Thesis on

Diphtheritic Paralysis of Accommodation

and Allied Conditions.

with special reference

to the Pathology of Such Paralyses.

William George Syme
"In Diphtheritic paralysis, so far as I have seen, the pupils retain their power of contracting to light, how they behave in an effort at accommodation I cannot say, and several ophthalmological friends to whom I have applied have not been able to tell me, but have kindly promised to observe the point. In view of the peculiar affection of accommodation in this disease, it would be most interesting to know whether there is normal contraction of the pupil during convergence for accommodation."


"Wir würden um einen Schritt vorwärts kommen wenn wir nur wüssten ob die Funktionstörung eigentliche in den Muskeln selbst oder in deren Nerven gelegen ist:" 

Menz. Nagels Jahrestheil. 1871.
I have been induced to take up as the subject of this Thesis, Post-Diphtheritic Paralysis of Accommodation and some allied conditions, because I have within the last few months met with several very interesting cases bearing on this matter.

It is however a subject which from the standpoint of the observer has several disadvantages, all which disadvantages are so many "points" in favour of the patient. These disadvantages (from our point of view) are briefly 1. That all patients, with very few exceptions recover; 2. That the condition is never permanent; 3. That all cases recover spontaneously, treatment, except of the most general type, being entirely unnecessary.

The pathology and therapeutics of the disease are thus somewhat uncertain, and there several different opinions of the pathology are maintained with much plausibility, and several drugs are approved by various observers which have probably little or no action on the course of the disease, but are thought effective because the patient recovers while taking them.

It is a subject however in which I
Manigault. De la paralysie diphtérique. 31.
take a great interest, and is one the proper consideration of which can hardly fail to be useful in increasing our knowledge on the one hand of the nature of Diphtheria, and on the other of the various relations of various organs.

Diphtheritic paralysis of accommodation has also this very important clinical interest that we are able to give an almost certainly favourable prognosis to one who having recovered from a serious illness of which all dreaded the name, is horrified to find that his sight is becoming lost.

"Il comprend l'incertitude des formes chez lesquels survient d'affaiblissement ou la partie de la vie, et celle du médecin qui se trouve en présence d'un accident aussi grave, et qu'on se trouve signalé comme conséquence de l'angième commence dans aucun des traités de pathologie."

Other palsy's consequent on Diphtheria are likewise of great interest, but shall avoid any mention of them so far as possible except where they have a direct bearing on any subject but they should lead away from the principal matter. There are other symp-
Symptoms occasionally present besides those of paralysis of accommodation, but I shall omit even these, and for the same reason.

Paralysis of accommodation being one of the most common paralytic manifestations, and being associated sometimes with affection of the pupillary muscles, affords a good ground on which to work at the pathology. I shall devote a considerable part of this treatise to the discussion of the pathology, since it is on this subject that opinion is most divided, and I find that the observations which I shall bring forward will assist in clearing up doubts. Others have recorded somewhat similar conditions noticed in some of their cases, but do not appear to have drawn from them the conclusions which I have.

The time of occurrence, the duration of the paralysis, and the treatment, I shall not discuss at length, since I have not been long enough in practice to have had opportunities of seeing the very large number of cases which one would require to see in order to formulate rules reliable in such matters. They are so uncertain, vary so very much in different cases without any apparent reason.
reason, and often result in so unexpected a way, that one would need many years of experience before being able to bring forward any new reliable facts on these subjects.

I do not propose to follow writers on Diphtheria into the discussion of the question whether Diphtheria and Emphysema are or are not one and the same disease. I merely say that from my own part I believe them to be entirely different; the one being a disease akin to, not infection, and not liable to be followed by paralytic; the other a disease ashyemic, contagious, often epidemic, often the result of nervous influence coming within the province of the Medical Officer of Health, and liable to be followed by paralysis.

