ON PARALYSIS FOLLOWING DIPHTHERIA IN CHILDREN

by

JOHN McGREGOR, M.B., C.M.

ON PARALYSIS FOLLOWING DIPHTHERIA IN CHILDREN.

INTRODUCTION.

During the terms of my House-physicianship at two of the London Children's Hospitals, it was my good fortune to see a considerable number of cases of paralysis following diphtheria, both in the out-patient departments and in the Wards; and I was early struck with the difficulty experienced in many instances of making a correct diagnosis, and, from the subsequent progress of the cases, of the great importance of so doing. The diversity of reasons for bringing the patients to the Hospital, the want often of a definite history, frequently none obtainable, and the numerous cases presenting apparently but little departure from the normal, all helped to make the subject as interesting as it was often perplexing.

In this paper I have entered into the subject almost entirely from a clinical point of view, and have tried to show the great necessity there is of regarding all cases of diphtheritic paralysis in children as serious, and of always pursuing a definite line of treatment. The fatal cases which I have seen have fortunately been few in number; and so far as our present knowledge goes, the result could not be avoided.

It has been stated that grave complications are more likely to occur where the paralytic symptoms are multiple. This is undeniably true; but the frequency of these multiple cases seems to have been quite overlooked by the majority of writers on the subject.

In another part of this paper I have gone into this question in detail, and would here only draw attention to the cases in Appendix II which have been tabulated mainly to bring out this point. I have put in parallel columns the four most common symptoms, and a glance over these will show how often they are all present and how seldom only one or two. This being so, these cases are thus multiple, grave symptoms may arise at any time, and death ensue. The prognosis then in any given case must necessarily be very guarded and treatment rigorously carried out.

I am much indebted to the various physicians of both Hospitals for permission to incorporate in this thesis the cases which were under their care.

Whilst relying mainly on my own experience for the description of the various symptoms, I have found it necessary to include several cases which I did not see personally, for the reason that they bring out some points which my own do not, as for example. recovery after severe cardio-pulmonary symptoms. Similarly I have translated several cases from the works of the leading French writers on the subject.

In conclusion I may state, that the main points which I wish to make prominent are:- that paralysis following diphtheria in children is much more frequent than is commonly supposed; that in the majority of instances it is of a multiple nature; and

finally, that under appropriate treatment, as that described in the text, grave symptoms will often be avoided and a happy result obtained.

SITE OF THE MEMBRANE IN RELATIONSHIP TO THE PARALYSIS.

Formerly it was held that paralysis only followed on faucial diphtheria and was due to direct action on the muscles and nerves of the part, causing locally paralysis of the palate and spreading by the sympathetic to the cervical ganglia (whence eye troubles) by the palatine nerves to Meckel's ganglion etc. The weakness of the legs and other distant symptoms were attributed to the asthenia ordinarily following acute maladies. This was soon abandoned; it failed to adequately explain anything like the whole of the symptoms, and cases of paralysis following the presence of membrane elsewhere, on the fingers, genitals etc. and not on the throat upset it completely. Trousseau, Gueneau de Mussy, Sanné, Paterson, Guthrie and others have published cases where the membrane was limited to other parts, on wounds, on the fingers, genitals, ears, umbilicus (in children) etc. with well marked paralysis afterwards. The actual site of

the membrane is therefore of little importance; paralytic symptoms may follow its appearance anywhere with characters and course exactly the same. Cadet de Gassicourt+ states that in cutaneous diphtheria the paralysis not infrequently begins in the legs. His statistics show a considerably higher per centage (of paralysis) in laryngeal cases.

Diphtheria	Cases	Paralysis	Percentage of Paralysis.
Mild	155	28	18%
Grave	135	30	22%
Laryngeal	135	43	31%

This may be explained by the fact that laryngeal cases are almost always severe.

CHARACTER OF THE PRECEDING DIPHTHERIA.

From the mildness or the severity of the attack of Diphtheria it is quite impossible to foretell whether paralysis will follow or not, or of what character it will be. A severe attack may have no such sequela and a mild one prove fatal from it. Neither can we tell what kind of paralysis to expect; a slight case may be followed by severe or fatal paralysis or vice versa.

+ Maladies des Enfants, Paris. 1889

As a general rule paralysis is more likely to follow a grave case of diphtheria and to be of a more severe type. According to the table just quoted from C. de Gassicourt this is so, but he is careful to add that exceptions are not at all uncommon. Other observers have stated much the same opinion.

Henoch[+] and some others have said that paralysis is more common after mild cases. This however is not so, the real reason being that the majority of severe diphtherias prove fatal and consequently paralytic symptoms have no time to develop.

The same reasoning applies to the statement that it is more common in adults. Diphtheria is a far more fatal disease in young children than in adults.

FREQUENCY.

In estimating the frequency of paralysis following Diphtheria there are several sources of error which must be carefully guarded against.

The principal are:-

(1) The heavy mortality in Diphtheria.

(2) The want of extended observation.

(3) The slight nature of many of the cases.

+ Lectures on children's diseases. New. Syden. Soc. Tran. 1889.

As regards the first cause we must remember that death occurs in many cases before any paralysis has time to develop, and that in consequence the average, if these fatal cases be included in the statistics, will be greatly lowered.

Take for instance Cadet de Gassicourt's figures. Out of 937 cases of diphtheria there were 128 with paralysis or 13·6% - a very moderate proportion; but if we go into the figures carefully we find how utterly erroneous this is. Of the 937 there were 497 which proved directly fatal from the diphtheria most of which required tracheotomy. Amongst these there were only 27 cases of paralysis or 5·4% whilst out of the remainder there were 101 cases, giving a percentage of 23·7. This at once shows how inferior the first statement was to the truth. And when we remember, further, that paralysis is more likely to follow grave cases of diphtheria we may safely say that had they not proved fatal the proportion of paralysis amongst them would have been even higher than amongst the milder ones. C. de Gassicourt evidently considers this very probable for he says "Je suis convaincu que tous ces chiffres sont inférieurs à la vérité".

The second great source of error is the want of sufficiently extended observation. In fever Hospitals for example children are sent home as soon as the actual diphtheria is better and all risk of infection practically removed (provided no complication has arisen); and paralysis coming on a few weeks later is, of course, unrecorded in their books. Their figures must in consequence be considerably under the truth.

These are the cases which are so generally seen at Children's Hospitals but it is obvious that they are quite useless for statistical purposes.

Looking over all the cases which I have collected and which are appended to this paper it will be seen that in the majority of them the paralytic symptoms did not appear till the fourth or fifth week after the diphtheria. Now had these been followed up by the original investigators I have no doubt that their figures would have been considerably modified and their percentages much higher. McKenzie+ out of 955 cases of Diphtheria of which 452 were fatal had 20% of paralysis. This he evidently feels is an underestimate for he adds: "I feel sure that had I been able to follow up all the cases there would have been comparatively few in which some symptoms of affection of the nervous system were not found".

The third cause of error, the mild nature of many of the cases must be remembered. Many children show such slight symptoms that the parents never detect anything wrong and if they do come under the observation of the Physician it is only for some other affection. This differs from the preceding source of error where the paralysis may have been expected but observation was not continued long enough. Here they are never seen after the primary throat condition is better. The result

---

+ On Diphtheric Paralysis etc. St. Thomas' Hospital Reports 1891.

is the same however; in both cases the physician enters them into his case book as having had no paralysis.

Barthez and Sanné out of 1382 cases of diphtheria had 155 of paralysis or 11%, but no statement is made as to the elimination of fatal cases, and the truth of the statement is thereby called in question. Roger[+] had 16·6%; and Landouzy[++] writing on this subject states that various observers have given widely different percentages from 1·15% to 66%. Taking it as a whole we may confidently put the average at 25% at least, that is one child in every four attacked by diphtheria will suffer from paralysis. The actual proportion will be found to vary with different epidemics, greater after a severe type of the disease, lower after a milder form.

[+] Archiv. Gen. de Med. 1862.

[++] Des paralysies dans les maladies äigues, Paris 1880.

FORMS OF THE PARALYSIS.

Cadet de Gassicourt in his "Maladies des Enfants" divides diphtheritic paralysis as follows:-

1. Limited paralysis.
2. Generalised paralysis.
3. The cardio-pulmonary form.

The vast majority of his cases (103 out of 128) were of the first variety, limited to the palate and pharynx but in exceptional cases to the legs alone, or to the eyes or diaphragm, etc. Of the second form there were 13 cases and of the last 15.

The observations detailed in the accompanying tables show a marked contrast to this; limited paralysis has not a single example, whilst in them all there is ample evidence of the generalised, or as I prefer to call it, the multiple character.

Why this difference should exist I do not pretend to say. It cannot be due to the peculiarities of an epidemic for these cases extend over several years and include many epidemics in various parts of London and of various degrees of severity; nor can it be that cases of the limited form are not seen because of the slight nature of the attack, soon passing off and never causing the children to be brought to hospital, for how then can we explain the fact that he only had 13 multiple cases whilst in London we find them in abundance. Many cases seem at first

sight to be limited to the palate and pharynx but careful and extended observation will generally show the evidence of paralysis elsewhere, loss of the power of accommodation or squint, or absence of knee jerk, etc. This I have had frequent opportunities of seeing in cases brought into the Wards with only the palate apparently affected.

Of course there is a great difference in the use of the word "generalised". He may reserve it for very widespread paralysis as in the examples he gives, but where then does he put such cases as are given in Appendix II. with palate, eyes and legs affected?

They are not limited, neither can they enter the cardio-pulmonary division, and if they are his generalised cases why has he not seen more of them?

The term "multiple" seems to me much better. It has a much wider range of meaning including all cases from those with 2 or 3 muscles affected to those where almost every part is involved.

And in this respect it becomes important to observe the parts most frequently affected. Maingault+ gives his results as follows:-

---

+ Quoted by Landouzy.

Proportion of paralysis in various organs.

Palate	70
Generalised paralysis	64
Blindness	39
Legs	13
Squint	10
Head and Neck	9

etc., etc.

Now these figures are much more in accord with observations in this country than those of C. de Gassicourt. It will be noticed that there are 64 generalised cases; almost equalling the paralyses of the palate in number. Blindness is probably loss of accommodative power and this is very common in his experience and likewise in ours.

Taking it from the point of view of my own experience, and from the observations made in this country, cases of limited paralysis are very rare. With these as the only exceptions we may regard diphtheritic paralysis as always multiple; and that as regards the cardio-pulmonary form it never occurs alone and is but a further evidence of the multiple nature of the cases in which it appears.

S Y M P T O M S.

Onset. Paralytic symptoms may appear during the course of the diphtheria or from this time to several weeks after the membrane has disappeared.

Where the paralysis comes on with the membrane still present or shortly afterwards the case is very liable to end fatally from cardiac paralysis. Generally the palate becomes affected, then in a day or two the heart as well, and death ensues more or less suddenly. In some instances heart failure comes on without any preceding palate signs.

In a large proportion of the cases the 3rd. to the 5th week after the diphtheria is the time when paralysis first appears. Cases have been reported at the 7th or 8th weeks or even later. The 6th week is not uncommon. Usually there is nothing to indicate its approach. Some observers have noticed a slight rise or irregularity of the temperature but this is by no means constant. What is more important is the general health of the child. It does not seem "to get over" the diphtheria properly; it is anaemic and listless and may be brought up to the Hospital for "strengthening medicine". On the other hand many children seem in perfect health and are attending school regularly.

In the matter of symptoms it is not generally the first to appear which leads the parents to seek advice. And herein lies a difficulty in deciding what symptom appears earliest. The

truth of the matter is that nothing can be taken as reliable unless observed by a medical man. Take for instance some of the symptoms. Thickness of speech and nasal voice are often unnoticed by the parents. Even fairly well marked squint may pass unseen. On the other hand involvement of the legs attracts their attention at once and they not only consider it the beginning of the child's illness but may attribute the palate condition to falls sustained thereby.

In the case of a little girl brought up to Paddington Green Children's Hospital the complaint was that she had fallen and bruised her nose a few days previously and since then her speech had been thick and she had had regurgitation through the nose when she drank. On examination however, I found, besides a large bruise over the bridge of the nose, well marked palate and ocular palsy, absent knee jerks and paresis of the legs, and questioning elicited a distinct history of recent diphtheria. We further learnt that regurgitation had occurred on more than one occasion a day or two before the fall, but until attention was directed to the part nothing wrong was noticed. Such a state of affairs at once shows how unreliable parents' statements in general are, and how difficult it is to obtain accurate statistics outside of an Hospital.

In the vast majority of cases the palate is the first part to suffer. This is particularly true of children. In adults the legs may be attacked first, and after cutaneous diphtheria

we may see the same thing. The next part may be either the eyes or the legs. In the eyes loss of the power of accommodation comes on early generally before strabismus or other paralysis. After these the symptoms appear in no definite order.

Landouzy's order was palate, eyes and legs; and Henoch similarly, palate and then loss of accommodation.

As a general rule it may be stated that the parts most frequently attacked are also the parts earliest attacked and the order of frequency and the order of appearance are the same.

Though generally spoken of as paralysis the condition is more one of paresis of the affected muscles. Complete loss of power is uncommon. Sometimes the affection is so slight that careful examination has to be made. This point however will be gone into when the various symptoms are treated of in detail.

Before doing so it might be better briefly to refer to the mutability of the paralysis. It was Trousseau who first pointed out this curious fact. He observed that while on one visit the patient lay absolutely helpless, unable to swallow without regurgitation or choking, or raise his head from the pillow, yet on the next he could manage these fairly well. This improvement lasted but a short time and he relapsed into his former condition of weakness. The palate shows this more frequently than any other part possibly because it is more constantly affected. We find the nasal tone of the voice varying considerably from time

to time in some cases and this quite apart from any effort on the part of the patient. Squint may present this also and indeed paralysis of any of the voluntary muscles.

In many cases we find the child looks very well. In others there is anaemia. In the majority we will find a marked disinclination for exertion; he wishes to lie in bed and is often very irritable if aroused to do anything. This lassitude is often a cause why children are brought up to Hospital.

In the bulk of my cases the paralysis has been decidedly multiple, the palate, the eyes and the legs generally all suffering. A simple limited case I have not seen, but on the continent they seem common.

