

this country has not been very long, although it already includes several generations. Such as it is, some of its results are:—1. In no children or descendants born in Minnesota of lepers—there are great-grandchildren—has there been any sign of the disease discovered, although under frequent observation. 2. Up to date no leper has been born in Minnesota. 3. In many cases the disease was not recognised for a long time after the arrival of the persons affected in this country—how long it is difficult to state, as the first symptoms are obscure, not noticed, or misunderstood; but in every single instance the leper has some time had his home in some place in the old country where the disease was endemic, while in but a single case has the possibility of infection been pointed out, and that came from a locality in the old country that has been for years a nest of leprosy. These facts, as far as they go, seem to suggest that the disease in this country is not so easily acquired as in some other countries. Looking for the cause, some points present themselves for consideration.

1. New houses with new furniture and furnishings in a new country harbour no sources of infection, and the better economical condition promotes the sense of need for cleanliness both for health and comfort,—a cleanliness which seems in leprosy to be the main reliance against contagion. 2. The commonly dry and always windy climate of this great inland plateau, with its great and sudden changes of temperature, open as it is towards the Gulf of Mexico and towards the North Pole, may perhaps make it more difficult for the *materia peccans* to fix itself on persons and things. The hot summer that opens up the pores of the skin and drains the system that way, and the cold stormy winters acting on the body somewhat differently, may have influence in that direction. That the climate must have some influence in preventing the spread of the disease seems yet more probable when we remember that the early settlers, often with a large family of children, lived for a long time in small, close, and badly ventilated log houses, closer even than they were accustomed to in their old homes. Even if they did not bring with them such sources of contagion as unclean houses and old furniture, they at least had for some time old clothes, so that it would be a wonder if old and young could have observed the cleanliness necessary to prevent contagion if the effect of climate had not been to make contagion more difficult. It seems certain that the disease, once established, runs its regular course here as elsewhere, perhaps a little slower. 3. The change in the physical constitution of people who have lived here some time, the effect of acclimatisation and of other influences, may make the individual less susceptible to contagion. In 1888 Dr. G. Armauer Hansen of Bergen, Norway, the discoverer of the bacillus lepræ, came to this country to study leprosy in the immigrated Norwegians and their descendants. He had taken the position in 1874 that contagion is the only source at present of leprosy, and that the disease is not hereditary. He gives the results of his investigations.²

"I cannot here relate all my observations in detail. I will only tell what I have found in regard to the occurrence, or rather the disappearance, of lepra in America (N.W. States). Of about 160 lepers who have immigrated into the three States named (Wisconsin, Iowa, Minnesota), thirteen are alive, whom I have seen myself, and perhaps three or four more. All the others are dead. Of all the descendants of lepers (and that includes the great-grandchildren of some of them), not a single one has become leprous. This is, in short, the result of my investigations."

SOME RECENT MODIFICATIONS IN OUR VIEWS OF ENTERIC FEVER AND ITS TREATMENT.¹

By M. A. BOYD, M.D., F.R.C.S.I., M.R.C.P.I.,
PHYSICIAN TO THE MATER MISERICORDIÆ HOSPITAL, DUBLIN.

SINCE Murchison wrote his classic treatise on Enteric Fever we have scarcely added anything clinically to the accurate and scientific description he gave of the disease. He covered in that description almost the entire ground in connexion with the disease and its history, and in the

closeness of his reasoning as to its etiology he foreshadowed all that bacteriology has since discovered regarding it. In fact, it is only in its bacteriological aspect that any additions have been made to the subject, and it is from this side alone that any additional knowledge is likely to come which can finally determine the mode of its origin. The additional bacteriological data, however, we now possess have considerably narrowed the issues and enabled us to regard the disease from the standpoint of being an acute infective disease, which heretofore it was not considered, in which, like phthisis and pneumonia, a special bacillus plays the important part. Since that bacillus was discovered by Koch and Eberth, and its peculiarities and mode of growth studied, more especially by the latter observer, our views as to the etiology of the disease have considerably changed, and the time has, I think, now come when, taking advantage of the experiments of various other bacteriologists in addition, we may form more definite ideas as to its mode of propagation, to its prevalence at particular seasons, and the causation of the symptoms present in its various stages, as well as to suggest some rational means for treating it.