Nor do I propose to discuss whether Galen, Hippocrates, Aretaeus and others of the Ancients recognized the disease; whether Diphtheria is identical with the Syphilitic or Syphilitic ulcer of the Romano-Era, or with the Morbus Shangulaturnus of the XVII century. These speculations are no doubt interesting, and perhaps to some degree instructive, but they are foreign to my present purpose.
* Bretonneau read his paper on this subject to the French Academy of Medicine in 1821, and in the same year his views were embodied by Guissart in various articles in his Dictionnaire de Médecine. Bretonneau did not publish his paper till 1826. (From Bibliography at end of Volonos on Diphtheria published by the Sydneyean Society.)

Mangault, loc. cit. 2.

Ironsean. Clinical Médecine. II. 544.

We may say, I think, that the history of Diphtheria began contemporaneously with the use of the name, when in 1826 Dr. Berton-Noira published his "Recherches sur l'Inflammation Spéciale du Throat Frangin et en particulier sur le Diphtherite." Since that time the books and papers written on the subject of Diphtheria are legion.

Paralysis following sore throat of some kind or other was first recorded, it is said, by Flourens in 1749. One reads in Flourens' Dissertation, says Manigault, "of a case in which the patient could not be said to be "hors d'affaire" till the forty-fifth day of his illness, "ayant toujours de la peine à s'""éprier, parlant du reg. et ayant la lèvre "tranchante." De or about the same year Glièr of Grenoble recorded some cases also. Trousseau says that in 1747-8 Glièr mentioned the case of his own son, who had suffered from sore throat.

"Pendant environ un mois après la guérison de l'au-""gine, l'enfant continuant à parler du reg. et ses aliments,""neul de souffrir le chemin de l'œsophage, revenant "souvent par les narines, principalement ceux qui "étaient les morons solides."

I doubt feel myself in a position to decide the
Medical Times and Gazette. 1:1859. 387.
ruval merits of these two observers to the honours of priority. Samuel Bard of New York made in 1771 some apparently independent observations leading to the same result, but Manigault says that the physician who first described with exactitude the paralysis which occurred after diphtheria, was Prof. Pillard, then (1834) a practitioner in the Department of la Vieille, and that Guinier de Verauray first noted the occurrence of amaurosis.

It is interesting to read the account of the first case of post-diphtheritic paralysis of accommodation diagnosed at the great Eystersital in Moorfields. It was that of a girl aged 15 who was under the care of Dr. Dicken. She had suffered from sore throat two months previously—at a time when and in a district where diphtheria was prevalent. During convalescence the sight had failed, and she was now unable to read any print. Distant vision had likewise deteriorated, but was improved by the use of a converging lens—[showing her to have been hypermetropic]. The pupils were of normal size and were mobile. Dr. Dicken had previously seen four similar cases in private and in speaking of them said, 'In all it appeared that the power of ocular adjustment was much interfered with.
Ironsean.- Clinical Medicine. Sydenham-

Transactions of Medico-Chirurgical Society of
was much as the use of a lens greatly improved the sight—did the affection depend on an impaired tonicity of the muscular apparatus of the globe inducing difficulty of regulating the focus?" According to Irons, the notion seems to have been the first to attribute the "Amblyopia" to paralysis of certain muscles of the eye—a view which Irons, apparently, not quite understanding the action of the ciliary muscle, discarded, even while admitting that the strabismus, ptosis, and other symptoms present in some cases were due to a paralytic condition of other eye muscles. He preferred to consider the cause of the "Amblyopia" to be albuminuria, a view which we shall see is quite untenable.

Diphtheria is nowhere specifically defined by Brotzmann or any committee of the Medical and Chirurgical Society of London appointed to investigate the subject, but from various passages in his works they have collected material for a definition which would express his ideas on the subject. They have thus compiled the following statement: "Diphtheria is a specific inflammation..."
inflammation of mucous membrane or of ex-coriated skin. That is, it consists in an inflammation and is at the same time a specific disease, analogous to typhoid, scarlet, and other fevers. Whether this inflammation is of a specific type is another question—implying, whether an inflammation indistinguishable from diphtheria may not be produced by other causes, is another question. The Committee publish also a case observed by Dr. Whitehead Reid, of membranous laryngitis which, though produced by the inhalation of Eau de Cologne, was clinically indistinguishable from diphtheria. Trendelenburg has seen the same effect produced by ammonia vapor, and carbonic acids may cause the same condition. Of course such cases are not followed by paralysis unless it may be a temporary paralysis of the part actually inflamed, because the disease stimulated clinically, but was not really, the specific disease called diphtheria.