We will now take up the various parts of the body affected and the symptoms generally met with in detail.

Palate. The first symptom which generally appears is an alteration in the character of the voice. It seems thick and has a distinctly nasal tone which soon becomes more decided till it is patent to every one that the child "talks through his nose". Then follows difficulty in swallowing due to implication of the pharynx and upper part of the œsophagus, so that the patient complains of the food sticking in his throat. Often it is difficult to say which comes on first, the thickness of speech or the difficulty in deglutition, and in the case of children mothers generally state that they were noticed about the same time.

Where the paralysis increases the difficulty in swallowing is soon accompanied by two other symptoms, regurgitation of food through the anterior nares, at first only occasionally and of fluids merely, but later on in some cases of both solids and liquids and that constantly; and choking and coughing on attempting to drink. These last are due to anaesthesia of the epiglottis and upper part of the larynx and probably to paralysis of the depressors of the epiglottis as well.

On examining the throat we at once notice the condition of the soft palate. It hangs down motionless and relaxed, the uvula trailing on the tongue, and it does not rise when the patient phonates or takes a deep breath. If we touch it with a feather or a pen we often find that it is anaesthetic. The posterior wall of the pharynx is in many cases likewise insensitive.

In the majority of instances the paralysis is symmetrical and in consequence the uvula hangs straight down in the middle line; but we may find it affected unilaterally, the uvula being then drawn to the healthy side. Henoch says that this is not infrequent. In one case seen lately there was complete unilateral paralysis of soft palate and pharynx.

Very often the paralysis and anaesthesia are by no means complete; indeed, we find all degrees from a mere limitation in movement without any anaesthesia, to total abolition of both sensation and movement. One striking fact is that it requires

but a slight involvement to produce a markedly nasal tone of voice.

From the implication of the larynx, and the consequent risk of fluids entering, we have children drinking in a slow, hesitating and frightened manner, taking very small sips and retaining them for some time in the mouth before swallowing. They manage best when the head is held well back or when they are lying down allowing the fluids to trickle along the posterior wall of the pharynx.

Paralysis of the palate is by far the most constant symptom. Cases where it is absent are very rare but some have been recorded, mainly in adults.

Personally I have never seen a case where it alone was affected nor one where it was not.

As regards frequency M^{c}Kenzie[+], out of 955 cases of Diphtheria had 178 with the mechanism of deglutition affected. Of these 77 were fatal.

C. de Gassicourt[++] out of 128 cases of paralysis had the palate involved alone in 103. This is very unusual judging from the cases reported in this country.

Maingault[=] gives the proportion at 70. (see his table further back).

+ St. Thomas's Hospital Reports 1891.

++ loc cit.

= loc. cit. noted by Landouzy.

The time at which this paralysis comes on varies very much, probably in direct proportion to the severity of the preceding diphtheria, severity judged not from the amount of membrane present but from the effect on the patient. In severe cases it appears early in the first or second week, and in milder ones in the third, fourth, or even later.

The following table from McKenzie's paper showing when the palate was affected in his cases is very interesting.

Period of Disease.

	Days 1-7	Days 8-11	Days 12-14	3rd week	later	
Fatal	33	19	10	11	4	latest date
Non fatal	7	15	15	30	34	was 10th week.
Totals	40	34	25	41	38	

These figures bear out the statements just made.

The high percentage of deaths in the first column particularly, but also in the second and third, is very striking.

As regards the duration of the paralysis we have a considerable variation from a week or ten days to four or five weeks or even longer.

The recovery is generally gradual, regurgitation and choking ceasing - sensation returning and finally movement. Complete restoration of function is the rule.

Eyes. In at least half of the cases we find that the pupils are more or less dilated though they react perfectly to light and accommodation. Early observers commented on this and most recent writers confirm it.

The earliest and most frequent paralytic symptom is loss of the power of accommodation. This is probably what the older writers referred to when they spoke of blindness occurring often after diphtheria. Usually it is symmetrical but may be unilateral. It is very often incomplete but there are all degrees found from mere sluggishness to paralysis. In older children the attempt to thread a needle brings out the condition very well. The attempt is generally futile, and if we try them from day to day we find that they gradually improve. This experiment may serve to discover mutability, success being attained one day after days of failure only to be followed by another period of inability.

The next most common symptom is strabismus. There is rarely complete paralysis of any of the muscles of movement, paresis being the rule. Any of the extrinsic muscles may suffer, or two or more in the same eye. The external Recti are affected much more frequently than any of the others and one eye more often than both.+ Where we have squinting we have of course the formation of a double image. This is very difficult to make out in young children where only one eye is affected. On holding a

---

+ Gowers states that the internal recti suffer most frequently.

small toy in front of the child he seizes it readily and accurately: but on putting it to one side so as to bring the paralysed muscle into action, thus to the right side with the right external rectus affected, we find that he extends his hand to one side being probably confused between the two images. Children who can read sometimes discover this for themselves, and will close one eye thereby getting only one image and being enabled to see the letters clearly. The over-action of the healthy internal rectus on the opposite side is often very marked.

Of other paralysis ptosis may be seen occasionally both uni and bilateral. It generally accompanies paresis of the facial muscles. Guthrie has noted horizontal nystagmus in one case.

As regards frequency McKenzie had the external recti affected in 15, accommodation in 36, out of his 178 cases; Remak, external rectus in 10 cases out of 100. The time of appearance is generally the fourth to the sixth week and the duration one to four weeks.

Legs. Weakness of the legs is very often the cause for which children are brought up to the Hospital. The complaint is that they have "gone off their legs", or are unsteady on their feet, or fall about generally and are easily knocked over. Examination shows a certain loss of muscular power but frequently not enough to account for the marked instability.

The gait is staggering with feet moderately widely apart, in fact somewhat ataxic. Sometimes it resembles the march in cerebellar disease. In ordinary cases where the paralysis is slight, and this is the rule, we fail to find anything wrong when the child is lying down. It performs all the movements easily and accurately though often in a more feeble manner than naturally. In severe cases there is considerable loss of power and there may be some wasting of the muscles. Complete paralysis is very rare.

Loss of power of flexion of the foot causing foot drop sometimes occurs in both children and adults but only where the paralysis is very wide-spread.

Alterations in sensibility are sometimes noticed, more or less complete anaesthesia of feet, particularly of the soles occurs not infrequently, but in young children it is very difficult to be accurate. Older patients sometimes complain of numbness or tingling ("pins and needles") in the feet and legs preceding the actual loss of power by a few hours or even a day or two.

The lower extremities are generally affected after the palate but it is very difficult to be sure on this point unless we have the child under observation; for a slight palate paresis may escape the mother's notice whilst the unsteadiness and falling about attract her attention very readily.

In adults, however, they may be implicated first, as in the case of a nurse who had some weeks previously suffered from a sore throat which was thought to be an ordinary Hospital throat. A month later when in the garden she fell twice, on succeeding days, and examination showed loss of the knee-jerk.

Cadet de Gassicourt states that in cutaneous diphtheria the legs are frequently paralysed first.

The occurrence of this paralysis is rather late, the fourth to the sixth week. It is always accompanied and generally preceded by loss of the knee-jerk.

As regards its frequency Magne+ found it in 50 cases out of 100; McKenzie 35 out of 178. In Maingault's table it was present only 13 times to the 70 times of the palate.

Recovery takes rather longer than in the palate or eyes but is usually complete.

Diaphragm. Paralysis of this muscle is not infrequently met with in a slight degree. In severe cases it may be completely involved and is then a symptom of the greatest gravity.

If we examine a child where it is only moderately affected we notice at once the impairment or absence of abdominal movement and the increased costal respiration. This is such a striking departure from the normal state in childhood, where diaphragmatic action is well marked, that the condition can

---

+ Quoted by Landouzy loc. cit.

hardly be overlooked. Over-action of the thorax is thus necessarily produced; the alae nasi begin to act, and many extraordinary muscles of respiration are called into play.

In rapidly progressing cases, with the increasing paralysis we find that the abdomen no longer remains at rest but begins to show signs of thoracic suction, producing a retraction with each inspiration instead of the usual protrusion.

This is best shewn where the abdominal muscles are also involved. If the diaphragm be markedly affected we have at each inspiration a sulcus of greater or less depth produced in the epigastric and hypochrondiac regions.

Dyspnoea may or may not be a marked feature at first but cyanosis is soon produced, the bases of the lungs become engorged, râles are heard all over the chest and the child dies asphyxiated. It is very difficult to say when this complication will likely ensue. Over action of the lower ribs has been said to herald its approach. It may come on in any case but generally where there is widespread paralysis.

When marked it generally proves fatal. This result is the more likely when there is implication of the abdominal and intercostal muscles as well. In such cases death is not long delayed.

Complete paralysis may come on gradually or more or less rapidly. With this latter course there is often cardio- pulmonary involvement as well.

As regards duration there is little to be said. If it be slight it soon passes off, generally in a day or two, whilst if severe it soon causes death.

Arms, Neck and Trunk. The arms generally escape altogether or are but slightly affected. Occasionally in severe cases they may be so paralysed that they lie quite limp and useless or if they can be raised it is only very slightly and with great difficulty.

The grasp of the hand is often diminished in ordinary cases.

When the neck muscles are involved to any great extent the head may hang forward or roll about on the shoulders quite out of the patient's control. It is a very striking condition.

If the muscles of the back suffer the spine becomes curved with the convexity backwards and the child is unable to straighten himself or sit up.

Other trunk muscles may be affected in very widespread cases but total involvement of all the muscles of the trunk and limbs is rare.

Face. Paralysis of the muscles of the lips and cheeks and even of the whole face occurs in some very generalised cases. The features then remain in absolute rest, and expressions of pain or other sensations are quite impossible. Unilateral

facial paralysis has been recorded sometimes of the whole side but always more marked in the lower half. It is mostly incomplete.

Where the lips and cheeks are much affected dribbling of saliva becomes very troublesome. As swallowing is very difficult and apt to cause choking the patient will not voluntarily attempt it and prefers to allow the saliva to run on to his pillow. It has been thought that in these cases the secretion was much increased but this is often more apparent than real. In two cases reported in Appendix I + there was distinct facial paralysis, in one the whole of the right side of the face, in the other only the lower half of one side. Such cases are generally very severe and are very liable to end fatally.

Larynx and Vocal cords Some authorities have laid considerable stress on the frequency of paralysis and anaesthesia of the epiglottis and upper part of the larynx, and where there has been a tracheotomy wound have introduced a probe upwards and proved the absence of reflex coughing.

The character of the voice is difficult to make out in most cases due to the distinct nasal tone. Generally there is but little hoarseness and no aphonia. The cords may, however, be paralysed, sometimes only one, sometimes both. The former is the more common condition. The child is brought up quite aphonic and laryngoscopic examination shows one or other cord in

---

+ Cases 2 and 3.

the cadaveric position. This single adductor paralysis is probably the most common lesion. Double abductor paralysis has been recorded. In one case at Paddington Green Children's Hospital the child was brought in with this condition and tracheotomy had to be performed at once. In another case it was observed only when the tracheotomy tube was taken out. It was then found that the child could hardly breathe and the tube had to be re-inserted. That this was not due to fright on the child's part was proved by the laryngoscope. Complete recovery of function occurred after some weeks.

Cord paralysis is more frequent after tracheotomy cases and this may account in part for the long time it seems to last, a kind of functional paralysis due to want of use taking place+. It may be months before the restoration of function is complete. Several of the cases in Appendices I and II had cord paralysis lasting from a few days to several weeks.

Bladder and Rectum. Incontinence of urine is not an uncommon occurrence, especially with widespread paralysis. McKenzie noted it on several occasions. It usually lasts but a few days and may vary from day to day. In one case which I have detailed further on this symptom lasted for a considerable time. (Append I. Cases 2,3,10)Retention may sometimes occur. Trophic changes are very unusual.

+ It is just possible that adhesions may form and restrict movement for some time.

Only in severe cases is the rectum affected,[††] and then only towards the close of the case. Then we may get constant passing of urine and faeces.

Knee jerk. The condition of the knee jerk is one of the most important points not only in the investigation and diagnosis but also in the prognosis.

For a long time its presence or absence was not looked for or if noticed had but little importance attached to it. More recently, however, its true significance has been appreciated and valuable statistics particularly those by McKenzie[+] have been prepared.

In almost all cases of diphtheritic paralysis the knee jerk will be found at a very early period either absent or elicited with difficulty.

The exact time of its disappearance varies a good deal and seems to bear a distinct relationship to the severity of the preceding throat affection. That it may be lost very early in the acute disease in some cases soon after the membrane appears on the throat is a well known fact. And experience has shown that the prognosis is thereby made much graver. It shows to what a considerable extent the system has been invaded by the diphtheritic poison whatever its nature may be; and the percentage of deaths from the acute disease or from severe paralysis coming

---

[+] St. Thomas's Hospital Reports 1890.

[††] See Appendix I cases 2 & 3.

on during it is in such cases very high. Early exaggeration has been observed in some cases as pointed out by Buzzard, Herringham, Money and others. This however does not last long, soon giving place to sluggish reaction, and finally to total abolition. The frequency of this primary condition can only be surmised: it is essentially an early symptom coming on generally before the others and consequently before the patients are brought under the notice of the physician.

Sometimes even with well marked paralyses there is persistence of the knee jerks throughout. This was shown in the case of a little girl in Paddington Green Children's Hospital. On admission there was well marked palatal and ocular palsies but the knee jerks were rather brisker than usual. A few days later they were less so but remained practically normal during the remainder of her stay in the Hospital, a matter of five weeks or nearly eleven weeks from the date of the diphtheria.

The condition of the knee jerk is generally the same on both sides but differences between them, even unilateral loss have been noted.

With young children there is sometimes considerable trouble in examining this reflex. They have a trick of keeping the muscles in a state of rigidity or just short of it which may make it seem absent when with complete relaxation it would be obtained. By attracting their attention to a toy or in some other way we may generally get sufficient relaxation to arrive at an accurate conclusion.

