

NOTES AND REMARKS  
ON  
DIPHTHERIA  
By  
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In taking the subject of Diphtheria as a Thesis, I do not propose to write a treatise on the disease, but simply to lay before you some of my observations as a general practitioner, referring occasionally to those of others.

Etiology. Klebs-Loeffler bacillus is now acknowledged to be the direct cause of the disease. It produces the characteristic appearances on the surface on which it settles, and there secretes, as Dr Martin says, "a ferment which, absorbed into the body, produces, by acting on the proteids of the body, digestive products, chief of which are albumoses, active poisons, which are the cause of the symptoms produced." Non-pathogenic bacilli, morphologically the same, have, however, been found in healthy throats, and also in cases of tonsillitis, and ordinary sore throat, which must throw considerable doubt on a purely microscopical diagnosis of the disease. Professor Crookshank, in his text-book of bacteriology, 1896, says: "The value of a microscopical examination as an aid in the diagnosis of diphtheria has been considerably exaggerated, and

unless the bacillus when isolated is tested by inoculation the test may prove to be entirely fallacious."

Dr Kanthack and Mr White in St Bartholomew's Hospital Reports, 1895, say: "We must unreservedly consider every case of tonsillitis, laryngitis, or sore throat, in which diphtheria bacillus is found to be true diphtheria." I am inclined to agree with the latter until some more definite distinction between the pathogenic and non-pathogenic bacillus has been arrived at.

Professor Crookshank also says in his treatise: "Bacilli deprived of their virulence, and bacilli non-virulent cannot be made to assume virulent properties." This may be correct in laboratory experience at the present time, but is not proof that it may not occur in nature. Laboratory experience is sometimes opposed to what is found in practice.

The habitat of the bacillus outside the body is still a matter of conjecture, nevertheless the appearance of the disease under certain conditions suggests the possibility of its permanent

existence in the soil." Dr Thorne Thorne, in the Milroy Lectures, British Medical Journal, 1891, points out, that with improved water supply enteric fever has diminished, whilst with improved sewerage and water supply, diphtheria has increased.

Dr Klein states the bacillus dies in a few days in pure water, owing to want of sufficient nourishment. Carlo Reyes, in an article in the Annali di Igiene Sperimentale, November, 1895, says he found the bacilli living in moist earth after the lapse of 120 days, and suggested the possibility of their permanent existence and reproduction in soil charged with pabulum in the form of decomposing organic matter. Dr Gordon Sharp, in a paper read at the Annual Meeting of the British Medical Association, 1896, quotes the above, and further states he found microbes in every way resembling the Klebs Loeffler bacillus in two soils taken from what he calls diphtheria districts, and had his observations confirmed by an expert. He finishes his paper by stating: "A moist rather than a water-laden soil seems to be the home of the organism of diphtheria."

In a part of Essex where I practised some years ago (Manor Park), which is flat, damp, unsheltered, and freely exposed to the north-east winds, a large area of ground was purchased by a building company. In a short time about one hundred small undetached houses were built, suitable for well-to-do artizans and clerks. Being within easy reach of London by rail, they were quickly occupied. In the autumn following the first occupation of these houses and, while building operations were still going on, a mild epidemic of diphtheria appeared, confining itself to the newly occupied houses. The following autumn a severe epidemic of the disease took place which spread to a neighbouring district. For four or five successive years an epidemic occurred, varying in intensity. Since building operations have ceased, epidemics have not occurred, although sporadic cases are reported every year.

The soil originally of this estate was about 10 feet of sand and gravel, supported on clay. The gravel and sand to a depth of five or six feet had been dug out and sold, and was replaced by all sorts of rubbish and refuse. During the building of the

houses, the smell from the upturning of the soil was very noticeable. The reason given at the time for the outbreaks, was this smell.