The Committee go on to say that "an important point is the anomalous position which diphtheria holds in the rank of syphitic diseases, and the difficulty of deciding precisely what..."
Kingsford, Lancet, 2, 1858, 485.

Memoirs on Diphtheria, Sydenham Society.
"What is and what is not Diphtheria? It is at present an undecided question whether Diphtheria is as distinct and definite a disease as Scarlet fever or Small pox, and whether its poison is not readily generated under conditions of foul air and decomposing sewage. Its peculiarities of origin, absence of skin eruption, peculiar paralytic sequelae &c., and the fact that one attack, instead of affording a certain amount of immunity from the disease, rather predisposes to future attacks, all conspire to give to Diphtheria the "anomalous position" referred to. Dr. Kingford believed Diphtheria to be more closely allied to Syphilis than to any other disease, and there is a good deal to say for this view. Dr. Empey defined Diphtheria as a "specific disease characterized anatomically by the development of a perfectly specific pseudomembraneous exudation on the mucous or cutaneous surfaces." Neither the definition compiled by the Committee of the Medical Chirurgical Society, nor that of Empey mentions the occurrence of Diphtheria of the surface of wounds, of the occasional existence of which there can be no doubt."
I should have liked much to have been able to give an approximate statement in regard to the frequency of paralytic sequelae after Diphtheria, and particularly of paralysis of accommodation. Many circumstances, however, conspire to prevent this. In the first place, in many cases the diagnosis of Diphtheria is difficult, if not impossible; in the second, great diversity of opinion exists among physicians as to what actually constitutes Diphtheria. In every case one of Diphtheria in which there is a mem branous or "pseudo-membranous" deposit in the tongue, pharynx and tonsils? Does Diphtheria never occur without a distinctly membranous deposit? The first of these questions I trust be answered in the negative, and certainly the second. When Diphtheria is epidemic, it is probable that most of the cases are highly interpreted, but when the disease occurs sporadically, there is no room for doubt that many cases are considered to be simple sore throat, and perhaps not a few are classed as Scarlet Fever. For example, I have seen an apparently typical Diphtheritic sore throat, with a false membrane or what was indistinguishable from membrane, in
Murchison.

Semen. Two Lectures on Diphtheria. 1861.

see Jacob. A Treatise on Diphtheria. 19.

Lancet. 1. 1859. 1157.
a patient whose tongue soon began to show a distinctly strawberry appearance, and who, a short time afterwards began to desquamate. The case had been diagnosed as one of Diphtheria by a physician of skill and of considerable experience in both diseases, and until the case had progressed for some time I entertained no doubt of the correctness of his opinion. It is just possible that the complication of the two diseases was present here, as may be presented in the case of Typhoid fever and Diphtheria, but this is improbable. Jenner says "It is not uncommon to have a little diphtheritic exudation on the tonsils and arches of the palate and pharynx and have seen it extend to the tongue." This is surely a very loose use of the term "diphtheritic." Cordie in 1858 described Diphtheria as occurring with Scarletina; and Soth of Litchfield regarded Diphtheria as an abnormal form of Scarletina. Forbes wrote an important paper giving a careful description of the distinguishing characters of each disease, and in their Report the "Lancet" special committee on Diphtheria said: "There are some physicians of intelligence who maintain that Diphtheria is closely allied to Scarletina, in
Lanceet 1. 1859. 171.

"The nature of its poison, and should be
classed with it. — The confusion between
Scarlatina, Sore Throat and Diphtheria is
still so common as to be an element deserving
of consideration in any numerical analysis of
the returns of the disease. Again they say, "It
[Diphtheria] is often confounded with Scarlatina
"Angina and Gangrenous Lymphatic".
But besides all this, many of the paralyses
which follow are either not diagnosed, or are not
supposed to have any connection with the disease;
especially if the original throat affection has been
so slight that, as many say, the patient has al-
most ceased to remember it. Finally there are
some cases where the existence of more serious or
organic paralyses has caused the minor slight af-
ficution to be overlooked.