In examining from day to day we must on no account have the patient sitting up in order to hang the legs over the side of the bed as one does in ordinary cases. The horizontal position must be maintained. We may turn the child across the bed and allow the legs to dangle over or what is better we can raise the leg we are examining, by one hand under the popliteal space, till a right angle is formed. With the leg in this position supported by the heel resting on the bed we can feel the contraction of the vastus internus in cases where no actual jerk is obtained.

A false jerk due to voluntary or semi-voluntary movement of the limb on the part of the child is not uncommon but we can guard against this by tapping the front of the tibia when it will occur all the same.

The date of the disappearance varies a good deal as I have just said with the severity of the preceding diphtheria. Both Bernhardt and McKenzie have proved that it is lost in at least 60% of all cases of diphtheria whether there follow paralysis or not. McKenzie's statistics on this point are elaborate and interesting.

Speaking generally the fifth week is the most common time for the disappearance to occur but the statistics just referred to show that no absolute date can be fixed.

The condition persists mostly for many weeks often indeed for many months. I have failed to elicit it after three and

four months and McKenzie records one case where at the end of nine months it was still absent.

Sensory troubles. In the case of children investigation into the condition of sensation is not only difficult but untrustworthy. Where there should be well marked reflexes due to the stimulation of a part as on touching the palate or pharynx and we cannot elicit them we are not so much at a loss; but in the matter of cutaneous sensations children are apt to give very contradictory answers. In older children and adults it is generally found that sensation is distinctly impaired in the affected limbs especially at the extremities, i.e. the fingers and toes. Light touches are no longer readily recognised and no distinction is made between the point of the finger and the head of a pin. Localization is often inaccurate but never very far out if the touch be firmly made. Complete anaesthesia is very rare. Heat and cold are recognised correctly.

In many patients sensation of pins and needles is complained of in the feet and hands and this may precede the actual diminution in power.

In one very interesting, and so far as I am aware unique case, which I detail at length later on (see Appendix I case X.) there seemed to be a distinct loss of muscular sense.

Inco-ordination in both hands and feet occurs not infrequently.

All these sensory changes last but a short time compared to the paralysis. They disappear before movement is restored. Thus in the palate anaesthesia may last a week, then disappear, and a day or two later the voice becomes less nasal and in another week or so quite normal.

The Superficial reflexes. are generally exaggerated. Sometimes the plantar reflex is much dulled or even entirely abolished. This is often found where the gait is ataxic and the instability quite out of proportion to the loss of power.

Heart and Lungs. Leaving aside for the present the cases with only cardiac symptoms, it is, from a clinical point of view, much better to consider these two organs together.

They are generally both attacked though the one may precede the other by a longer or shorter interval. Despite the exceptions that have been taken to it Cadet de Gassicourt's term of "Cardio-pulmonary" for these complications is one of great use at the bedside. Alfred Suss+ and others have objected to it as being incomplete, that from the frequency of abdominal symptoms gastro-cardio-pulmonary would be more correct. As will be shown further on this objection is from a clinical point of view of not much value. No doubt symptoms referable to the abdomen often precede or accompany, indeed, often herald the approaching implication of the heart and lungs; yet the outstanding features of the case are essentially cardiac or pulmonary or both acting together. It is on this understanding that the term is here used.

In all cases of diphtheritic paralysis, be they of a slight or of a grave character, the one thing which dominates prognosis and treatment is the possible occurrence of this dreaded complication. And this is necessarily so; we can never tell when it may not come on; the case may seem progressing favourably, and the prognostic horizon quite clear yet in a few hours death may supervene.

+ De la paralysie Diphthéritique du Pneumo-gastric. (Revue Mensuelle des Maladies de l'enfance. July 1887).

Cardio-pulmonary paralysis may come on during the course of diphtheria - as soon as the membrane has gone, or from this time to six or seven weeks later.

Of its early occurrence, that is soon after diphtheria, I have the notes of only one case (See Appendix I. Case 1.). The following cases from C. de Gassicourt will suffice.

CASE I. Child of two years of age admitted with diphtheria of two days duration - larynx not implicated - no albuminuria. Two days later membrane not so thick but palate paralysed(shown by nasal voice). Next day paralysis the same, throat clearing up, but at 1 p.m. sickness came on suddenly, followed by dyspnœa with alæ nasi acting. Pulse 160, cyanosis of face and extremities, slight cries of distress and at 2 p.m. child raised herself up, gave a loud cry, and fell back dead.

CASE II. Child of five years, admitted January 5. Diphtheria of two days duration - larynx free, no albuminuria. On 12th. January membrane gone; paralysis of palate. 13th. January no change in the morning but vomiting during the day; rested well at night, but at 7 a.m. on the 14th. gave several cries, respiration became embarrassed, dyspnœa appeared, pulse became rapid, cyanosis at first limited to face and hands but soon becoming general, and in a few hours child died without any cries or agitation.

These two cases shew typically the cardio-pulmonary complications coming on, in the one case with membrane still present, in the other a few days after its disappearance; and both proved fatal very speedily. Both had,it will be seen,premonitory abdominal symptoms.

From the present state of knowledge on the subject any classification of the clinical forms and varieties of this complication is almost impossible. Cases might be divided into rapid and prolonged, into those with definite crises (of which I will speak later on) and those without, or again according as one or other organ was primarily or mainly affected.

Perhaps the recital of several cases will bring out the chief clinical features best.

CASE III. W. A., aged two and a half years, admitted into Shadwell Children's Hospital under Dr. Coutts on December 3rd. 1894. Had diphtheria five weeks previously and one other child died from it. Regurgitation came on in second week of illness and continued till three days before admission. He could not walk when taken out of bed (this was of course before admission) and had been "weak in his back" for three or four days.

On admission there was noted marked lassitude, paresis of muscles of head and trunk - head rolling about on shoulders; respiration mainly costal; diaphragm acting very feebly; no regurgitation; no squint. Head and lungs normal to auscultation and percussion. December 5th. Child worse - lies quietly

on his back but is very irritable when touched; diaphragm scarcely acting becoming slowly paralysed; respiration almost entirely costal; heart and lungs normal; swallowing very bad, requiring feeding by nasal tube.

December 6th. Respiration feebler and more rapid; much sweating.

December 7th. At 8 15. p.m. child got suddenly much worse, became cyanosed; breathing very irregular; given injection of Digitalis, brandy etc. At 8.45 lips black, face dusky, clonic spasm of lower jaw. Pulse imperceptible. Death.

Here there was evidently much embarrassment to respiration from the diaphragmatic paralysis, but the sudden onset of the fatal symptoms pointed to the heart and lungs.

CASE IV. V. W., aged two and a half years, (F.) admitted to Paddington Green Children's Hospital, September 23rd. 1890., in third week of paralysis. Usual symptoms present. History of sore throat four weeks previously. Well nourished, healthy looking child - screamed passionately until exhausted, whilst being put to bed, and mucus began to collect in pharynx and air passages. Slept and took well during day.

24th. Attack of vomiting at midday. At 3 p.m. child dull, eyes semi-closed and turning up, pupils equal, dilated; colour good. Pulse 130 reg., vol. good; temp. 100·4, resp. 44. Irre-

gular, gasping, forcible inspirations. Expirations short and weak. Deep sighs at intervals. Weak loose cough. Signs on auscultation of capillary bronchitis. Ordered Strychnia c. Atropia ãã 1/100 gr. sub. cutem. Revived for the time, but towards evening temperature rose to 102·4 and dyspnœa again became urgent. Dry cupping seemed to relieve condition.

25th. Unable to swallow all day, so fed by nasal tube and enemata. Respiration not embarrassed.

26th. 9.30 a.m. Severe attack of dyspnœa. Child seemed moribund - extremely restless - face bluish - eyes glazed and turned up - neck extended - lungs full of mucus - gurgling throat râles - pulse extremely weak and irregular, 148. Strychniæ ℈ Atropia gr.1/100 injection repeated. Revived within an hour after much vomiting of dark greenish brown fluid. Asked for drink and swallowed brandy and milk readily. Breathing much easier. Râles in throat and lungs almost disappeared. 6 p.m. another attack - similarly treated - slept, and swallowed well during night.

27th. No more crises. Respirations quiet and regular - Pulse 122, good and regular. Occasionally chokes over fluids. Voice, a hoarse nasal croak.

Oct. 2nd. Progress good. Respiration still sighing at times. Pulse fallen to 84. Double ptosis. Internal strabismus. Voice still hoarse, weak, and nasal.

16th. Discharged with no other symptoms than slightly nasal

voice and loss of knee jerks.

(This case of Recovery is of great interest).

CASE V. R.S. admitted to Shadwell Children's Hospital on September 25th. 1894, under Dr. Donkin. Had diphtheria six weeks ago. Regurgitation came on three weeks ago and has persisted. On admission there were noted much lassitude amounting to apathy - paresis of muscles of neck, head rolling in all directions; palate acting very slightly; regurgitation of fluids. Heart and lungs apparently normal. Progress was uneventful till October 2nd. when child made a choking noise whilst lying quietly in cot. Nurse ran to him and found he was not breathing. Heart acting very rapidly - pulse quite uncountable. Strychnine and brandy injected. Heart stopped in a few minutes Choking from entrance of matter into larynx was definitely eliminated.

CASE VI. E.H. aged five and a half years, admitted into Paddington Green Children's Hospital, January 24th. 1890. History of sore throat five weeks ago; three weeks ago fluids came back through nose and voice became nasal. Two weeks ago, complained of aching legs. One week ago gait became staggering. On admission, healthy looking, well nourished child. Heart and lung sounds normal, pulse 100 regular and of good volume. Respirations easy and regular. Temp. 99-99.8. Tonsils enlarged and red. Soft palate motionless. Voice markedly nasal. Knee jerks absent. Walked unsteadily, swaying from side to side.

27th. Regurgitation of fluids occasionally. Voice as before. Slight ptosis of left eye-lid.

31st. No regurgitation of liquids when swallowed slowly. Bowels confined. Pulse 100, regular and good. Temperature normal [illegible].

Feb. 4th. Swallows quite well, but no other improvement. Lies on back without attempting to move. Smiles when spoken to, but nevers answers, unless pressed to do so - then with obvious effort. Takes a sudden deep inspiration, and voice is produced in gasps, and is weak, hoarse and nasal.

Feb. 9th. No change, except that the pulse had risen from 80 to 120 during the last few days. This morning the pulse was 136, very weak, and irregular. A few hours later the child became suddenly very restless, with cyanosis. Pupils widely dilated. Alæ nasi working vigorously. Neck extended. Submental muscles bulging with each respiration like those of a frog. Respiration 56, gasping, and irregular. Intercostals only, working. Diaphragm retracted with inspiration, bulging with expiration. Much accumulation of mucus in throat, and loud râles heard all over chest. Pulse weak, irregular, 160. Vomiting from time to time. Quite unable to swallow. Conscious. When asked if in pain, pointed to throat. Temp. 103·6.

Ordered injections of strychnia 1/100 gr. every hour. Mustard poultices to epigastrium. Nutrient enemata.

10th. Seemed to rally slightly from time to time, but improvement only temporary. Faradisation of diaphragm was tried

and digitalin 1/100 gr. added to strychnine injections. Died 30 hours from the onset of the crisis.

Remarks. This case illustrates well the sudden onset of a crisis, and also the warning of danger afforded by the increased pulse rate.

An unusual form is given by C. de Gassicourt. A boy of twelve years who had paralysis of palate, muscles of neck, arms and legs, and loss of accommodation which had lasted three weeks.

He seemed to be getting better when next day at 7 p.m.he cried out, remained motionless and asphyxia slowly appeared without any other symptom except cyanosis. At 10 a.m. next morning the note made was:- Face earthy and blue, extremities cold and cyanosed, tongue white and moist. Respiration hurried and anxious, but sounds very pure to auscultation. Heart acting rapidly 148 to the minute but quite regular. Sphincters of rectum and bladder paralysed. Eight minutes after this note was made boy died suddenly.

In most of these cases it will be seen that death occurred suddenly by a kind of crisis and before going further it will be necessary to describe these more fully.

Physicians on the continent and more particularly in France have for a long time recognised the occurrence of these crises, especially in connection with the cardio-pulmonary complications.

These crises vary a good deal in appearance; some related

more to the heart - others to both heart and lungs, so that considerable difference has existed as to the exact meaning to be attached to the term.

Some physicians have preferred to limit it to the more or less marked anginiform attacks, while others have given it a much wider application. Even now the point is far from being definitely settled, the actual pathological condition being as yet uncertain. Alfred Suss who referred all the cardio-pulmonary (and abdominal) symptoms to lesions of the pneumo-gastric, took naturally a different view from those who regarded the crises as bulbar in origin or from those who looked upon them as simply attacks of syncope. The fact of the occurrence of the phenomena is equally admitted by all. Guthrie+ who has studied the subject carefully regards them as bulbar in nature and likens them to the crises occurring in loco-motor ataxia. The importance of the subject can hardly be overestimated. Most writers speak of the frequency of these crises; in my opinion the term should be the constancy of them, for I hold that in 80 per cent. of the deaths in diphtheritic paralysis (apart from such accidents as choking etc.) are due to a crisis, it may be the first and only one, or the last of a series. And this I have convinced myself of not only from the cases I have seen, but from the large number I have examined in the literature of the subject.

+ Lancet, 1891. Vol. I.

These crises may be divided clinically into two chief forms; the grave form implicating usually both heart and lungs and generally ending fatally; and the mild form which is mainly cardiac and mostly followed by recovery. We will take the latter variety first.

In appearance it is as I have just said typically cardiac. It may vary from an attack closely resembling true angina-pectoris with pallor of the face, coldness of the extremities, sudden slowing of the pulse, præcordial oppression, and pain which may radiate down the left arm, round to the angle of the scapula, to the shoulder etc., and with a sense of impending dissolution: to sudden attacks of cardiac irregularity and dyspnœa without any pain or oppression. These attacks are generally brought on by some exertion or violent emotion. Straining at stool is a not uncommon cause in adults, whilst in children a fit of anger - and most children suffering from diphtheritic paralysis are liable t them if aroused or interfered with in any way - may be the excitant. As regards frequency they vary from a single attack up to several, generally with a good interval, a day or more, between them. The first is usually the most severe, those succeeding becoming milder. Another interesting point is that they mostly occur in mild cases where there is but slight paralysis. There are no definite premonitory symptoms, the exciting cause bringing the attack on suddenly.