One outbreak is particularly impressed on my mind, on account of the great fatality which occurred, all ages being attacked. This took place in the spring, after a long winter. During the previous autumn there had been a number of cases, but in the early part of the year the district was free. When building operations commenced in the spring, an epidemic of unusual severity broke out, the first cases being notified from the road in which building had commenced, ultimately spreading to the whole district. On looking back at these epidemics, I cannot but think if the bacillus is to be found in the soil, and if Carlo Reyes' theory as to the kind of soil most favourable for its existence is the correct one, this soil would be peculiarly suitable to it. Naturally a damp soil owing to the clay sub-soil, added on to this we have the organic matter present in the refuse and rubbish, the upturning of which might set the bacillus free if it were present.

It has been noted that the drainage of districts

has not had the marked effect in decreasing this disease that has occurred in other diseases, and it has suggested itself to me, that the partial drying of a previously wet soil might in some cases render it a more suitable pabulum for the development of the non-pathogenic organism into the pathogenic, (if such does take place) or a less virulent one into a more virulent one, whilst at the same time inhibiting or perhaps destroying the lives of other micro-organisms. If sanitation is responsible for any increase, we must go on with sanitation. John Stuart Mill said, the cure for the evils of education was more education, and the same may be said of sanitation.

Again the disease is more prevalent in the late autumn and early winter, the seasons when fires in the rooms are generally commenced. This will draw the subsoil and sewer air (the microbe is said to live in sewage) into defectively constructed houses, and find suitable throats for its development, on account of increase at this time of the year of sore throat, tonsillitis, scarlet-fever, and other diseases which attack the throat. Some observers lay great



stress upon defective sanitary arrangements in the causation of the disease, and from experience of many epidemics and sporadic cases, I feel convinced they do indirectly, and possibly in two ways:

1. By causing a condition of the body, and especially the throat, suitable for the growth of the bacillus.

2. By converting the non-pathogenic or less virulent organism present in the throat into the pathogenic or virulent, and so inducing a sporadic case, which may be the means of causing a widespread epidemic.

Mr Sidney Thomas Steele, speaking at the annual general meeting of the British Medical Association in 1891, after referring to causes, said: "Information was wanted as to the history of the first case in an epidemic, and under the Notification Act the cause of an outbreak would no doubt be elucidated."

A case pointing to deficient sanitary arrangements came under my notice lately.

A family recently removed to this neighbourhood (Teddington, Middlesex) ~~and~~ had repeated attacks of sore throat. One girl about 12 years of

age had a broken chilblain, followed by a severe attack of erysipelas of the same leg. Very shortly afterwards a younger child had a mild attack of diphtheria. The only assignable cause was to be found in the bathroom which had a fixed W.C. The waste water from the bath passed directly into the soil pipe. Each time the plug was pulled an unpleasant effluvia was experienced, proceeding from the waste pipe in bath. When this was rectified the sore throats disappeared.

Cold, exposure to bleak winds, dampness of houses, effluvia from manure heaps I consider predisposing causes, although the latter may sometimes be a direct exciting cause. Dr Thresh, in a paper on "London Manure and Diphtheria" instances a case in a child occurring at Burnham-on-Crouch when, after every other imaginable cause had been excluded, the only one left was the fact that the child had been playing with some filthy manure which was unloading from a barge the day before. In the same paper he speaks of the filthy smells arising from such manure and instances several outbreaks which appeared to have no other cause for their production than the

manure. (I mention a case on p. 14 which possibly might have had its origin from the same cause.) It is therefore just possible that this manure contained the bacillus and also that it may have rendered a non-virulent bacillus virulent.

A good deal of experimental work has lately been going on, as to the relation between the pseudo-bacillus and the true bacillus which, when completed may throw a good deal of light on the etiology of this disease.

I am inclined to believe that the bacillus is to be found in the soil, and specially in a damp and polluted soil, the upturning of which sets it free. That the partial drying of a previously wet soil is favourable to it, and if present in this wet soil, in an inactive form, this partial drying renders it active, and that possibly the same circumstances may convert the non-virulent into the virulent bacillus.