I shall first refer to the typhoid bacillus and its discovery. In 1880 Koch and Eberth almost simultaneously discovered in the intestines, the mesenteric glands, and lymphatics, and especially in the spleen, of patients dying of enteric fever a bacillus which, though frequently found in the intestines as the common bacterium termo, assumed at particular seasons, or owing to some alteration in the normal vital resistance of the individual, an acutely infective process, infiltrating the adenoid tissue and lymphatics of the intestine, accompanied by the chain of febrile phenomena we designate typhoid or enteric fever. Owing to the difficulty of finding a suitable nutrient medium on which to grow it outside of the body, this bacillus could not be differentiated from other bacteria inhabiting the alimentary canal, until Koch succeeded in isolating and growing it on dry gelatine plates. Since then all pathologists are familiar with its appearance and mode of growth. So far all experiments have failed in producing the disease from these cultivations by inoculation; but bearing in mind that it grows both as an aerophyte and as an anaerophyte, it may be innocuous in the former condition and infectious in the latter when in the alimentary canal, when the vital resistance of the tissues in that situation may be from some reason altered. What may produce these alterations I shall allude to presently. Gaffky, in his observations on this bacillus, has given us some valuable information as to its situation and growth. It is one of the few bacilli found to develop freely in water, and it grows abundantly in milk. He also found it in the soil through which water percolates, and it grew freely in all albuminous media. He also found it more abundant in all these media in the autumn than at any other season—a fact of considerable significance. If, however, this bacillus is found so frequently in the food we eat, the water we drink, and in our intestines, how, we may ask, is it that it does not infect the intestinal glands when present and produce enteric fever in every case? For we must all take it in at some time or another, if it is not already present in our intestines. With this question I may link two others: Why is it we do not suffer from pneumonia constantly, when we always carry about with us the germs of the disease in Fraenkel's diplococci? Or why not frequently suffer from circumscribed or diffuse suppurations, when the micrococci that produce them are frequently present in our blood and tissues? Why we do not recent investigations in bacteriology have made clear. Bacilli or micrococci are in themselves harmless, either in the blood or tissues, until the vital resistance of some tissue is lowered, from either functional alteration or injury, when they readily find a suitable soil in it on which to grow and multiply. It is by this growth and the chemical products generated during it that the mischief is produced, and the poisoning of the tissues around that are most susceptible to its action afford a further field for the growth of these micro-organisms.

Let me take first the experiments of a distinguished physiologist, and secondly the course of a fatal disease which clinically we are only lately becoming familiar with, to illustrate my meaning. Professor Kocher of Berne, in making experiments on animals by destructive injury to tissues down even to the marrow of bones with a hot iron, could not produce septic inflammation as long as the animal experimented on was healthy, but if he lowered the vitality

² Virchow's Archiv, Band cxiv, 1888.

¹ Read before the Medical Section of the Academy of Medicine in Ireland, Dec. 18th, 1891.

of it by feeding it on putrid matters, permitting thereby septic micrococci to enter its blood, a septic inflammation was at once produced. The other disease I have alluded to in illustration—namely, septic or suppurative endocarditis—we know arises from either the staphylococcus or streptococcus when present in the blood from any accidental cause finding a nidus in an inflamed endocardium or damaged valve, and the chemical product of its growth is then wafted in the blood current to set up mischief in other situations, where, from anatomical causes or lowered vital resistance, the blood and tissues cannot overcome its invasion. The germs of typhoid fever, like all other septic germs, are now regarded as in themselves harmless as long as the tissues with which they are in contact are healthy, else how can we explain the immunity from the disease that exists in healthy individuals who constantly either receive them through food or drink into the alimentary canal, or have them as a normal and constant resident in that channel?