The following typical case recorded by Suss+ illustrates many of these points.

A young House-surgeon had diphtheria followed by paralysis of palate. Whilst at stool he had a sudden attack of præcordial pain, shooting down the left arm and through to the angle of left scapula. The pulse fell from 84 to 47 per minute. A companion auscultated his heart at once but found nothing abnormal. Three days later the same thing occurred and the third and last a day or two afterwards. The last was not nearly so violent. He recovered.

C. de Gassicourt mentions a case with three similar crises where there were also dyspnœa, palpitation and cardiac irregularity, and where likewise the first attack was the most severe, the second and third diminishing considerably.

With children a fit of anger is as I have said the common excitant. One case I remember well. A little girl three and a half years of age was admitted to Shadwell Children's Hospital with aphonia and slight strabismus. There was a definite history of diphtheria some weeks previously. The child was languid and sleepy, and, if left alone, the heart acted quietly and regularly On attempting to examine her throat she became very angry - turned pale, had slight dyspnœa and marked tachycardia and irregularity. The same thing occurred next day when Dr. Donkin, under whose

---

+ loc. cit.

care she was, tried to repeat the examination. Auscultation showed the heart sounds to be quite clear. She was unfortunately removed by her parents in a few days and lost sight of.

In these cases we have what may be termed a severe and acute functional derangement of the heart, occurring without any warning. Functional it may be, for physical examination shows nothing wrong and there have been no previous symptoms to lead one to expect any such phenomena. On the other hand it may be that there is actually some predisposing cause, for instance some fine change in the cardiac nerves which in ordinary circumstances does not give rise to any symptoms but under any exciting cause as in the straining at stool, or the fit of anger in the cases I have mentioned, makes itself apparent. In the present state of knowledge it is almost impossible to decide this question.

Coming now to the graver form we find many marked differences, there is often no exciting cause, the attacks generally implicate both heart and lungs, they increase rather than decrease in severity, and the result is generally fatal. Besides these the condition is more definitely an acute exacerbation of already existing symptoms. But calling it an exacerbation does not help us much out of the difficulty of causation, or rather localisation. If we have a child with both heart and lungs implicated, the one shewn by a rapid and irregular pulse, the other by deep sighing and irregular breathing, and he has a well marked crisis, are we to explain it by changes in the vagus trunk, or

peripheral filaments as Suss would, or must it be regarded as an invasion of the medulla, where not only are the roots of the vagi involved but probably also the respiratory and other centres? Guthrie has well argued for this latter explanation, and the parallel he draws between the crises in locomotor ataxy and these here considered is very apt from the point of view of implication of the central nervous system.

This grave form may occur at any period of the illness (from the actual diphtheria onwards) and with any form of it, with simple paralysis, multiple or cardio-pulmonary. It is, however, much more common with the two last; cardio-pulmonary complications indeed, so seldom occur without one or more crises that we will in continuing naturally include that subject as well.

A typical crisis presents a most striking picture. The apathy of the child changes to restlessness; the face becomes pale and anxious; the pulse is rapid, fluttering, and irregular; the breathing is quick and jerky with inspiratory gasps, and the stethoscope shows abundant râles all over the chest. Soon dyspnœa and cyanosis more or less pronounced appear, and death seems inevitable. Occasionally at the height of the paroxysm vomiting will occur, of mucus mainly, and some relief is given to the embarrassed respiration. Often the temperature will be found to have risen to 101°-102°F. or even higher (See Case II.Appendix I.) If death do not supervene, then the symptoms gradually abate, breathing becomes easier, the lungs freer from râles, the anx-

ious expression gives place to one of relief and the child may even fall asleep. Often, however, the amelioration, though great compared to the recent condition, is not enough to give any feeling of relief, and he lies gasping and in evident terror of another attack.

In almost all cases the condition in the interval is one of great prostration, with the heart and lungs acting rapidly and irregularly, and the whole aspect as if another paroxysm might occur at any moment. Here we have evidently an acute exacerbation of both cardiac and respiratory symptoms. If these had appeared gradually, with increasing dyspnœa and cyanosis, we would have naturally looked on the condition as one of progressing cardio-pulmonary paralysis. Instead, however, we have a sudden increase, it may be the matter of only a few minutes, and "crisis" is the only term we can fitly apply. But if we have a child dying in the first crisis,without having had any very apparent cardio-pulmonary signs, how are we tc call it definitely a crisis occurring in cardio-pulmonary paralysis?

This question brings us to the consideration of prodromata. There are several well marked premonitory symptoms which I shall detail presently, and which herald the approaching implication of the heart and lungs. Now if we have recognised these as present we will have indisputable evidence that the cardio-pulmonary apparatus is no longer free, and the early appearance of a crisis will not appear puzzling. The prodromata may be divide

into two classes: (1) Cardiac. (2) Abdominal.

(1) As regards the cardiac, increasing rapidity of the pulse from 80 or 90 to 120 or more in a day or two without any evident cause, such as temperature etc., is a very bad omen, and should warn us not only that the heart is affected but that a crisis is imminent.

H. Weber, Suss, and others have observed slowing of the pulse before the acceleration, probably due to irritation of the vagus.

Præcardial oppression and even pain are sometimes complained of especially by adults before the grave symptoms appear.

(2) Of the abdominal symptoms, pain and sickness are the chief. The pain may be umbilical, epigastric, or more or less generalised. It is often described by the patient as of a colicy nature. It other cases it is more or less fixed. Sometimes the pain is accompanied by calls to stool, but actual diarrhœa is rare.

More important still is sickness. This has rightly been regarded as a very bad omen. It may come on a considerable time before the grave symptoms. There is nothing peculiar about the vomited matter.

Rise of temperature precedes the crisis in many cases.

The breathing often gives us help. It will generally be found rather shallow with occasional deep sighs, and auscultation may show fine râles appearing at the bases of the lungs.

The value of all these signs of course depends to a great extent on the length of time they appear before the actual crisis. Twelve or twenty-four hours or longer periods may sometimes be gained. This however is only with experienced physicians. A casual examination even from day to day may reveal nothing apparently much the matter. The tachycardia may be ascribed to the excitement of the visit, the sickness to injudicious diet and similarly with the abdominal pains. In hospital where records of the pulse etc. are taken regularly and accurately we may be able to foretell a crisis by many hours. In one case (Case VI, page 37.) the heart was noticed to become more rapid in action during a period of several days and when finally the fatal symptoms appeared they were not unexpected. Suss records a case where sickness gave a twelve hours warning. (See Appendix I, case I. where sickness came on several days before death) In most instances we can gain several hours and the importance of this if only from a prognostic point of view is very great.

Guthrie has well summed up the state of the child in whose case crises may be expected.

"It is in children with the above symptoms, viz.,

(1) Marked listlessness and apathy.

(2) A weak hoarse and nasal voice.

(3) Irregular and sighing respiration.

(4) A loose, weak and almost noiseless cough.

(5) A rapid pulse, together with the minor signs of

"diphtheritic paralysis - that bulbar crises are apt to occur."

As regards the frequency of crises there is not much latitude. There may be only one and that fatal or there may be three or four. The intervening period varies from an hour or two to several. Guthrie has recorded forty-eight hours and longer. In cases 2 & 3 Appendix I the critical period was of considerable length. Rapid pulse, sickness and sighing respiration all foretold the coming crises. This however is an exceptionally long time.

The duration of the crisis is much shorter generally a few minutes up to an hour. Recovery is rare.

In all these cases of cardio-pulmonary involvement it will be found that the lungs soon tend to become choked up with mucus. If vomiting occur much relief is generally obtained; but whether or not, as the crisis passes off the lungs become much clearer. What is this accumulation due to? The generally accepted explanation is that there is paralysis of the muscles of Reissessen due to the involvement of the terminal twigs of the Vagus in the lungs. Whether this be so or not there is no doubt a distinct inability to expectorate or cough up mucus which therefore accumulates. Besides this there is probably some acute oedema of the bases of the lungs coming on during a crisis and passing off rapidly as the child begins to breathe more easily.

The effect of atropine in these cases is often very strikingly beneficial.

These cases of involvement of the heart and lungs are unfortunately not uncommon and the result is almost invariably death. C. de Gassicourt had 15 out of 128 cases of paralysis and of these 15, only one recovered. Alfred Suss had five cases one of forty all fatal. Personally I have only seen six cases all of which died.

Death generally occurs more or less suddenly; the child has a crisis, gets a little better, but is still very blue and dyspnoeic, then suddenly dies; or he may die more gradually being simply choked by the mucus in the air passages.

In appendix I, I have given several cases which show marked crises. Where we have a child under continuous observation no mistake can possibly occur about the nature of a crisis but if a child be seen for the first time at the height of one it is very probable that some wrong diagnosis such as capillary bronchitis will be made.

In reviewing now the whole subject of cardio-pulmonary complications it will be seen that there is a very great difference in the clinical aspect of the cases. The conclusions however, may be summed up as follows:-

(1) The cardio-pulmonary complications may occur during or after diphtheria, mild or severe though much more frequently in connection with the latter and particularly if the larynx has been involved.

(2) They may come on at any time, early or late in the paralysis and with all forms of it.

(3) They may be heralded by several fairly trustworthy signs of which tachycardia, sickness, and abdominal pain are the chief.

(4) In most cases there are crises more or less well marked, particularly towards the close.

(5) There may be considerable differences in the clinical features of the cases according as to whether one or other organ suffers the more immediately.

(6) A fatal result is the rule; it may be suddenly, in a minute or two, or after several hours.

PATHOLOGY.

In few diseases is the pathological condition so little understood as in diphtheritic paralysis. Why this should be so may seem at first sight strange: there have been many cases examined post-mortem and many skilled pathologists have worked at the subject. In reality, however, the difficulties are numerous, and so far Trousseau's prediction that we should never know much about it seems like to be verified.

The first great difficulty is the extraordinary variety of the symptoms and of their course. Thus we have:-

(1) The date of appearance, sometimes early, sometimes late.

(2) The extent, sometimes limited, at others widespread: affecting only the voluntary muscles or including involuntary as well.

(3) The symmetry in many, the want of it in other cases.

(4) The absence of definite groupings.

(5) The absence of marked trophic changes.

(6) The occurrence of well marked crises.

(7) The mutability of the paralysis.

(8) The rapid and complete recovery in favourable cases.

How can we fit all these into one theory or indeed where can we rationally begin our investigations?

Pathological examination has in many cases been very complete; the heart and lungs, the paralysed muscles and their

nerves, and the whole nervous system have been searched for some constant change that will offer a satisfactory explanation of all cases.

So far the results have been disappointing. Some observers have found one lesion, others have failed to find it but have discovered another, whilst in many instances no departure from the normal could be detected.

Leaving aside the theories of Trousseau and Gubler, the first definite changes recorded are those of Charcot and Vulpian in 1862. They examined the nerves of the paralysed palate and found well-marked degeneration. Ten years later Liouville observed similar changes in the phrenic nerves; and in 1875 Roger and Damaschino found the same condition in 4 cases.

In 1867 Buhl shewed a case where the anterior and posterior roots were doubled in volume due to thickening of the neurilemma, and presented bloody extravasations.

Professor Pierret in 1876 found disseminated patches of meningitis on the cord, but Vulpian later on in the same year failed to find this but found changes in the anterior horn of grey matter.

Thus we see pathologists had gradually worked up from the nerves, to the roots, to the meninges, and finally into the cord itself.

Of great importance are the more recent researches of Gombault and Déjerine.

Gombault examined 3 of C. de Gassicourt's cases which had proved fatal with cardio-pulmonary symptoms. C. de Gassicourt gives a full description of the results in his treatise on the diseases of children. Here it will suffice to say that in all 3 cases changes in the anterior roots with swelling of the axis-cylinders were discovered. In one case the heart and vagus nerve were examined and found healthy.

Déjerine[+] in 5 cases noted lesions in the anterior roots and in the grey matter of the cord. The white matter was healthy.

Of other workers, Oertel saw changes in the anterior horns: and Hochhaus failed to discover anything wrong in the central nervous system or in the peripheral nerves, but found the paralysed muscles affected. Indefinite bulbar changes have also been recorded by some authorities.

Now though we have from these researches lesions discovered in four sites, in the muscles, in the nerves, in the anterior roots and in the cord, none of them can explain anything like a majority of ordinary cases nor indeed all the symptoms in the cases where they occurred.

Experimentally the same lack of uniformity has been experienced. Roux produced diphtheritic paralysis in two rabbits and examined the nerves through the muscles, in some instances right to the end plates, without discovering any lesion. On the other hand Hallion and Enriquez have recently found lesions

---

[+] Archiv. de Physiolog. normale et Patholog. 1878.

in 3 dogs under whose skins they had injected diphtheritic toxines. These lesions were in the roots and in the cord, and consisted in congestion and haemorrhages, and in 2 out of the 3 cases, in foci of destructive myelitis localised especially in the white substance.

Of extreme importance are the investigations of Roux and Yersin.+ According to them we can have, in many animals, paralysis following the introduction of cultures of the microbe (Klebs-Loeffler) into the pharynx or trachea or into the veins; and also by the filtered products of these microbes (a Chamberland filter was used). This last point is particularly noteworthy and shows that diphtheritic paralysis in man is probably of a toxic nature.

As so many cases die with symptoms of cardiac involvement it was quite natural that attention should early be drawn to the heart. Here also many theories based on misconstrued appearances were advanced. Some of the investigators, even, seem to have considered the cardiac complications as something quite apart from the paralysis, as for instance, the supposed endocarditis of Bouchut and Lagrave. The lesions were soon proved to be haematomata on the edges of the valves and to be quite distinct from any inflammatory process.

The two main theories, however, have been the cardiac, and the nervous.

+ For this and many other points see article by Babinski in "Traité de Medicine" (charcot, Bouchard, et Brissaud) Paris. 1892—

For the first Werner (as early as 1842), Winkle, Richardson, Barry - Meigs, Beverley-Robinson, etc. have recorded cases in which they attributed the death to clots forming in the heart. That their conclusions were probably wrong and that what they saw were the ordinary antemortem clots has been abundantly proved by C. de Gassicourt and others.