The inhalation of bad smells from sewers, drains, dungheaps, etc. may have the effect of converting, under special conditions, the bacillus, morphologically the same, present in the throat,

into the true diphtheria bacillus, as well as producing such conditions of the throat and general system as would be favourable for its growth and the production of diphtheria.

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Dissemination. Flügge and others point out that the degree of desiccation necessary to float the bacilli would be fatal to them. This is borne out in practice. Direct contact with the patient's breath is necessary, and not only that, there must be some direct medium by which they are conveyed, such as saliva or membrane. Clothing, utensils, and such-like, used by patients, or which have been in contact with them also can carry the infection in a lesser degree. It is not carried any great distance by air, and hence an early diagnosis of the disease with isolation will prevent often its dissemination. The aggregation of cases increases its virulence. The middle class seem to me more prone to the disease than the poor, or the better class, and I think for this reason: they frequently change their residence, and prefer new houses, sometimes moving into them before they are thoroughly dry. The houses are often "jerry-built", with bad sanitary arrangements. I have over and over again traced first cases to these new houses.

As to dissemination directly by animals, I have no experience, and the difference of opinion on

this point at the present time is very great. Suffice it to say a patient of mine is a bird-fancier, and his pigeons often have "roup", when there is no diphtheria in the neighbourhood, and I have at times examined the exudation myself, and have had it examined by an expert, but have never found the bacillus of diphtheria. I have not had an opportunity of examining other animals supposed to be suffering from anything approaching the disease.

Milk has long been given credit for spreading this disease, and the evidence in favour of it is too strong to doubt it. An epidemic occurred in a district where I was acting as locum tenens. The fact of it suddenly springing up, and confining itself to some families in which two or three were attacked at the same time, pointed to milk infection.

After prolonged enquiry it was found that the milkman who had supplied the families first attacked, and whose milk had been stopped, had clandestinely removed one of his children who was ill with what proved to be diphtheria, just before the epidemic broke out.

Epidemics are related in which no cause could

be found for the infection of the milk, except the cow itself. This may be the case; still a human source of infection can in most milk-epidemics be traced. If cows are liable to be attacked with diphtheria, it appears to me that milk-epidemics would be more common, and when an ordinary epidemic occurred, there would be the greater danger of a milk epidemic ensuing, on account of the cows being subjected to the same influences as human beings.

The attendance of a number of children at school has been suggested at various times as a potent means of dissemination, and many theories are put forward, such as recrudescence, progressive development of virulence, or infectiousness, and such-like. I have not the slightest doubt that schools often are the centre for spreading the disease, but are no more the originators than the ordinary over-crowded house.

Just as the aggregation of cases in a sick room will increase the virulence, so in the same way aggregation at school may convert less virulent into virulent, or it may be non-virulent into virulent, in the latter case if the proper factors are

present.

That schools are the means of disseminating it, the following case, which occurred in my own practice, will serve to illustrate. I was asked to see a labourer living at a farm about a mile and a half from the Board Schools in Manor Park, Essex, who had been ill for two or three days with sore-throat. He had been employed for some time in taking produce to market in London. On the homeward journey the carts were loaded up with manure wherever he could procure it, for farm purposes. I found him suffering from diphtheria well advanced in the first week. His daughter of 10 years was attending the Board Schools, and on my return journey I called and ordered her home. On enquiry, I found that two of her most intimate companions at school, and who resided quite in another direction, were absent from school that day. Subsequently I learned that these two children, members of different families, were laid up with diphtheria. Neither the labourer's child or any of his family developed the disease. Every precaution was taken to prevent it spreading. He was removed to Hospital at once,



disinfection carried out, and the family closely watched. No other cases of diphtheria appeared in the neighbourhood, and the only cause of the two cases, in my mind, was contact with this labourer's child at school.

This case I think also serves to illustrate how infection may be carried by an individual who is not suffering from the disease.

Symptoms.