This brings me to the vexed question of the etiology of the disease. If we take it for granted that the bacillus of Eberth is by its infiltration of the glandular tissue of the intestine the cause of all the mischief (and apart from the absence of the inoculation evidence most pathologists are agreed that this is so), what are the conditions that favour or produce its acute infection of those glands? We have first the evidence that this bacillus is found growing most luxuriantly—and we must presume consequently more virulent, and more capable of making a vigorous battle for its existence—in the autumn. But it must find the tissues with which it comes in contact in a weakened condition to get the upper hand in the struggle. Are the intestines at this period of the year in a more weakly condition than at any other, and, if so, from what cause? I think we can answer that question in the affirmative. Most of us are familiar with the gastro-intestinal troubles that are characteristic of the early autumn months, the gastro-intestinal catarrh especially, the catarrhal or autumnal diarrhoea, and the frequency of so-called bilious attacks at this season. How these catarrhs are produced we have a ready explanation in the rapid fall of temperature in the evenings after perhaps a warm mid-day, when the action of the skin is suddenly checked, and no additional precautions as to clothing adopted. All hospital physicians are familiar with the sudden onset of bronchial catarrhs at this season, also from the same cause, and the rapid filling of hospital wards with cases of asthma, emphysema, and fresh bronchopneumonia in patients the subjects of phthisis during the previous summer and spring. Now let us see if this gastro-enteric catarrh is a usual precursor of enteric fever. Murchison, with that acute power of observation which was characteristic of him, mentions it as a most usual symptom, preceding and accompanying the fever in its early stages; and he further adds that catarrhal diarrhoea was frequently present, preceding the fever, and that it was often difficult to say whether the disease would remain catarrhal diarrhoea or end in enteric fever. Now it seems to me that this catarrh, in addition to being brought about by atmospheric changes or food, may be produced also from the absorption of the chemical products of the typhoid bacilli growing on the intestinal contents, when present in large numbers, in either food or drink containing them; and that this promaine or toxine was only the weapon they used, as Professor Burdon-Sanderson expresses it, in their struggle for existence, to weaken the vital resistance of the tissues with which they were in contact, and make it fall an easy prey. Why, however, should the glandular tissue be the first overcome in this struggle? The reason seems to me obvious, looking at it from a pathological point of view. Adenoid tissue is endowed with a very poor vitality, and very little power of resistance, or of repair, when infiltrated or choked from any cause. We see this when it is attacked by a similar bacillus, the tubercular one, whose life history and the readiness with which it infiltrates and destroys glandular structures being somewhat similar to the typhoid one in many ways. The epithelial shedding and proliferation of the mucous membrane which take place must also weaken the defences against the bacillary invasion. The bacilli find the glandular tissue in a condition of derangement from the effects of this catarrh, and it becomes the centre of their habitation. That this invasion is sudden and followed by rapid changes in the glands there can be no doubt, Murchison having found infiltration and swelling of the glands in the case of a patient dying on the second day of the

disease, and other observers bear similar testimony. The entire process in connexion with the gland so far as the typhoid bacillus is concerned, from the time of invasion to death or disablement of the gland, is over in fourteen days—the normal time of enteric fever from a pathological point of view. After this period, however, a new set of enemies appear on the scene in the shape of the suppurative micrococci, which, forming colonies on the injured or necrotic tissues around the glands, begin to generate their peculiar toxins, producing the hectic character of the symptoms and temperature with which we are all familiar after the first fortnight of the disease; so that we must regard enteric fever as the result of the growth in the intestines of two sets of micrococci. The so-called typhoid bacilli producing the symptoms during the first fortnight, and the suppurative micrococci producing the characteristic symptoms of its further stages. That other micrococci, as well as these latter, occasionally infect the patient from the intestinal canal there can be no doubt, and I have at present under my care in the Mater Misericordiae Hospital a patient with a well-marked attack of erysipelas of the face in the fourth week of his enteric fever.