For the second we find more numerous and more powerful supporters. First of all Perate in 1858 called it paralysie cardiaque; Gubler in 1861 referred the symptoms to the vagus; Duchenne termed it "Paralysie bulbaire diphtheritique;" whilst Revilliard and also Sanne have regarded these complications as due to extension of the paralysis to the terminal twigs of the pneumogastric in the heart and lungs. Now-a-days we take it for granted that what is true of the other paralysfs is probably true of this complication, and that more accurate knowledge will explain to us why we should get it in one case and not in another. In examining the theory of neuritis being the main factor in diphtheritic paralysis we find numerous objections; the frequent absence of marked paralysis, the absence of marked trophic changes, the mutability, and the rapid and perfect recovery. It has been sought to account in a measure for these by supposing that in most cases the neuritis is far from complete, that only a few fibres in the nerve are affected. Such a condition has actually been observed but the explanation has not been regarded as satisfactory.

Against the cord changes being the chief and the most constant we have the absence of definite groupings in the paralysis. With such well marked lesions we would naturally expect a group of muscles to be involved and not the picking out of a muscle here and there as is generally the case. But whilst neither neuritis nor cord changes can of themselves elucidate the question we cannot deny their existence; and viewed in the light of the researches of Roux and Yersin, and Hallion and Enriquez they become of extreme interest. Since it is possible for the toxines alone to produce such changes as these observers have described we can well understand how they can occur in diphtheritic paralysis in the human subject; and the considerable time they there take to produce their effects need not seem astonishing when we remember the much longer period in hydrophobia.

But with this theory we have also difficulties to explain away, as for instance the unilateral paralysis as we often see it in the eye and occasionally in the palate and vocal cords. And the fact that there are seats of election for the paralysis may seem contrary to the idea of a general infection. In reality however, this is not so. Many diseases show a similar state of affairs. Lead affects the extensors of the forearm mainly, alcohol, both arms and legs before it becomes general. The constancy of the lesions in typhoid fever is of the same nature.

And for its want of symmetry have we not the same thing in pneumonia? The pneumo-coccus entering probably by the air passages does not in general attack both lungs but only one or a part of one; and to explain this by the existence of some local weakness allowing the organism to multiply would argue for a condition of affairs in the majority of human beings that most physicians would hesitate to accept.

Granting then this explanation of the want of symmetry in all cases and of the definite seats of election for the onset we find that the toxine theory accounts far more completely than any other for the various symptoms.

As a matter of fact we are still in the stage of theory and till further researches have been made and more light thrown on the subject it is needless to enter into elaborate speculation.

So far nothing sufficiently definite has been reached to aid us in the treatment of the actual condition.

DIAGNOSIS.

As a general rule in well marked cases there is little difficulty in this respect; the listless apathetic appearance of the child, the characteristic voice, the short ineffectual cough and the presence of squint or other paralysis, form a clinical picture that can hardly be mistaken for anything else.

But when we have presented to us a child suffering from cardio-pulmonary paralysis with face and extremities cyanosed and cold, respiration shallow and laboured and the lungs full of fine râles, it is extremely difficult, nay, sometimes almost impossible to come to a correct and definite conclusion. One such case I remember of a girl of 10 years admitted with signs and symptoms much as above. General tuberculosis was diagnosed. She died a few hours after admission and only then did we learn that her illness had been of but a day or two's duration. A post-mortem examination was unfortunately not obtained. A few days later another girl of about the same age and in much the same condition was admitted. Here, however, we had from the first a definite history of diphtheria with regurgitation and subsequent paralysis. She had three crises and died in the third having only been in Hospital about twelve hours. A post-mortem was made which excluded tuberculosis, capillary bronchitis, etc.

It is in early and slight cases that we in general experience the greatest difficulty in making a diagnosis and where it is of the greatest importance. When we have a child brought to us with one or more signs of paralysis our first thought naturally turns to the question of recent diphtheria. Here the difficulty often is very great. The malady may have been of so slight a nature that it was never noticed, or if noticed misjudged; the child may have been attending school regularly for

months past without complaining of sore throat, and the parents are most emphatic as to his having enjoyed perfect health. In short to any direct questioning as to recent diphtheria we in more than half the cases get a decided negative as answer. Indirectly we may gain sufficient evidence to set the matter at rest. A history of swelling of the neck or in the throat, "lumps in the throat", which may refer either to the inside or the outside, difficulty or pain in swallowing, difficulty in breathing, discharge from the nose particularly if bloody, may be elicited; and we may learn that there have either been actual cases of diphtheria in the same house, in the neighbourhood, or at school, or some other members of the family have suffered from sore throat some weeks previously. In the case of very young children this want of a definite history is the rule, the child has a sore throat but is unable to complain and the mother notices nothing amiss or merely that he does not seem to care for his food and cries when he drinks, symptoms attributed to teething or mere peevishness. The child recovers and when several weeks later it is brought up to the hospital with regurgitation or squint or other paralytic symptoms it is not wonderful that the closest questioning fails to elicit any history regarding a recent sore throat. Older children on the other hand can often tell of having had a sore throat some weeks previously but of so slight a nature that they did not complain of it and that in a few days it was quite better. Reference to the accompanying

cases will bear this out very fully.

Though nasal voice is in the majority of instances the first symptom to appear it is often not regarded as anything of consequence by the parents being put down to cold in the head, etc., and it is generally the falling about and the staggering gait due to paresis of the legs, or the squint, which leads them to seek advice. Sometimes the squint or regurgitation are only observed after and attributed to a fall caused by the condition of the legs. This was the case with Alice B - . (Appendix I case VI). In deciding whether a child shall be treated as a case of diphtheritic paralysis we have to consider:-

(1) The history of recent sore throat bearing in mind the fallacies just mentioned and the worthlessness of negative evidence.

(2) The presence of definite paralysis more especially of the palate, eyes and legs, of recent date.

(3) The state of the knee jerk. This is very helpful, for while it may, as I have said, be present or even exaggerated in some instances, yet in the majority it will be found absent.

(4) The general aspect of the child.

Whenever therefore we have a child with nasal voice and absent knee jerks whether we get a history of diphtheria or not we will be right in 90% of the cases to treat it as suffering from diphtheritic paralysis. Of the affections which might be

confused with it, infantile paralysis may be mentioned. It can generally be easily recognised. In older children and adults hysteria and Landry's paralysis may simulate wide spread cases. Alcoholic and other neuritis may also have to be excluded, particularly as in adults the legs are sometimes the first part affected in diphtheritic paralysis.

As regards individual symptoms we may have post-nasal growths producing a nasal tone of voice, but the aspect of the child, the snoring and sleeping with the mouth open and the chronicity are generally enough to lead us right.

Examination with the finger will of course settle the matter.

Squint from other causes is generally more a definite paralysis than a paresis and the history is longer.

Where we have a well marked crisis existing when we first see the child it is most important to examine the condition of the knee jerk and to look for squint or other paralysis, for only thus can we exclude capillary bronchitis or acute general tuberculosis.

This can never be given with absolute certainty; a mild case may suddenly develop grave symptoms and death ensue, whilst one giving us the greatest anxiety may go steadily on to recovery. Henoch speaking from a large experience says we should not give a good prognosis till four to six weeks after diphtheria. There are, however, some points which, other things being equal, make the outlook more hopeful and justify to a certain extent a better prognosis being given. These are:-

(1) The preceding attack of diphtheria having been of a mild type.

(2) The late onset of the paralysis.

(3) The limited extent of the paralysis.

(4) The treatment adopted.

To these may be added

(5) The late disappearance of the knee jerk.

(6) The absence of albuminuria.

As regards the first sufficient reference has already been made to it. The late onset of the symptoms is certainly favourable. This is well brought out in Mackenzie's cases. Paralysis coming on very early at the end of the first or in the second week is much more likely to end fatally than where it is delayed to the fifth or sixth week. This, however, can only be held to be generally true for there are numerous exceptions to both statements.

Though the heart may become affected in cases where the paralysis is more or less limited it is much less frequently so.

Cadet de Gassicourt's statistics bear this out. But as I have said the number of his simple limited cases is far greater in proportion than those of most British observers. The simple form of cardiac implication, the angina-form attack, occurs more frequently where there is little paralysis. The prognosis is, however, fairly good.

The fourth point, the fact of treatment having been adopted may seem rather superfluous. This, however, is not so. If the case be recognised early and treated efficiently the outlook is decidedly better. But where a child is allowed to go about and is not treated the risk of grave symptoms supervening is much increased.

Many cases of sudden death have undoubtedly been due to injudicious nursing, the child being allowed to play about as usual. Recovery in cases coming on in this way is very rare. The late disappearance of the knee jerk is as I have said when referring to it of good import. Albuminuria increases the gravity in so far as it points in all probability to a preceding severe attack of diphtheria. It is, however, not a common symptom as these cases generally succumb to the primary affection.

A bad prognosis will of course be given where the opposite of these conditions exists and in particular where any of the symptoms of approaching cardio-pulmonary involvement are observed.

Widespread paralysis is very likely to end fatally especially if the diaphragm or intercostal muscles are involved. The description quoted from Guthrie of the appearance of a child who will probably have bulbar crises is of great importance and in such a case none but the gravest fears can be entertained.

TREATMENT.

Under this heading I do not propose to enter into the different methods of treating these cases but shall give briefly the routine treatment which I follow. It is that carried out at Paddington Green Children's Hospital and the results have been exceedingly gratifying. Before its adoption the death rate in this affection was considerable but latterly this has been quite altered and now many consecutive cases will be treated without a bad symptom from start to finish.

The three great leading principles are:-

1. Absolute rest in the horizontal position.
2. The administration of strychnine.
3. The Regulation of the diet.

As soon as a child is admitted he is put to bed, the pillows being removed; and is strapped down by a special apparatus which may be described as follows:- a strong band is passed

across the bed at the level of the shoulders and tied underneath; small straps are then passed round the shoulders and under this cross strap and fastened, whilst a straight band joins these across the chest.

By this means it is possible to so fix the child that he cannot lie otherwise than on his back nor can he raise his shoulders from the bed. Sand bags and a sheet may be required for the legs if he should prove troublesome. In general, however, these patients are only too glad to be left alone and lie quiet and contented all day if not disturbed.

This position is maintained till all danger is considered past.

Food is given by the nurse. The patient must not help himself as this entails a certain amount of exertion.

The bladder and rectum are attended to as in typhoid fever.

In most cases regurgitation of fluids will cease as soon as the horizontal position is adopted, but if it continue, particularly after other means have failed, such as thickening the milk or giving mainly solid food, and if there be any difficulty in swallowing, any choking or reluctance to take nourishment, then nasal feeding should be commenced at once. This can easily be done by any competent nurse. A small rubber catheter and a glass funnel to fit are needed.

The operation does not in general cause retching, a fact explained by the anaesthesia of the palate and pharynx.

The catheter should be well lubricated, and it is of great importance that the child should not be frightened or the resistance and excitement may bring on some graver symptoms.

Rectal alimentation is not often requisite.

The next point is the administration of strychnine. Most of these cases show a marked tolerance to the drug much greater than in health. A child of 2 or 3 years will take 2 minims of the liquor strychniae of the B.P. every 4 hours for many days without showing any poisonous symptoms. In older children and in adults very large doses can be safely given. Suckling describes a case where the enormous dose of 36 minims of this preparation was given thrice daily without any outward effects.

The liquor is conveniently given in a little glycerine and water and children do not object to its bitter taste.

The amount prescribed must depend on the gravity of the case. In an ordinary case we can begin with a four-hourly administration and reduce it in a fortnight to thrice daily. It should be given for at least a month. Where there is any anaemia iron can of course be added.

Most of these cases have a rather rapid pulse, 90 to 100, even when at rest, and on slight exertion or even on wakening from sleep 100 - 120. In a week or two this will gradually fall to 80 - 85 and exertion will not produce much effect.

Where there is any irregularity or weakness of the pulse it is well to supplement the strychnine by alcohol. Brandy I have

found to answer very well. It can be given every four hours with the medicine or if nasal feeding be necessary with each meal Often it is requisite from the time of the child's admission.

Digitalis is useful where the heart is rapid and excitable.

Constipation is the rule and is best combated by liquid extract of cascara sagrada once or twice daily.

Of other drugs which may be required atropine is the chief. In some hospitals it is still regarded as the sheet anchor. I have only used it however where there is a great accumulation of mucus in the lungs and where the saliva tends to flow more freely than usual and to prove dangerous to the patient from its liability to enter the larynx. A 1/100 of a grain may be given as even young children bear it well.

Most of these methods however are for what may be termed normal cases. Under them I believe the majority of children will recover without any bad symptoms. When cardio-pulmonary complications ensue drugs are practically useless. Death occurs in many cases so rapidly that no time is allowed for active treatment. When there is time the application of electricity - the induction current - is the best mode of procedure. It can be applied over the heart, to the bases of the lungs, the phrenic nerves, the attachments of the diaphragm, etc. Duchenne records a case which reacted very well several times to this treatment but the improvement was always of short duration and death finally ensued. C. de Gassicourt sometimes treats his

patients by this means as a routine practice from their entrance into the hospital. Where electrical appliances are not at hand counter irritation can be applied over the heart, etc.

Where we have definite warnings of a coming crisis it is well to begin hypodermic injections of strychnine and also of atropine. They should be given every two hours till the danger seems past.

If the case be going on satisfactorily the next question is, when is the child to be allowed up? This must be done very gradually. At the end of 3 or 4 weeks, dating from the onset of the paralysis, if all symptoms have gone, one pillow may be allowed. In a few days another may be added and then he is allowed to sit up and finally get about at the end of 6 weeks. This may seem a long time in very mild cases, but it must be always borne in mind that grave symptoms may appear even as late as this as Henoch and others have recorded.

After treatment must be conducted on ordinary principles. Tonics, and change to some bracing air are the chief.

A P P E N D I X I.

------- ✖✖✖✖✖ ------

ILLUSTRATIVE CASES.

C A S E I. DEATH

James Vent: age 3 years 10 months. Diphtheritic paralysis.
(Shadwell) admitted Sept. 20. Died Oct. 2nd. 1894.