(1) Invasion. In the majority of cases there is an absence of the usual prodromata which characterise some of the other infectious diseases, and when they do occur, I am inclined to think they are dependent upon some other cause than the diphtheritic virus, such as, concomitant tonsillitis, follicular tonsillitis, or scarlet fever. The patient does not complain of the violent headache, sickness, pains in back and limbs, shivering, heat, etc., nor has he the flushed or excited appearance common in these diseases. But rather of general weakness, lassitude, with pains confined to the region of the throat and neck. His ailment, as he describes it himself, is pain and swelling behind the angles of his jaws, and down his neck, with a feeling of great prostration, complete indisposition, no desire for food, but still no repugnance for it, no hyper-pyrexia, no thirst, nothing characteristic about the tongue.

In other cases the prodromata are more severe, resembling those of the diseases named; but these are the exception, unless in the malignant form.

(2) Throat. On examining the throat in the very earliest stage before the formation of membrane, it will be noticed red and swollen. In some part or parts there is increased redness, the mucous membrane appearing drawn tightly over the part, giving it a glazed appearance. Secretion from these parts is less, and if secretion be present it appears quickly to be absorbed. The area of these different patches of increased redness is defined, and shows up in contrast to the general redness. There is a feeling of dryness in the throat and difficulty of swallowing, without any actual sense of pain. This appearance is soon followed by the patches described before assuming a pale colour, and on close examination this paleness is found to be due to a whitish exudation on the surface which can be easily brushed off. Gradually this exudation becomes thicker and more difficult to remove, darker in colour, and implicates the mucous membrane, which is torn off on removing it, revealing a bleeding surface with sharply defined angry-looking edges. During several epidemics which occurred in my practice, I made a routine practice, and always do so

now, of examining the throats of all persons in the house, isolating those whose throats presented this early appearance with the result in many cases of finding that this precaution was not in vain.

(3) Temperature. In an uncomplicated case the temperature ranges from 100° F. to 103° F., rarely exceeding this, often never more than 100° F. In other acute diseases affecting the throat, a high temperature is almost invariably present; in diphtheria it is the exception.

(4) Pulse. It is important that a correct record of the pulse should be kept day by day, no matter how benign the case appears, as its variations may be the only indication of subsequent serious complications. This has been forcibly impressed upon me by actual experience, in cases where, after apparent convalescence, and where my interference with full liberty had been over-ruled, the issue was fatal. The frequency of the pulse is not what would be expected from the temperature. With a low temperature there may be an exceedingly quick pulse or <sup>a slow pulse with a high temperature.</sup> ~~the reverse~~. Should the latter be present with persistent irregularity at an early stage, it

points to a severe attack of the disease. Generally it is quick, easily compressible, or soft with a want of impulse, and often, early in the disease, irregular. This irregularity may be in the strength of the pulse, the length of the interval between each beat, or the frequency. It is not persistent, and may be absent for days. As the disease advances these features become more marked and may continue far into convalescence, when all other symptoms have gone. In the majority of cases, after the acute symptoms have passed off, which generally occurs in about a week, the pulse slows down, and may become perfectly regular, but oftener with occasional irregularity. Any abnormal quickness now, with persistent irregularity, is an indication of heart trouble.

(5) Heart Sounds. From the onset of the disease the heart sounds are weak and the action more frequent, but this varies greatly according to the severity of the attack. Dr Sidney Phillips has studied the circulation in a great number of cases, and shortly after his article appeared in the British Medical Journal in 1890, I had an oppor-

tunity, during an epidemic, to make myself acquainted with some of the phenomena which he pointed out. The patient, when first seen, very often has marked languor, the apex beat imperceptible to sight or touch, and only to be localised by auscultation. At the same time the first sound is short and weak, as heard over the apex, and the second sound seems accentuated. For some time I considered the latter was actual accentuation, but this I found was due to the weakness of the first sound, making the second appear as if it were accentuated. A systolic murmur sometimes can be heard over this part during the first week increasing in intensity, and is a cause of anxiety, even although other symptoms seem favourable. This generally clears up permanently during the second week, but may return any time during convalescence, or may appear for the first time in convalescence. The presence of this murmur early is important for prognosis, and also in the convalescent stage when the heart is troublesome. In the majority of my cases which suddenly terminated fatally this murmur was present. Over the base of the heart the first sound is sometimes inaudible