If recent bacteriological work has enabled us to grasp these facts with regard to the disease, we may ask ourselves what practical deductions can we draw from them so as to enable us to treat it. Looking at the disease as primarily a catarrhal one of the intestines, and secondarily as one of septic poisoning, our treatment resolves itself into suitable diet and antiseptics. We know already how all-important is the treatment of enteric fever by bland and unirritating diet, and such as will be mainly absorbed by the stomach and duodenum, leaving little to be dealt with by the lower part of the small intestine. The medicinal treatment of typhoid fever by antiseptics is now receiving that amount of attention which our more perfect knowledge of its bacteriological origin would suggest, and we see occasionally in the medical journals glowing accounts of the success of this method of treatment in the hands of some; while others confess it has not realised their expectations. As far as I am concerned, I have used this method of treatment for several years in both hospital and private practice, and have every reason to be satisfied with the results. I do not profess to believe that it will abort a case of typhoid fever when the characteristic fever has begun, but I do assert that it will prevent in the majority of cases the septicæmia—for it is nothing but septicæmia—which we have to deal with after the second week of the fever is passed. The typhoid bacillus has by this time done its work, so far as the intestinal glands are concerned, and hereafter we have only saprophytic bacteria and their effects to deal with. The characteristic hectic type of temperature during the third and subsequent weeks of enteric fever, such as we have in connexion with suppurating cavities in the lungs or elsewhere, shows this to be the case. In seeking for a suitable antiseptic for this purpose we must choose one which will fulfil the following objects. It must first exercise its effects in the intestinal canal, and not in the stomach. Its action must be thorough, antisepticising not alone the contents of the bowel, but it must permeate the intestinal wall as well, where septic micrococci may have already established themselves, and even enter the blood. To fulfil these conditions the form of antiseptic must be, in my opinion, a gaseous one. We know how readily the intestines absorb gases and pass them into the blood. The antiseptic I am in the habit of using is chlorine in an alkaline solution, as in this form it mingles best with the contents of the intestines, which in enteric fever exhibit a strongly alkaline reaction. This treatment is not original, as Murchison, who expresses himself in general as dissatisfied with antiseptics, speaks favourably of chlorine, and regarded it as an admirable method of treatment. He administered it in an acid solution, which in my experience is not so satisfactory. That this method of treatment produces a fall in the temperature and makes the type of the disease milder there can be no doubt, and in over a fourth of the cases when begun early brings the febrile process to an end about the fourteenth or sixteenth day. Murchison, in his careful statistics, found only seven cases out of 200 to terminate on the fourteenth day by the ordinary method of treatment, so the cessation of the fever by this method of treatment in such a large percentage must be more than a mere coincidence. I do not claim for it that it is the best form of intestinal antiseptic, as

more extended experience may enable me to procure a more beneficial one, and I hope on some future occasion to bring before the profession the results of my experiments on this subject in conjunction with our bacteriologist at the Mater Misericordiae Hospital. I have not in these notes entered the broader field of etiological theories by trying to explain how enteric fever arises through the medium of sewers, or sewer air, or why it is more prevalent among the wealthier classes than the poor, or whether it can arise as a miasma-producing infection through the air. These are matters that I did not intend should enter into the scope of my paper, as I have been content to regard the disease from the bacillary point of view alone, and to see in it sufficient explanation of all the group of phenomena which we call typhoid or enteric fever.

Dublin.

ATTEMPTED LITHOTRITY IN A BOY; SUPRA-PUBIC LITHOTOMY.

By W. T. CLEGG,

SENIOR ASSISTANT SURGEON TO THE LIVERPOOL HOSPITAL FOR SKIN DISEASES, ETC.

R. P—, aged ten, but small for his age, was sent to me, suffering from stone in the bladder, in October, 1891. When seven weeks old the patient had retention of urine, which required catheterisation, and he has had urinary trouble more or less ever since. He walks bent forwards and cautiously. He has retracted testicles, a long foreskin, and a prolapsed rectum. His clothing has a urinous odour. The urine is fetid. On Oct. 7th I attempted to pass a No. 10 Weiss completely fenestrated lithotrite, to ascertain the size of the urethra and of the calculus. The meatus was freely incised, but it would not pass. I then passed a No. 6 Weiss fenestrated lithotrite, which was gripped by the urethra all along, and a large calculus was discovered. Although under chloroform, violent spasmodic straining, amounting to emprosthotonus, even involving the sternomastoids, accompanied every movement of the instruments. On Oct. 8th the boy was placed thoroughly under the influence of chloroform by Dr. Stevenson. Dr. Tisdall assisted me, and Drs. Barnes, German, and Price, dropping in as the operation proceeded, were also kind enough to help in various ways. About two ounces of boiled water were injected into the bladder, and a No. 8 Weiss fenestrated lithotrite was passed carefully, but with some force, especially when passing the triangular ligament. The instrument was such a tight fit for the urethra that when moved about the penis moved with it, and this greatly hampered me. The stone would not fall into the jaws of the instrument. I soon found out that it retained a position on the left side of the bladder, and was always to be found there. The only manoeuvre that would enable me to grip it was with the lithotrite held vertically, its beak just inside the bladder and turned to my right. I seized the stone and carried it towards the centre of the bladder, locked the male blade, and screwed down upon some rather soft outer coating, coming to a stop as the hard stone was reached. I could trust the lithotrite, and so still screwed on with full force, when with a jolt the stone escaped. The length, weight, and shape of the stone and the small jaws of the instrument fully accounted for this. Three times this occurred. The phosphatic material that came away was mixed with offensive mucus; the urethra was small and in a state of spasm; the stone was large and hard, therefore I reluctantly gave up, thinking it more discreet to do supra-pubic lithotomy. We had no rectal bag, nor did I think one necessary. A light chair was inverted on the operating table, and the boy arranged along the back of it. His head only remained on the table, his knees were bent over the spindle joining the two back legs of the chair, and retained in that position by clove-hitch knots. A sponge was thrust into the rectum lest the contracted condition of the recti and bladder should prevent the viscera gravitating freely enough into the arch of the diaphragm to draw the bladder sufficiently out of the pelvis. This proved to be quite unnecessary. I cut down upon and into the bladder and extracted a stone two inches long and one and one-eighth inches broad, weighing 370 grains without the