Family History. Unimportant.

History of illness. Admitted with diphtheria of two days duration. Membrane on both tonsils and fauces; voice absent; cough weakly laryngeal; much recession. Temperature normal. Tracheotomy four hours after admission.

Progress. Much improvement after operation, except for a great deal of difficulty in coughing up tenacious mucus.

Sept. 21st. and 22nd. Temperature varying from 101° F to 103° F.

Sept. 23rd. Temperature still elevated: albuminuria: sickness once.

Sept. 24th. Temperature still high: paresis of palate noticed first by coughing on drinking.

Sept. 25th. Temperature still up; knee jerks absent; albuminuria; sickness five times. Internal squint, accommodation impaired; slight regurgitation.

Sept. 26th. Temperature normal; sick twice; other signs as before.

Sept. 27th. Sick seven times. Temperature normal.

Sept. 28th. Sick twice; tracheotomy tube left out.

Sept. 29th. Sick three times; wound closed.

Sept. 30th. Strabismus less marked; swallowing better. Sickness worse; nine times. Diaphragm became suddenly paralysed, heart acting regularly but quickly. Child much worse.

Oct. 1st. Pulse became very rapid and irregular. Child became much worse at night.

Oct. 2nd. Died at 7 a.m.

Here the sickness was a very marked feature. The temperature was highest at night: tachycardia came on over thirty hours before death.

C A S E II. DEATH

A. W. aged five years: admitted into Shadwell Childrens' Hospital under Dr Eustace Smith, May 24th. 1895.

History. Five weeks ago had fit one Sunday and one week later was found to have a sore throat which lasted four or five days. Talked through his nose since sore throat. Ten days later limbs became weak and he had regurgitation of fluids through nose. Has had diarrhœa for some days (before admission) seven to nine times a day. No loss of control over sphincters.

State on Admission. Well nourished but anæmic; marked nasal voice; aphonia; regurgitation and choking whenever he attempts to drink; nasal feeding therefore begun at once; right external rectus paralysed; pupils dilated; accommodation defective on both sides; soft palate quite motionless; right angle of mouth droops decidedly and saliva is constantly running out of mouth, or down the larynx causing choking and coughing. Diaphragm not acting well: intercostal muscles weak; paresis of neck muscles, head rolling about on shoulders; hands very feeble; legs weak; child very weak altogether; K.J. absent; cutaneous reflexes active; heart and lungs nil; pulse 100 soft and irregular. Put to bed and given strychnine etc.

May 25th. Child in much the same condition but the muscles of the whole right side of face are now affected; ptosis, right eye; marked aphonia; no albuminuria; saliva very abundant and

no attempt is made to swallow it now for fear of choking; bowels moved involuntarily since admission.

May 26th. Still incontinence of fæces; bowels moved three times; bladder not affected; no sickness; child very irritable.

May 27th. Still loss of control over rectum. At night the note made was that the child was evidently worse; gets rathe blue at times and is very restless; breathing is sighing at times; diaphragm not acting so well; other paralysis seem rather less marked.

May 28th. Remained as at last visit. Pulse becoming more rapid and irregular.

May 29th. Pulse now 140 to 150, irregular; sick three times; much sighing; looks very blue and ill; incontinence of urine.

7 p m. Crisis; became suddenly much worse, very restless and excited; breathing rapid and laboured; pulse feeble and irregular; child looks very blue and cold but temperature is 102°F. chest full of râles; much rattling of mucus in throat; diaphragm paralysed; total inability to cough; given strychnine hypodermically

8 p.m. Temperature 103°F. child a little better but still very blue; pulse 140; incontinence both urine and fæces (has been so all day) lungs much choked up with mucus; given one

minim of liquor atropinæ hypodermically with strychnine.

9 p.m. Better after atropinæ; not quite so blue; pulse stronger, but still rapid, 140 per minute; lungs clearer and breathing easier; temperature 103·2 F.; still very restless and another crisis seems imminent. Diaphragm completely paralysed; sulcus formed at epigastric and hypochondriac regions with each inspiration; sucking in of intercostal spaces; right facial paralysis very marked.

10 p.m. Breathing again becoming embarrassed from mucus, a second injection of atropine was given. Breathing decidedly better after it; diaphragm acting slightly. Temperature falling.

May 30th. Patient slept for two hours; pulse very rapid and irregular; breathing quite clear but jerky; lips very blue.

2 p.m. Patient very blue; diaphragm absolutely paralysed pulse 145, irregular; was sick after nasal feeding

6 p.m. Very restless and blue; sick after nasal feeding

7 p.m. Crisis: pulse uncountable, can hardly be detected at wrist; breathing very rapid and shallow; lungs becoming choked up; given strychnine hypodermically but did not rally; died at 8 p.m.

NOTES:- This was a very widespread case where paralytic symptoms came on early but the child was allowed to go about till brought to Hospital some weeks later. A bad prognosis was given

from beginning for this reason. The long interval between the start of the paralysis and the onset of a crisis is unusual. The history of preceding diphtheria is however very meagre. The paralysis of the rectum with diarrhœa is unusual. Paralysis of the bladder came on towards the close only before the crisis; well marked premonitory signs, tachycardia and sickness.

C A S E III. DEATH.

G. W. aged four years. Admitted to Shadwell Childrens' Hospital under the care of Dr. Eustace Smith, on 21st May 1895.

History. Sore throat six weeks ago; treated at home; in bed two weeks. Two days ago had nasal voice and regurgitation of fluids for first time and yesterday squint was noticed.

On admission. Well nourished child, admitted looking very ill; face cyanosed; pulse rapid and irregular; breathing rapid and jerky; lungs shew some râles; given strychnine and put to bed at once.

Examination made later shewed:- Muscles everywhere weak; general limpness; neck muscles very weak; hands very weak; legs not tested; muscles of lower part of right side of face involved, angle of mouth drops; much saliva running from it, or, if he attempt to swallow, entering his larynx and causing choking; soft palate motionless; regurgitation and choking so as to render swallowing impossible; (nasal feeding from admission complete aphonia; cough quite typical; some rattling of mucus in throat: external rectus paralysed both sides; accommodation defective; slight nystagmus on fixation for near vision. Heart rapid and irregular; lungs full of râles; diaphragm acting feebly; slight sucking in of intercostal spaces; K.J. absent

superficial reflexes active; incontinence of urine and fæces.

May 21st. evening. Child much the same; respiration however has suddenly changed from rapid to very slow only three respirations per minute, of a Cheyne Stokes type. Given brandy and strychnine hypodermically and improved.

May 22nd. Breathing rapid now; other signs rather improved

May 23rd. Can't take even thickened food; still nasal feeding; nystagmus more marked. Sphincters of both bladder and rectum still paralysed. Breathing and pulse better, stronger and less frequent; child very irritable.

May 27th. No change for better or worse; diaphragm remaining the same.

May 29th. Still quite aphonic; much saliva running from mouth; is becoming more restless and the general apathy of past few days is passing off; cheeks very flushed; blueness of lips.

May 30th. Had good night, but is very restless this morning; pulse has run up to 130 per minute without any evident cause; lips blue.

11 p.m. Sick after nasal feed; mucus collecting in trachea inability to cough it up.

May 31st. Been very restless all night. Sick twice; pulse 146, irregular; diaphragm hardly acting.

11 a.m. Crisis: choked suddenly; pulse hardly felt at wrist; given strychine minim 2 hypodermically.

11.30. Improved a little; pulse stronger, very rapid 160; coughing quite impossible; face and lips very blue; given minim $\frac{1}{7}$ of liquor atropinæ

2 p.m. Sick after nasal feeding; crisis; quite pulseless; breathing hardly perceptible.

2 30. Did not improve after strychnine, brandy etc. and died at 2.45.p.m..

Remarks. Another widespread case; child probably had paralytic symptoms some time before admission but they were not noticed. There was probably a crisis just before admission but mother would not give any information; the aspect of the child was like it; The premonitory tachycardia and sickness were noticed early. The early bladder and rectum paralysis is unusual. The history however is extremely unsatisfactory.

(As regards the slow breathing which occurred on the evening of admission I was once shewn a similar case but unfortunately could not get any notes of it, nor learn how it ended.)

C A S E IV. RECOVERY.

Alice G. aged seven years. Diphtheritic Paralysis.

Diphtheria on 21st. December 1894; mild attack; was quite well in ten days.

Seven weeks later mother noticed that she spoke through her nose and shortly afterwards she began to squint. Was not brought up to Hospital till one week later. There were then nasal voice; occasional regurgitation of fluids; aphonia; left vocal cord in cadaveric position, right cord healthy; palate paralysed on the right side only, the uvula pointing towards the left; left external rectus muscle paralysis; K.J. much diminished; heart acting irregularly; pulse small and rapid; diaphragm etc., normal. Parents would not allow child to be taken into hospital but case was seen at home; ordered complete rest in bed with strychnine etc. Four days later note made was:- K.J. not elicited; heart still rapid but not so irregular; other signs as before.

A fortnight later:- Squint now gone; palate moving better no regurgitation for past week; aphonia better, but left cord not moving quite normally. K.J. absent.

Was quite well in another fortnight; K.J. absent.

C A S E V. RECOVERY

Daisy Cottle: aged three years and three months. Diphtheritic Paralysis. (Shadwell) admitted February 25.

History. Had diphtheria eight weeks before admission. Weakness of legs was first symptom noticed two weeks before admission; then nasal voice, squinting and general weakness; complained of pains in legs three days ago.

State on admission. Well nourished child; no wasting anywhere; moves arms and legs very weakly; no anæsthesia; paresis of palate, movement very slight; slight nasal voice; internal squint right side; no paralysis of accommodation; diaphragm acting well; lungs normal; heart acting regularly; Knee jerks absent.

Child lies very quietly; coughs on trying to drink.

Continued thus till March 4th. when the squint became much more marked, respiration shallow, and diaphragm acting badly. Pulse however remained good and not increased in rate; no sickness.

March 7th. Diaphragm has been getting better; now acting very well; no coughing on swallowing; pulse very good. Knee jerk still absent.

March 16th. Squint gone; palate acting normally; is taking her food well.

March 17th. Apparently quite well. K.J. still absent;

sat up a little to-day.

Improvement continued and a fortnight later child went home well.

C A S E VI. RECOVERED.

Alice Blackman (2½ years) Diph: Paralysis. Admitted Oct. 18th discharged Nov. 21st. 1894.

Family History. Unimportant.

Personal History. Nothing noted antecedent to present illness. Child was brought up to Casualty Department, Paddington Green Children's Hospital on account of having had fall on her face. Diph: Paralysis diagnosed.

Mother's history of present illness. It is not known that she has had sore throat: but the child fell on her face two weeks ago, her nose bled badly and since then for about a week she has spoken through her nose. She is very weak on her legs and when she cries her lips go blue. Appetite very good but fluids returned through nose at the same time as child began to have nasal voice. Bowels regular. A child in near house had diphtheria some weeks ago but no one in patient's house is known to have had a sore throat.

Present condition. Well nourished but anaemic child - voice nasal - general aspect languid and drowsy - lies on her back with all her muscles relaxed (Spread out), and seems very disinclined for exertion of any kind; is irritable if aroused. She has no actual regurgitation of fluids, but drinks very slowly taking very small sips. Lungs normal. Heart normal but very rapid (120) No paralysis of diaphragm. K.J. gone. Paralysis

of palate - squinting both eyes. (Ext. rectas paresis). Ordered. Liq.strych. in mixture m ℥ 4 tis horis.

Oct. 19. Child apathetic irritable on being aroused. Pulse small and rapid - no regurgitation but drinks very slowly and unwillingly. Temp: 100° F.

Oct. 26. Not so pale - takes food better - Respiration during sleep this morning was irregular. Two respirations then long pause, then other two, and so on. On waking breathing became regular. (Pulse 120 m. 102 evening. R. 24 m. 22.e.) Takes food better.

Oct. 27. Much brighter and better - voice not so nasal - drinks better - only sighing on dropping off to sleep or on waking.

Nov. 5. Temperature still rather irregular. (was 99-100 from admission till Nov. 11th, and after that normal. Highest point 100·2°F.) Heart acting well. Still kept lying flat in bed. K.J. absent.

Nov.10. Allowed one pillow - straps taken off - voice better. P. 100. R. 22. No sighing - squint gone. K.J. absent

Nov. 21. Went home quite well but with K.J. still absent

C A S E VII. DEATH

E. S. æt. 4 (F). Under care of Dr. Ogilvie. May 30th. 1890

History. Five days ago said "neck hurt her", and moved with head on one side. There was no history of sore throat. Three days ago "had difficulty in speaking". Two weeks ago had fallen and bruised left temple.

On admission.- Fairly nourished. Very pale, but shortly after became flushed. Languid and drowsy, rolled head about, as if too heavy for her. Swayed unsteadily from side to side, and would have fallen if not supported. Knee jerks absent.

Pupils moderately dilated. Re-action to light good - to accommodation doubtful. Squint O

Tonsils slightly swollen. Soft palate, very sluggish, but not motionless. Voice weak and nasal. Mucous râles in throat. Pointed to larynx when asked if in pain.

Respirations 36.

Inspiration short, laboured. Expiration sudden, short and puffing. Upper intercostals seemed to be alone acting. Diaphragm paralysed.

On auscultation coarse mucous râles heard all over. Entry generally weak.

Pulse 126, feeble and irregular. Temp. 100. ordered liq. Strychniæ, M.ii. ivtis h. Brandy 3i iidis h.

Urine.- Slight trace of albumen.

May 31st. Swallows fairly well, but slight regurgitation of fluids. Voice more nasal, hoarse and weak.

June 1st. 4.5 a.m.- Attack of extreme dyspnœa, with cyanosis, restlessness and vomiting. Ether sub cutem and brandy freely given. Died at 9 a.m.

P.M.- Lower half of left lobe of lung collapsed, dark purple, shiny, smooth, and airless. Much mucus in bronchioles. Right auricle and ventricle distended by yellowish white adherent clot.

The medulla looked healthy, and, on microscopical examination, the cells showed similar appearances to those seen in the preceding case.

The vessels were engorged.