and may be occupied wholly by the murmur referred to. The second sound is not altered in character, but it appears as if delayed. The heart sounds are quicker, the first sound is short, and there is a distinct pause between first and second sounds, due to the short and weak first sound. The pause between the second and first sound seems shortened but this I think is due to the same cause.

Associated with this condition, irregularity is generally to be noticed, which may either be due to delay of the <sup>second</sup> ~~first~~ sound, or great diminution in <sup>of the first</sup> ~~the~~ force, <sup>frequently</sup> followed in either case by increased frequency and impulse, which causes the patient great alarm, and this, with the other conditions of the heart (fatty degeneration and nerve degeneration) and those already mentioned, is <sup>sometimes an</sup> ~~an~~ immediate cause of the sudden death in the convalescent state.

In the early stage of the disease irregularity is frequently present but not persistent, and indicates of itself very little in giving a prognosis.

(6) Albuminuria. There seems to be a general idea with some practitioners that albumen should be present in all cases of diphtheria, and I have heard men with large experience in private practice state that a case was not diphtheria because there was no albumen in the urine, giving follicular tonsillitis the credit for the symptoms. A greater fallacy cannot exist. When I had first to deal with this disease I was greatly discouraged in not finding albumen in all my cases. The generality of patients a practitioner like myself has to deal with are young, the nursing devolves mostly upon an over-worked mother, the difficulty under these circumstances of obtaining a reliable sample of urine daily must be evident. It is more generally found in the early stages of the disease and may disappear for days to again appear. Sometimes it is present at one part of the day and absent at another. Its continual persistence is to be considered unfavourable, but when it is absent now and again its appearance is not to be feared. I have rarely found it associated with the general weakness which follows the disease, its appearance after apparent



convalescence with irregularity of the heart's action is to be looked upon with a certain amount of anxiety. On the whole, the presence of albumen in the urine is a concomitant rather than a sequel of the disease. Its absence is by no means an indication of a mild attack or a favourable termination.

(7) Paralysis. Some have spoken of paralysis coming on early in the disease. The only indication of nerve lesion which I have noticed early is loss of knee jerk, and its recognition is often useful in obscure cases of sore throat. This may often be detected in the first week, and leads one to anticipate paralysis at a later stage; and it is well to warn friends that such may arise. Dr Sidney Martin, in Professor Crookshank's book on Bacteriology, gives some interesting experiments on the different effects of the ferment and the albumoses in producing paralysis in animals. These are interesting when compared with what takes place in the human subject when paralysis sets in. The first indication of this sequel generally is the peculiar nasal tone the voice acquires, soon followed by difficulty of swallowing, paralysis of some of the ocular

muscles, more commonly the ciliary muscle, producing loss of accommodation, which in young children is often of great assistance in the early diagnosis of the paralysis. Paralysis of the lower limbs generally follows, and is often preceded by prickling sensations, and occasionally shooting and darting pains in the legs. The paralysis most to be feared is that affecting the respiratory muscles, and when this is accompanied with a weak irregular heart the danger of a sudden and fatal termination is very great. I attended a young lady suffering from a slight attack of diphtheria, followed by paralysis of the soft palate and lower limbs. From the onset of the disease her heart was irregular and weak, with distinct systolic murmur. When quite convalescent from the acute disease, but still confined to bed owing to the irregularity of the heart, she suddenly developed great difficulty of breathing one day which soon passed off. Recurrent attacks of this took place, although the heart's action appeared to increase in strength. One night she was allowed to dress her own hair in bed, and had hardly begun the operation when she fell back dead. I pre-

sume in this case death was due to cardiac failure, combined with paralysis of the muscles of respiration which was indicated by the recurrent attacks of difficulty of breathing. I invariably take the precaution of warning my patients, no matter how slight the paralysis appears, or where there is persistent irregularity of the heart, of the great danger they are in.