débris. On first putting the finger into the bladder the impression was that the stone lay in a pouch owing to the contraction of the bladder walls around it, but after its removal no pouch remained. The bladder opening was anchored to the opening left in the linea alba so as to shut off the bladder discharge from the neighbouring tissue. On Friday, Oct. 16th, the boy passed urine through the urethra, and about four weeks later the abdominal wound had healed. The boy is now attending school, and there is a wonderful change in his appearance. The anchoring of the bladder to the linea alba has not given rise to any symptoms.

Remarks.—Lithotritry has been for some years a hobby of mine; I am accustomed to handle the weapons; therefore it will be granted, I think, that there was good cause for abandoning the attempt, and this makes the case worth reporting. Strange to say, lithotritry is as yet the rule only with the few; yet it is almost always applicable to old and young, male and female. It is, in my opinion, the safer proceeding where the kidneys are damaged. An elderly gentleman with damaged kidneys was to have been submitted to me for lithotritry. Meanwhile, he was advised to go elsewhere, was cut, and died the same evening. During the past two years two other similar cases have come to my knowledge. Lithotritry could not have met with worse success in these cases. The completely fenestrated lithotrite is the only form required, and numbers 6, 8, 10, A, B, and C, form a series sufficient to meet every requirement. I much prefer the wheel and button action—the Weiss-Thompson form to Bigelow's form of handle. I have used both, and found the bruising of the palm about the same in each; but with the Bigelow pattern the fluted surface of the lock mechanism makes the skin over the ends of the right index finger and thumb very tender when in use for an hour or more. There is more lurching, too, in working this lock. The jamming of the male blade, to which Mr. Cadge refers in his recent lectures, occurred to me on one occasion whilst sliding the male blade backwards and forwards to ascertain whether it was free after withdrawal from the bladder. I sent it up in its jammed condition to Messrs. Weiss and Son, and they wrote complaining that the instrument had been very badly treated. This remark gave me the clue to the cause. A visitor had taken up the instrument previously to the operation, and had allowed the male blade to fall on the floor. It had bent slightly upwards out of the straight and a small hard fragment of stone had worked under the heel of the male blade preventing any further movement backwards or forwards. The simplest and most convenient of aspirators is the latest Bigelow pattern (Fig. 1950 of Weiss' Catalogue). I do not know of a single fault in it. It has no trap; none is needed. Fragments in the glass bulb are very little affected by the currents passing overhead, and blood or mucus obscures the contents of the glass bulb last. This aspirator can be completely and readily filled from a jug by removing the funnel from the supply-pipe and attaching a foot or two of rubber tubing. Eighteen months ago, in conjunction with Dr. Price, I crushed a stone in the bladder of a seafaring man. I used on that occasion an excellent aspirator, but it required to be immersed in a bucketful of water to fill it, and there was not that quantity of hot water in the house. The filling and replenishing occupied more time in this case than the lithotritry. The patient, a man aged thirty-six, returned to his ship on the third day after the operation, and resumed his occupation without permission and without injury. It is a good plan always to have a second aspirator fitting the catheters. It saves waiting for the refilling. A friend operating on one occasion in private, an unfortunate movement of the patient threw the aspirator on to the floor, smashing the glass bulb. The evacuation had to be finished by plugging the opening which should have been occupied by the glass bulb.

I will conclude by mentioning two points that have impressed themselves upon me. In the first place, the slight symptoms often present with a large calculus; the largest I have met with occurred in a man suffering from strictured urethra, and gave rise to no special symptoms. In the second place, that calculi, however hard, owing to lamination, burst asunder in the jaws of the lithotrite much more readily than might be supposed, and seem to try the instrument less than an equal amount of red sandstone.

• Liverpool.