events of the times, but in 1868 it was again attracting the attention of writers on agricultural and sanitary subjects. These diseases, widely distributed in our own land, and threatening our food supply; with foot and mouth disease and rinderpest ravaging the herds of Europe and liable to be imported at any time, gave unmistakable evidence that our national and State governments would soon appreciate the value of veterinary science.

Dr. Salmon graduated at Cornell in 1875 with the degree of bachelor of veterinary science. The same year he visited Europe, and after remaining two terms at the French veterinary school at Alfort returned to America and began the practice of his profession in Newark, N. J. In 1875, on account of impaired health, he went to Asheville, N. C., for the benefit of the Southern mountain climate. In 1876 Cornell gave him the advanced degree of doctor of veterinary medicine. In 1877 we find him non-resident lecturer on veterinary science in the University of Georgia.

The appropriation by Congress in 1878 of \$10,000 for the investigation of animal diseases led to the appointment of a number of veterinarians, including Dr. Salmon, who were directed to devote two months to the study of swine diseases. The reports of these investigations were published by the department of agriculture in a special volume in 1879. It was not expected that this brief investigation would solve all the problems connected with hog cholera, but it was hoped that certain leading questions, such as its nature, cause, and identity in different parts of the country would be settled, and that some practical measures of prevention would be suggested. To this extent the inquiry was successful, and it was demonstrated that there was a specific contagious disease of swine widely disseminated over the country, and that prevention could only be accomplished by controlling the spread of the contagion and destroying it wherever found.

The State of New York attempted to stamp out contagious pleuro-pneumonia in 1879, and Dr. Salmon was appointed on the veterinary staff as an assistant of Prof. Law. The spring and summer were devoted to this work, but with the exhaustion of the appropriation in the autumn, he accepted a commission from

the United States Department of Agriculture to investigate animal diseases in the Southern States, and was instructed to make a specialty of Texas or Spanish fever. He then began that extensive series of studies of cattle diseases in the South which demonstrated the identity of the "distemper" of Virginia and North Carolina with the "murrain" of Georgia, Alabama, Mississippi, and Tennessee, and with the Texas fever of the Northern and Western States. During the investigation he directed a careful survey of the whole country, from the Atlantic seaboard in Virginia to the Rio Grande river, accurately outlining the district permanently infected with this contagion, and showing that Northern cattle taken into that district contracted the same disease that was disseminated by Texas and other Southern cattle when taken North.

In 1880 he began the investigation of fowl cholera, demonstrating that the germ found in that disease was its essential cause, that it could be made to multiply locally and act as a vaccine by simply diluting the virus to a sufficient degree.

He also at this time formulated a theory of immunity from contagious diseases, based upon numerous experiments, in which were foreshadowed a new method of prevention through the use of chemical substances formed during the growth of the specific germs. This method has since been satisfactorily demonstrated by experiments made under his direction, and he is still at work upon the details which must be learned before it is put into practical operation.

Early in 1883 he was called to Washington by Commissioner Loring to establish a veterinary division in the Department of Agriculture. In doing this a plan was adopted for an extensive investigation of animal diseases, a laboratory for histological and bacteriological research was fitted up, and seven acres of land near the city were rented for an experimental station. Within a year Congress passed an act establishing the

Bureau of Animal Industry, and Dr. Salmon was at once appointed chief of this bureau, a position which he still holds.

Since he has been in the Department of Agriculture a large number of investigations have been made under his direction. In 1883 he was appointed by President Arthur a member of a commission to investigate the quality of our pork products and the prevalence of trichiniasis, and his report on this subject has been received without question.

In 1884 he investigated the supposed outbreak of foot and mouth disease in Kansas, Missouri, and Illinois, and allayed apprehensions by deciding it to be ergotism. In the same year he discovered and traced out an outbreak of pleuro-pneumonia which extended to Ohio, Illinois, Kentucky, and Missouri, and succeeded by co-operating with State authorities in securing its extermination wherever found. It has since been discovered, however, that it had crept into Chicago unobserved, where it smoldered for two years and was brought to light by the State veterinarian in September, 1886. The Bureau of Animal Industry now has a force of inspectors engaged there for its suppression.

Since its organization the Bureau of Animal Industry has made an inspection of the district from Long Island, New York, to Virginia, to determine the extent and prevalence of pleuro-pneumonia. In August, 1886, an agreement of co-operation for stamping out this disease was made with the State authorities of Maryland, and the slaughter of diseased animals has been going steadily on. In March of this year, an appropriation of five hundred thousand dollars was made to the bureau, and the work extended to the slaughter of exposed as well as of diseased animals.

New and efficient rules and regulations for co-opera-