Treatment. During the last two years there has been quite a revolution in the treatment employed.

Local Applications. At one time it was universally held that local applications to the patches were of primary importance. Nowadays those best qualified to judge assert that, although of importance, they are not so necessary, and some go so far as to dispense with them altogether. No one unless those who have actually used an application themselves can conceive the physical energy required, the worry, anxiety, and danger to those applying the remedy, combined with the excitement, alarm, and strugglings of the little sufferer, followed often by prostration. For these reasons I have over and over again debated with myself the advisability of continuing this treatment, and some years ago I came to the conclusion, with all the difficulties, dangers, etc. attendant thereto, local applications should be persisted in. Since the introduction of anti-toxin, I have modified my opinion, but my experience of this remedy being confined to so few cases in comparison to those treated without anti-

toxin, will not warrant me in neglecting these applications, no matter how favourable the case appears. The patches of membrane are the seat of the manufacture of the poison, by using applications with the object of destroying these, we attack the very source of the poison, and to neglect this, no matter what the ultimate effect of other remedies may be, is a serious matter. The membrane is broken up and becomes much softer and less tenacious in a saturated solution of bicarbonate of soda. This I satisfied myself of in a case where, death seeming imminent from obstruction in the larynx, I induced vomiting by pushing a bent throat-brush into the larynx, when a complete cast was expelled, which I subsequently treated in the soda solution, and found it rapidly became soft and pulpy. In this case I continued to apply the solution of soda, following up each application with a paint consisting of Tinct. Ferri Perchlor., Ac. Carbolis,  $\bar{c}$  Glycerine. I afterwards adopted this form of treatment with a varying amount of success, which was most marked in those cases where the treatment was able to be thoroughly carried out. Some have said the membrane

will dissolve<sup>w</sup> this solution outside the body, but question its efficacy if applied to the membrane in situ. My experience is that it softens the membrane which is more easily detached, and it also allows the more potent application to sink in. In using the solution of soda, a soft, large, straight camel's hair brush is best, some of the undissolved salt should be taken up as well, and the patches well daubed two or three times. If there is membrane in the nose, the solution should be injected up the nostrils. Having done this, in about five minutes apply freely the perchloride of iron, etc., paint. Repeat this every three hours, so long as any membrane can be seen. Since the germicidal effects of the various preparations of mercury have been so generally acknowledged, in place of the iron paint I have employed a paint of the biniodide of mercury in glycerine<sup>Water</sup>, 1 in 2,000. These applications should be used by the physician himself on each visit, and the nurse or mother made thoroughly acquainted with the necessity of applying them frequently. The good effects will not be evident unless so done, and when not done thoroughly are better left alone, as they only worry the

patient. In those cases where the throats of those living in infected houses have shown the characteristic patches of redness, without exudation, which I have mentioned before, the free swabbing of the throat frequently with biniodide paint has in some cases prevented the further development of the disease. This was particularly brought to my notice in my practice about eighteen months ago. A boy aged eight years, who attended the Board Schools in this neighbourhood, was taken ill with diphtheria. His three sisters attended a private girls' school. Several children attending the Board Schools were absent from diphtheria, but none from the private girls' school. The eldest of the girls was exceedingly useful to her mother in household matters. On the second day of my attendance, whilst examining the throats of all in the house, those of the three girls looked suspicious, the two younger ones more especially. The eldest girl I was undecided about and would have isolated her had her mother not said how useful she was to her. The younger ones were isolated, and all three were repainted with biniodide and glycerine, iron and mercury being given inter-

nally. On the following day the eldest girl had a distinct patch on one of the tonsils which proved to be diphtheritic. The younger children escaped. It might be argued here that the elder girl, coming frequently into contact with the mother, was more liable to contract the disease than the others, but it must not be forgotten that these younger children were closely attended by the mother, and evidence goes to show that direct infection is the most common way of acquiring the disease. On interrogating the mother, I found that the eldest girl's throat-painting had been neglected on account of the numerous other duties imposed on her. I think there can be no doubt in the case of the two younger children, the prompt measures adopted prevented them from developing the disease, and that the paint was fatal to the bacilli which to all appearance had begun their work.