Sections of the phrenic nerves showed no abnormality.

C A S E VIII. (Recovery)

Hetty Coggins (10) Diph. Paralysis. (P.G.Children's Hospital)
Admitted Nov. 21st. 1894. Taken home Dec. 19th. 1894.

Personal History. Has had measles and whooping cough; always "nervous". Six weeks ago had a sore throat - was kept in bed a fortnight; was then taken to a doctor who said she must have had a very bad throat and asked about Diphtheria. But no one in the house had had a bad throat. A fortnight ago child complained that she could not see properly; had pains in her legs; for past ten days fluids have returned through her nose - no regurgitation of solids. She speaks through her nose. Bowels constipated - appetite good. On questioning the first thing noticed was the nasal voice, then pains in legs, then impairment of vision.

Present condition. Well nourished, well coloured girl, nasal voice.

Chest. Normal both lungs and heart.

Abd. nil. (heart acting quite regularly)

Eyes. Accommodation - can't thread needle or read small print. Pupils do not react much if at all to accommodation, but do so readily to light - no squint.

Palate. Moves but very slightly and there is same anaesthesia of it. There is no regurgitation of fluids or solids now,

but patient drinks very slowly. Voice is very nasal. When spoken to she is very jerky not only in hands and face but also in trunk.

K.J. present. (active) on both sides.

Nov. 24. This morning there is a profuse papular rash over both knees slightly also on back, between scapulae, on shoulders, slightly on back of upper arms and on ankles. There are no wheals but general appearance (especially on knees) of urticaria. They are rose-red papules with solid whitish tops. The rash is not itchy.

Nov. 25. Rash almost gone - only slightly on knees and shoulder. K.J. active - nasal voice still present.

Nov. 28. Rash gone - K.J. active - accommodation much improved, can thread a needle fairly well. Still nasal voice.

Dec. 3. K.J. less active but still present. Accommodation improving, still nasal voice.

Dec. 12. Voice practically normal: eye normal. K.J. active; allowed one pillow.

Dec. 19. K.J. active. Child practically well. Taken home although she had not been up. (against advice).

(Temperature normal throughout.)

(Treated by Liq. Strych. m $\frac{ii}{ij}$ ter in die) (Cascara for Constipation).

Note:- This child was first seen by an out patient physician two days before admission: and he noted the interesting

fact that though the pupils reacted perfectly to light and accommodation, yet she could not see to read or thread a needle. The reason of this inability is not very clear.

C A S E. IX. (Recovery)

Samuel Smith aged 3 years and 10 months. Admitted Nov. 28th. 1894. Discharged Jan. 4th. 1895.

Family and Personal history. Unimportant.

Present illness. Five weeks ago, his mother states, he had "lumps in his neck with a breaking out on his mouth and discharge from his nose". She did not notice that he had a sore throat and he was not kept in bed. Four weeks ago she observed that he spoke through his nose. Regurgitation of fluids occurred during the week previous to admission. She has not noticed any squint or falling about.

State on admission. Well nourished but rather anaemic child. Voice markedly nasal. Soft palate hangs quite motionless but sensation is not abolished only dulled. When child is at rest the pupils are dilated. There is no squint and reaction to light and accommodation is quite natural. No other paralyses. Knee jerk absent. Heart seems normal. Lungs normal. There is very often deep sighing.

Nov. 30. Has been sick this morning. Marked nasal voice still.

Dec. 4. Child has been in much the same condition since last note. Yesterday he broke the straps confining him and sat up. He was almost immediately seen and strapped down again. Following this he had last night regurgitation of fluids once or

twice but only slightly. To-day there is slight aphonia more marked on crying. Accommodation of the eye is very defective the pupils contracting only very slightly. No squint. Heart rapid but regular. Still occasional deep sighing - K.J. absent.

Dec. 10. Child looks better - Accommodation normal to-day - Still marked nasal voice. Aphonia gone.

Dec. 16. Improving. Heart acting more slowly - 84 per minute. Voice not so nasal.

Dec. 19. Voice improving - Boy cheerful.

Jan. 4. Discharged well. K.J. still absent.

Note:- This Boy unlike most of the other cases, was bright and cheerful from admission.

The paralysis of accommodation, the aphonia, and rapid heart following the sitting up are very noteworthy and show the necessity of absolute rest.

C A S E X. (Recovery)

Margaret Mary Dean aged 2 years 9 months. Admitted Feb. 13th. 1895 discharged March 19th. 1895.

Family and personal history. Unimportant.

History of present illness. Had sore throat just before Christmas 1894 for which she was in bed for a few days. Got quite well and remained so till fortnight ago. (i.e. beginning of February) when she had thickness of voice and squint. Was thought to have a sore throat and was put to bed for two days. When she got up was very tottery in her legs. Incontinence of urine was then noticed but did not become marked till a week later. Then also urine began to smell offensively. For past week has complained of abdominal pain at night causing her to scream. It lasts but a short time. No sickness or regurgitation. Has been very fretful for 6 weeks.

State on admission. Well nourished child; is very apathetic; very fretful if disturbed. Arms and hands are tremulous on movement and power is much diminished. Legs move naturally enough but are weak. Thorax expansion rather limited but there is no sucking in of spaces. Abdominal movement very slight in inspiration but there is no definite involvement of diaphragm.

Eyes. Paresis of Right external Rectus and slightly of left. Right pupil larger than left and does not contract fully to light. Accommodation slightly affected pupils contracting to half size. With child lying quietly and not facing bright light there is

dilatation of both pupils.

Palate. Paralysis of soft palate; anaesthesia doubtful, marked nasal voice; no regurgitation but patient drinks slowly as if afraid.

When she cries there is inspiratory ~~shudder~~ Stridor but speech is not affected.

Knee jerk absent.

Lungs. Same Rhonchus over both lungs. Respirations 24 with occasional deep sighs.

Heart. Normal to percussion and auscultation Pulse 120, regular in rhythm but rather small and compressible. (In sleep P = 96.) Marked incontinence of urine night and day.

Progress. 15th. February. Voice more nasal; breathing shallow and sound rather trachfal; no fresh symptoms.

16th. Pupils not so unequal; other signs as before. She complains of being very tired and wishes to be left alone. Ever since admission she has been very afraid of being moved, crying out that she is falling and this merely when rolled over from her back to her side, or when the bed was moved. Generally when she wakens up she screams loudly and does not cease till made to understand that she is lying in bed. There seems to be a distinct loss of muscular sense; she can't locate her position after the slightest movement.

17th. No thread worms or any local cause for the incontinence which still continues.

19th. Pupils now equal, squint much less; nasal tone of voice less marked; is not so afraid of movement but still screams on awakening. Co-ordination of movement of hands seems normal; sensation normal.

20th. Squint and nasal voice same; had rise of Temperature to 100·5°F. no known cause, Incontinence less frequent.

22nd. Not so languid - and seems generally better - Still rather frightened on awakening but does not scream.

24th. Incontinence now only at night. Child bright.

25th. Early this morning had fit of screaming: complained of abdominal pain; no sickness; enema given.

Evening:- Child looks very pale and washed out; complained of feeling sick but was not actually so. P. 90 regular. Complained of pain on palpating abdomen. Tongue furred. Still constipation. Voice and eyes nearly well.

March 2. Palate and eyes well. Incontinence only occasionally at night. Allowed one pillow.

March 6. Has had Temp. 104° F. falling in 24 hours to normal. (? Influenza which is going round the Wards)

March 7. Child very bright: no longer frightened on awakening. K. J. still absent; still occasional incontinence: Allowed to sit up.

March 23. Went home well. Incontinence quite gone. K. J. still absent.

Notes:- Constipation all through for which given Cascara Sagrada. Treated by Liq. Strychniae ṁ. 2 every 4 hours from admission till Feb. 20; then thrice daily till March 6th. after that Syrup Hypophosph. Co. ṁ. 30 thrice daily.

Pulse during 1st. 10 days	=	106	-	112	at night.		94	-	100	in day.
Next week.	=	90	-	100	"	"	80	-	90	" "
" "	=	80	-	90	"	"	76	-	80	" "

Respiration for first fortnight remained at this rate = 24 - 30 at night, 18 - 22 in day. Then became 20 - 22 regularly night and day.

C A S E. XI. (Recovery)

Gertrude Berridge. (4 years and 11 months) Diph. Paralysis.

Admitted March 19th. 1895, to Paddington Green Children's Hospital.

Family History. Unimportant. Other children well.

Personal History. Had Diphtheria at end of January 1895; was in bed for a fortnight and was noticed to be weak on legs when she got up. Has been falling about ever since. For the past fortnight has lost her voice. Drinks slowly but there has been no regurgitation. Appetite not so good past few days. Squint noticed 4 days ago; no incontinence.

Condition on admission, Fairly well nourished but anaemic. (Has been seen by me at O.P.) (walked into room) Gait very tottery. Legs are flabby and power is much less than normal. Hands and arms are likewise affected. Muscles of neck seem normal. Diaphragm is not acting well, the abdominal walls expand a very little on inspiration. There is no thoracic suction - Abdominal muscles flabby.

Chest. Breathing fairly good but with occasional deep sighing. Heart and lungs normal to percussion. Systolic bruit over pulmonary area. Heart is very easily excited, when asleep P = 110. on awakening 120.

Throat and Pharynx. Soft palate motionless and anaesthetic uvula trailing on tongue. There is evidently some cord paralysis

as patient cannot phonate properly. Sounds made are in a hoarse whisper and very nasal in tone.

Eyes. Loss of parts of accommodation both eyes - pupils dilated. Right external Rectus paralysed - K.J. gone.

March 23. Going on all right; paralyses the same; pupils still dilated but if shaded seem to contract a little to accommodation. Pulse stronger and less frequent now 94. Resp.20. On waking from sleep pulse rose from 96 - 120 but in 1½ minutes fell to 100 quite regular in force and Rhythm. K.J. absent. Tongue furred; constipation.

March 24. Child better - Squint less. Voice louder and more distinct - pupils the same. No sighing - P. = 82 regular. Still drinks very slowly - no regurgitation or choking; no trouble over solids. To-day palate touched and found no longer anaesthetic but reflex as usual. K.J. absent.

March 25. Pupils now contract nearly to half on accommodation. Still some squint.

March 30. Ocular symptoms now gone: palate nearly normal. K.J. still absent.

April 5. Allowed one pillow. Child went home quite well but with K.J. still absent.

Note:- The gradual diminution in pulse rate is well shown in this case.

C A S E XII. DEATH.

G.D., aet. 6½. - Admitted under care of Dr. HERRINGHAM, September 2nd, 1889.

History. Five weeks ago, "ulcerated sore throat", lasting about a fortnight.

Two weeks ago, after a long walk, complained of "legs feeling tired, and began to totter about." Throat quite well then.

Six days ago, food, both fluid and solid, began to come back through his nose.

Weakness of hands and arms noticed at same time. Has been unable to write for a week. Has held head down as though his neck were weak.

On admission. - 12.30 p.m. Anaemic, but well nourished. Pupils equal, dilated. Squint, ○ Soft palate motionless. Uvula deflected to right. Voice indistinct and nasal. Fluids regurgitate through nose even when spoon fed.

Could not walk or stand without assistance. Knee jerks absent. Grasp of hands weak, especially left. Heart's action slightly irregular. Pulse 144, irregular. Respiration 32, irregular and sighing. Diaphragm working but apparently rather feebly.

No abnormal signs on auscultation. Temp. 99. Ordered Syr.

Eastonii m.xx. vi^{tis} h. Port wine, $^{ss.}$ t.d.s. To be fed by nasal tube and by nutrient enemata.

At 7.20 p.m. Crisis. - Much restlessness and cyanosis. Neck extended, extremities cold.

Respirations 32. Irregular laboured and gasping. Much mucous accumulation in throat and lungs. Pulse 144, weak and irregular. Complained of headache.

Ether m.xx injected sub cutem, after which he vomited much greenish brown frothy mucus, and seemed to revive.

At 8 p.m. pulse fairly good, though still fast, respiration easier and child quieter.

8.15. (Crisis). Another attack of restlessness and dyspnoea. Respirations very laboured and irregular. Fauces choked with mucus. Loud râles all over lungs. Jaw muscles working convulsively at each inspiration. Pulse weak, irregular, uncountable. Temp. 99·2. Died at 8.40 p.m.

P.M. - Lungs extremely congested, and bronchi full of watery fluid. No other abnormality. Heart empty in diastole. Left ventricle somewhat dilated. Brain dark and congested.

A P P E N D I X II.

EXPLANATION OF SIGNS USED.

K.J. Knee jerk

\+ = affected.

\- = not affected.

0 = not recorded.

A = absent.

C = Cured.

D = death.