Internal Remedies. Of all internal remedies Iron holds the first place, and when the patient can take it in the form of the perchloride, I think it is the best. It is often difficult to get young children to take anything in the form of medicine, and in these cases I prescribe the perchloride to be dropped into some sweetened lemon water and given in the form of a drink. By this means I have been able to give large quantities of iron which otherwise I would not have been able to do. In combination with this I have frequently prescribed the perchloride of mercury; formerly I gave this as a routine practice, but latterly I have not done so for several reasons. The perchloride of mercury depresses, and the patient is subjected to enough depression by the absorption of the ferment. And again, I do not know positively that this drug has any effect in neutralising the poison already absorbed into the system, or that ~~how~~ it can prevent the further absorption of the poison, or that it has any effect on the bacilli by internal administration. The bacillus, as far as is known, remains at the seat of the exudation, and if our energies

are directed, by means of topical applications of mercury, to this part, the internal administration seems not to be wanted. However, in robust constitutions with a good action of the heart, or as a preventitive in a suspicious case I generally give it; immediately any heart symptoms appear I cease giving it. Quinine I find useful combined with the iron in those cases where there is extreme languor early in the disease, and it is useful in the convalescent stage. Late in the disease strychnine has a beneficial effect, and more especially where the nervous system is affected. For the affection of the heart nothing seems better than this drug, and in impending heart failure, hypodermically injected it is of undoubted benefit. Digitalis in my hands has not proved beneficial.

Where the bowels are constipated, I prefer a glycerine enema to purgatives; it is less distressing to the patient, and is especially to be preferred in those cases where absolute rest in bed is necessary. Many other remedies are useful according to the symptoms, but those mentioned I consider the best weapons with which to fight this disease.

Stimulants and Food. In treating of these, I cannot disassociate them. By stimulants I mean brandy, whiskey, port wine, champagne, and such-like. Many practitioners advocate one of these from the very beginning, pinning their faith to the stimulation of the patient, with the effect in many cases of completely narcotising him or her, and so hopelessly altering the symptoms. If the patient requires stimulants at the beginning of the disease, he doubly needs them in the second week. They mask and alter symptoms, so that<sup>a</sup> definite opinion as to the actual progress of the disease cannot be arrived at. The heart and pulse are altered, the tongue becomes dry and hard, the temperature varies according to the amount of stimulation or depression caused by them. Children get on much better without them, and I cannot recollect any appreciable benefit in those cases where they have been given at the commencement of the disease. They are only indicated where there is reason to fear collapse from heart failure. In the malignant form of the disease, with extensive formation of membrane, it is well to stimulate the patient early, as the

large quantity of poison rapidly absorbed into the system soon causes such an amount of depression that no quantity of stimulants can overcome. Champagne is to be preferred to any other, and this given to children in dessert-spoonful doses every hour has a salutary effect. In the case of adults, I am always guided by the previous habits of the patient. In all cases where stimulants have been habitually taken, they should be given during the whole course of the disease; in other cases they should be avoided unless the symptoms mentioned indicate their need.

Beef tea is the best stimulant, given in the peptonised form, and this is so easily made now by an ordinarily intelligent mother. Carnrick's preparations I have found useful. Milk curdled by means of a few drops of rennet whipped up, and a little salt and pepper added is also grateful to the patient when the beef tea is resented. Gelatine added to the beef tea is often useful, helping to prevent the great waste of tissue which is going on on account of its easy oxidation in the body. Fruit, eggs beaten up, jellies, soups, ice etc., when they can be taken are all appreciated by the patient.

When food is refused by the mouth it must be given by the rectum. These enemata should not be entrusted to ignorant hands, but should be done by a person properly instructed in their administration. Nourishment is one of our sheet anchors, and a great deal of success in the treatment of this disease is in seeing that it is properly given. I cannot mention this too strongly, as I have attributed many of my recoveries to this one thing. Nutrient suppositories I have no faith in; as a general rule the major portion of them is voided with the next action of the bowels.

The diet should be fluid until all acute symptoms have passed off, and where the heart is affected and the patient compelled to remain in bed, care must be taken that the food given is easily digested. Indigestion, accompanied by flatulence, is a cause of great discomfort to the patient.

Antitoxin. My experience of this remedy is limited to eleven cases, all of which recovered. In each of these cases I found Klebs-Löffler bacillus and had my observations confirmed by the Clinical Research Association.

Having read that no harm was to be anticipated from its use hypodermically when used with strict antiseptic precautions, I gave these injections in the following way, using the antitoxin prepared by the British Institute of Preventitive Medicine, 20 c.c. on the first appearance of the membrane or on first seeing the patient; 20 c.c. in 12 hours time; then 10 c.c. every twenty-four hours until the complete disappearance of the membrane, and in no case were there any bad effects. In young children I began with 20 c.c., but instead of repeating this amount in 12 hours I only gave 10 c.c., repeating this as in adults every 24 hours. The only noticeable improvement after 12 hours was that perhaps the membrane seemed softer. After 24 hours in three of the cases I was much struck with the improvement. The membrane seemed to peel off quite easily, leaving a clean, red, raw surface to which

the biniodide paint could be thoroughly applied with the effect of in another 24 or 36 hours the complete disappearance of the membrane. In the other cases the effects were more protracted, nevertheless I was impressed with the fact that they did not get worse after the first injection, as there was no material increase of the membrane such as I should have expected from the appearance of some of the patches. In the children there was an absence of the great languor and prostration so generally found. Twenty-four hours after the first injection some of them were sitting up in bed playing with their toys, although membrane was still to be seen. After about two days (when the membrane, which was much thinner, was removed) the ulcerations looked healthier and had lost the swollen, angry, punched out appearance, and the membrane did not cling, as it usually does, to the surface, but could be removed intact. In one of the cases, I was called in so late that I had to perform tracheotomy to save the child's life; notwithstanding, the effects of the antitoxin were very encouraging. The mucus from the tube soon became glairy, less viscid, and easily removed.

Local applications had to be stopped, but under the continued injection of the antitoxin the child made a good recovery. This case commended to me the value of antitoxin.

As far as I can judge from my very limited experience, antitoxin injections should be given early in the disease, and continued regularly until all membrane has disappeared, and not discarded (unless there are direct indications to do so) in twenty-four hours because the promised good effects are not apparent. The object in these injections is to anticipate what takes place in the natural course of the disease, viz: the production of a substance which has the effect of either directly antagonising the diphtheria toxin, thus preventing the paralysing of the phagocytes; or stimulating the cells of the blood, lymph, and tissues to resist this paralysing effect. As long as the patches are present so long are the bacilli manufacturing the ferment, and so long is it necessary to introduce the antitoxin. Hence it should not be left as a last resource, nor should it be given in a single dose. It should be given early in the disease with



a distinct object in view, and continued, not forgetting that the manufactory must be destroyed by local applications and the strength maintained by nourishments.

If antitoxin merits all its supporters assert, it will be of inestimable value as a preventitive in epidemics.

In conclusion, I reiterate what I have said at the beginning of this paper, that I do not present this as a treatise on the subject of diphtheria. I cannot lay before you a paper full of laboratory experiments or hospital experience, but simply personal observations on a disease which has occurred frequently in my practice as a general practitioner. Many important points I have omitted, but I have endeavoured to lay before you these points which have helped me in my diagnosis and prognosis, and the means I have adopted in battling with this formidable disease.

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