Name etc.	Age	Result	Date of Diphth.	K.J.	Palate	Legs	Eyes	1st.symptom	History of ailment previous to admission.	State on admission.	Progress	Notes.
No.1. A.F. P.G.C.H. 6/6 /94	6½	C	5 wks. ago	A	+	0	+	Regurgitation	Diph. 5 wks ago. In bed fortnight. Regurgitation ever since she was ill, before she got out of bed. Speaks thro' nose and can't always be understood. A week ago mother noticed that she squinted and could not see properly.	Anæmic - K.J.absent - can't see very well. Pupils dilate on attempted accommodation - no squint - voice weak and nasal. Diaphragm normal. Heart &c. normal.	Progress uneventful. Dismissed well.	
No.2. F. W. P.G.C.H. 13/6/94	4½	C	Sore throat one month ago	A	+	+	-	Nasal voice	No definite history of Diph. Sore throat 1 month ago. A fortnight ago began to speak thro' nose and to fall about seeming to have no power in legs. Never had regurgitation. Sometimes pains in abdomen lately.	Anæmic; swallows fluids badly. No regurgitation; walks feebly dragging the legs especially the right. Heart &c. normal. K.J. absent.	"	Legs much affected; one more than the other.
No.3. E. F. S.G.C.H. 24/1/94	5	C	3 wks before Xmas	A	+	+	0	Nasal voice	Diph. 3 wks before Xmas - in bed only 1 week. Nasal voice and regurgitation about 1st. Jan. For past week has stumbled when she tried to run, and has complained of feeling tired.	Well nourished, good colour; nasal voice but no regurgitation legs very weak especially the left. K.J.absent.	"	Legs much weakened; one more than the other.
No.4. R.M. P.G.C.H. 8/7/93	4½	C	5 wks. ago	Exag.	+	+?	+	Nasal voice and regurgitation	Had diph. very severely. eight days ago. Nasal voice and regurgitation noticed.	Anæmic: nasal voice: Soft palate moves slightly; regurgitation of fluids; K.J.exaggerated; tendency to rigidity of lower limbs; child apathetic. Heart irreg. in force and rhythm.	15.July. regurgitation stopped; pupils dilated;accomo. sluggish. K.J.diminished 16 July. Fæces passed involuntarily at times - diarrhœa 18 Aug.Has improved every day and now well. K.J. normal.	K.J. never absent in hospital.
No.5. A.F. P.G.C.H. 29/ 3/93	7	C	One month ago	A	+	-	+	Nasal voice	Was ill for a fortnight with Diph. Five days ago noticed he spoke thro' his nose and he complained that he could not read; things becoming dark. Regurgitation 2 days ago.	Anæmic; nasal voice; no regurgitation; swallows with difficulty; soft palate moves very slightly, but is not anæsthetic pupils widely dilated, accommodation defective. Diaphragm acting badly.Heart irreg. and feeble. Legs not affected.K.J.absent.	Ap.5. Voice better; swallows with difficulty; diaphragm acting badly. Ap.10.Still nasal voice; swallowing better K.J. present. Ap.23 Sitting up Got well	Note early return of K.J.
No.6. C.S. P.G.C.H. 17/1/93	6	C	2 or 3 months ago	A	+	+	+	? Legs	Date of diph. doubtful. One month ago weakness and dragging of legs & soon after squint; then hands became weak; 14 days ago nasal voice and choking over food; no regurgitation.	Anæmic; apathetic; speaks only when pressed to do so; nasal voice; palate moves very little; diaphragm acting badly; rectus abdo.paralysed; hands weak;K.J. absent.Heart.very irreg.Incoorn of movement espec. of hands. very staggering gait.	Jan.8.very ill; sickness, regurg; fed by nasal tube. Jan.19. Better; sick occas; diaphragm acting better; swallowing difficult. Jan 21. Still nasal voice; swallows better " 26 voice not so nasal. K.J.absent. Feb.14 better. Got up a little. Mar.17. K.J.feebly elicited. " 28 Went home well	Note return of K.J.

Name etc.	Age	Result	Date of Diphth.	K.J.	Palate	Legs	Eyes	1st.symptom. noticed	History of ailment previous to admission.	State on admission.	Progress.	Notes.
No.7. M.S. Dr.Donkin	3¾	C	Four months ago	A	+	+	+		History defective. Diph.4 months ago, since then could not speak plainly. Squints. Regurgitation. weakness of eyes. (See case of brother below.	Pupils large. no loss of accom. double converg.squint. Diaphragm weak. Cough ineffectual. Palate acting naturally. K.J.absent.	Uneventful. Went home well.	Very long duration of symptoms
No.8. R.S. Dr.Donkin ad.Sep.28	17 mos.	D	1st.wk Aug.	A	+	-	-	Regurgitation	History of diph. 6 weeks ago. Regurgitation came on 3 weeks later.	Head rolls in all directions. Palate very sluggish. (Heart &c. normal) Regurgitation of fluids Can take thickened food.	Oct.2. Child made a choking noise whilst lying quiet in cot. Nurse ran to him and found not breathing. Heart acting(rapidly) Liq.strych.½m. Brandy injected. Heart soon stopped.	Sudden death.
No.9. C.M. Dr.Donkin. ad.Dec.17	8 yrs.	C	1st.wk. Sept.	A	+	+	+	Nasal voice & regurgitation	When he got about week after Diph. nasal voice,regurgitation, weakness of legs, sight affected. Tingling in fingers and upper arms, later on in legs.	Diaphragm acting feebly - Resp. mainly costal. Eyes normal all other symptoms gone. K.J.	Was admitted for condition of diaphragm which soon improved and child went home well.	
No.10 A.H. Dr.Donkin ad.Nov.15	6 yrs.	C	1st.wk. Oct.	A	+	+	+	? eyes.	10 days before admission. Speech affected, nasal voice, regurgitation; weakness of legs since sore throat, decided for 1 week arms affected for past week; slow breathing at night with sighing for 3 or 4 days. Difficulty in seeing for past fortnight.	Occasional regurgitation; nasal voice; palate acting poorly; diaphragm acting feebly; Resps. mainly costal, and irregular. Sensation normal. Eyes normal.	Nov.28. More regurgitation 29. Diaphragm acting better. Dec. 5 Breathing 12 per minute 8. Speech normal. 10 K.J. still absent Went home well.	Had been taken to Moorfields and given spectacles for weakness of eyes.
No.11. A. R. Dr.Smith. ad.July.23	8 yrs.	C	3rd.wk. June	A	+	+	+	Nasal voice	Nasal voice soon after Diph. Regurgitation 2 days before admission.	Nasal voice. Regurgitation - weakness of legs and arms. Walk very unsteady - ataxic - can't stand steadily or touch tip of nose accurately with eyes shut. Plantar reflex absent.Other sup. reflexes normal.Both ext.Recti paralysed. Accom.very sluggish.	July 30. Squint well marked. Legs very weak K.J. and plantar reflex absent. Aug.3. Pupils large and active. 22. Walking improved. Voice much less nasal. Went home well.	Absence of plantar reflex and inco-ordination of movement.
No.12. A.L. Dr.Donkin ad.Ap.21.	2¼	1.S.Q.	Mid. March	A	+			Regurgitation	Became hoarse and had regurgitation 2 wks. ago. Has not spoken clearly since diphtheria.	Considerable aphonia. When examined child flies into rage. Heart becomes irreg. and child looks bad. Soon gets all right again.	Removed by friends in 2 or 3 days.	

Name etc.	Age	Result	Date of Diphth.	K.J.	Palate	Legs	Eyes	1st.symptom.	History of ailment previous to admission.	State on admission.	Progress	Notes.
No.13 E.P. Dr.Donkin. ad.Mar.19.	2 yrs.	C	No sore throat noticed. Another child sore throat 3 wks.before	A		+	+		Quite well until 15th.March when she could not hold up her head in morning. Has had no power in hands since and great difficulty in walking. No regurgitation.	Paralysis of super. Rectus in right side. K.J.absent.	Notes lost. Got well.	
No.14. W. A. Dr.Coutts. ad.Dec.3.	2½	D	End of October.	A	+	+		Regurgitation	Regurgitation on 2nd.week of illness till 3 days ago when it stopped. Patient could not walk when he got out of bed. Weak in back last 3 days. Diph. in same house with 1 death.	Marked lassitude: Heart and lungs normal. No squint. Paresis of muscles of neck and back. Cannot sit up for any length of time. Head hangs forward. Resp. mainly costal: diaphragm acting imperfectly. No Regurgitation.	Dec.5. Worse,lies quietly on back.Diaphragm becoming slowly paralysed,very slight movement now.Resp.almost entirely costal.Very irritable when touched.Heart & Lungs normal. Nasal fed, swallowing badly. Dec.6. Resp.worse. Sweating much. Dec.7. 8.15.suddenly much worse.Cyanosed. Breathing irreg.Injec.digitalin. 845.lips black.face deadly Resp.irreg.(clonic spas.lower jaw)Pulse imperçep. Death.	
No.15. J. D. P.G.C.H.	4½	C	6 wks. before	+	+	-	+	nasal voice	6 weeks after Diph. began to speak through nose and have regurgitation of liquids and solids; was very languid and tired; lips got very blue at times.	Well developed, anæmic, very apathetic. Soft palate paralysed accommodation defective. Pupils dilated. K.J. absent. Occasional deep sighing. Heart & lungs seem normal.	For first week had Temperature 99°-100°F. then normal. Progress uneventful.	
No.16. A.B. P.G.C.H.	2½	C	?	+	+	+	+	Falling about ?	No History of sore throat. but child in next house had diphtheria some weeks ago. Full notes in Appendix I. Case VI.	See Appendix I.	See Appendix I.	
No.17. H.C.	10	C	6 wks. before	-	+	?	+	Nasal voice	See Appendix I. Case VIII	See Appendix I.	See Appendix I.	
No.18. S.S	3 10/12	C	5 wks. ?	+	+	+	+	Nasal voice	See Appendix I. Case IX.	See Appendix I.	See Appendix I.	

Name etc.	Age	Result	Date of Diphth.	K.J.	Palate	Legs	Eyes	1st.symptom	History of illness previous to admission	State on admission.	Progress	Notes.
No.19. M.D.	2 7/12	C	5 wks. ?	+	+	+	+	Nasal voice and squint.	See Appendix I. Case X	See Appendix I.	See Appendix I.	
No.20. G.B.	4 11/12	C	2 wks.	+	+	+	+	Falling about	See Appendix I. Case XI.	See Appendix I.	See Appendix I.	
No.21. J.V.	3 10/12	D	6 days	+	+	0	+	Palate.	See Appendix I. Case I	See Appendix I.	See Appendix I.	
No.22 D.C.	3¼	C	6 wks.	+	+	+	+	weakness of legs.	See Appendix I. Case V.	See Appendix I.	See Appendix I.	
No.23 G.M. P.G.C.H. 11/1/93	2½	C	Diph. Nov.4. 1892	A	+	-	+	Nasal voice.	Taken to Fever Hospital - came home Dec.13 then had nasal voice - and difficulty in swallowing but no regurgitation. Regurgitation on 9th. January.	Anæmic - pupils dilated. Soft palate normal. Nasal voice - arms and legs normal. No regurgitation.	Jan.21. Much better - does not take liquids well Progress uneventful. Went home well.	
No.24. L.J. P.G.C.H. 22/8/93 ?	2¾	C	sore throat 6 wks. ago	A	+	+	+	legs and nasal voice. ?simultaneously	A fortnight ago"went off her legs"suddenly and talked through her nose. Has had regurgitation and squinting.	Pupils dilated.React feebly to light & Acco. Int.squint both eyes - nasal voice - chokes over liquids.No regurgitation.Diaph. acts feebly.(Can't walk alone) can move legs quite well however (Back much bent) no anæsthesia K.J. absent.	Sept.30.Squint gone - pupils normal - swallows slowly. Diaphragm acting better. K.J. absent. went on well till end of October when she had Follicular tonsillitis. Nov.15. Went home well. K.J. absent.	

Name etc.	Age	Result	Date of Diphth.	K.J.	Palate	Legs	Eyes	1st.symptom.	History of ailment previous to admission	State on admission.	Progress.	Notes.
No.25. W. F. P.G.C.H. 19/9/93	6 4/12 yrs.	C	Sore throat 5 wks. ago.	A	+	+	+	Nasal voice spoke thick	5 wks. ago sore throat. 1 month ago "spoke thick" 1 or 2 weeks ago regurgitation staggers in walking. 3 days ago squint was noticed and child said things looked double	Anæmic.Pupils sometimes dilated normal in reactions.No squint nasal voice. Breath sighing. Palate motionless.Paralysis of diaphragm. K.J.absent. Staggering gait. Heart irreg. force & rhythm. Sometimes rapid. Sometimes slow.	Sept.22.K.J. present but sluggish. Oct.10. Diaphragm acting well. 30. Went home well.	Return of K.J.
No.26. F.R. P.G.C.H. 8/11/93	9 yrs.	C	No history obtain able	A	+	+	+		Regurgitation once a few days before admission. Stumbles about and seems to walk to one side. Has complained of seeing double.	K.J.absent - tottering gait - nasal voice - eyes normal - palate moves only moderately	Nov.25. Voice well Dec. 7. K.J. just obtained.	Return of K.J.
No.27. F.S. ad.2/ 5/95	5 yrs.	C	8/3/95	A	+	-	+	Nasal voice	Diph. moderate severity; 2 other children died from it. Nasal voice noticed on April 22. and a day of two later regurgitation. Squint noticed on April 30. no difficulty in walking.	Nasal voice; regurgitation of fluids only,occasionally; palate motionless; pupils dilated loss of acco. ext.rectus affected in both eyes equally.Obvious diplopia,keeps one eye covered on looking at object.K.J.absent Heart,lungs &c.normal.	Uneventful. A fortnight after admission squint and palate paralysis became less and in another week gone. K.J. absent.	
No.28 A. W. Shadwell ad.24/5/95	5 yrs.	D	4 wks ago	A	+	+	+	Nasal voice	See Appendix I. Case. II			
No.29. G. W. ad.21/5/95	4 yrs.	D	6 wks ago	A	+	+	+	Regurgitation	See Appendix I. Case III			
No.30. A.G. Out-patient.	7 yrs.	C	21/12/94	A	+	-	+	Nasal voice	See Appendix I. Case IV.			

Name etc.	Age	Result	Date of Diphth.	K.J.	Palate	Legs	Eyes	1st.symptom	History of ailment previous to admission.	State on admission.	Progress.	Notes.
No. 31. E.F. Out-patient.	6	C	6 wks. ago.	A	+	+	+	?	Enlarged glands in neck and sore throat 6 wks. ago. Went to Concalescent Home 3 wks later. Came back 3 days ago with staggering gait and nasal voice. Next day couldn't see had to close one eye. Complains of pain in back.	Soft palate motionless. Voice very nasal. Paresis of Ext.rectus in both eyes. Inclined to count double with both eyes open K.J. absent; gait very staggering. Respiration sighing.	Refused to come into Hospital. Taken home confined to bed, and ultimately got well.	
No.32 F. G. Out-patient.	5	C	July 8. 1894	A	+	-	+	Nasal voice	Came up with probable Diph. on July 28. 1894. Sore throat and discharge from nose. No membrane seen anywhere. K.J. sluggish; no albuminuria.		July 31. Throat quite well, having been sore only 3 days. Nose better. Aug. 7. Nasal voice; K.J. absent, tires easily on walking. Diaphragm etc. normal. Aug.28. Nasal voice more marked. Regurg. past week: accommo. defective K.J.absent. Sept.4. Seems quite well. K.J.absent.	