It is probable too, that just as different epi-
sidemies of Scarlatina vary in regard to albuminum,
and different outbreaks of Diphtheroid fever in regard to
peritonitis or to malignancy, so in different epi-
sidemies of Diphtheria the occurrence of paralyses
varies in frequency.

Under these circumstances I can only give
the numbers of cases of post-diphtheritic paralysis
observed"

observed at the City Fever Hospital, Edinburgh, as an indication of the average percentage of cases. The numbers are far too small to enable one to generalise on the subject; I merely submit them for what they are worth. See page 39.

Cromer considers that, taking various epidemics into account, of all the cases which recover, about one in every four has subsequent paralysis. Monckton of Maidstone total thunder that he thought about 3 percent of all cases suffered from paralysis; doctors give his own percentage of paralysis among cases which recovered from the original disease as about 8½, that of Roger as 30, that of Bouillar as 9, and Lagrange as 8. None of these authorities who are thus seen to differ very widely, could depend upon the proportion of cases of paralysis of accommodation, but only of paralysis generally, and it is more than probable that some of such cases are omitted in their calculations.

I think
I think it will be well at this stage to stop a moment to glance at the nervous relations of the actions of Accommodation, convergence and contraction of the pupil.

These three movements are closely connected, but connected only to a certain extent. Thus convergence cannot take place to any great degree, without at the same time accommodation and Mydriasis occurring; accommodation only to a limited extent, takes place without convergence and contraction of the pupil. But lateral movements of the eye, in which one internal rectus or the other is used are unaccompanied (—as a rule; exceptions have been noticed—) by any change in the pupil or by accommodation; and extreme Mydriasis may be brought about—by the action of a bright light—without the occurrence of either convergence or accommodation.

In these circumstances one would expect that two centres prevailed over the action of the internal recti, and two over the sphincter pupillae, and such is nearly what we find. — in the case of the pupil, a nervous arrangement to provide for "light-Mydriasis," and one for "convergence."
Towers. loc. cit. note to page 159.


Adamik. Centralblatt für Medizinischen Wissenschaften, 1870, 12.
"Convergence-sympotie" in the case of the internal Recti, one centre for lateral movements and one for convergence. The Muscle of Accommodation, the Constrictor pupillae and the Internal Rectus all receive their nervous supply through the III nerve.

Below the Aqueduct of Sylvius, beneath the Anterior Quadrigeminal bodies and the posterior Commissure there lies a mass of gray nervous tissue on each side of the middle line. It is from a column of nerve cells which forms the anterior part of this mass that the III nerve has in dogs been ascertained to take its rise. Probably in man (Cowsers) the origin is a little further back than it is in dogs, and does not extend anterior to the position of the posterior Commissure. Sturge considers that some fibres arise in this region, some however go to the Vale of Meecnena, some to the Corpora Quadrigemina, and some to a nucleus in the floor of the III. Ventricule, which is the centre for Accommodation.

To Adamitis belongs the credit of having localized the place of origin of the III nerve. He found that stimulation of part of the floor of the Aqueductus Sylvii had results similar to that
Heuser and Völkers. Archiv für Ophthalmologie. xxiv.

Gowers. loc. cit. 157
Kähler and Rick. Centralblatt für Augenheilkunde 1883.

Middle
Eye
Ciliary Muscle
Sphincter Iris
L. j. L. j. Sup.
Rect. Inf.; Obliq. Sup.
Obliq.; Sup. II m. e.
stimulation of the trunk of the III nerve, and justly concluded that there the nerve arose. He was inclined to place the seat of origin of the pupil contracting fibers rather behind the other centers, but Hensen and Voelckers differ from him and place it more anterior. By a series of experiments Hensen and Voelckers established that in the dog the most anterior of these centers is that of accommodation, next follows that for the action of the iris, "whilst the remainder of the nucleus, comprising its chief part, subserves the external ocular muscles in the following order from before backwards: Internal Rectus, Superior Rectus, Levator Palpebrae Superioris, Inferior Rectus, Superior Oblique." Fahler and Pick observed a case which seems to throw a little doubt upon the correctness of this as true for man. They think that in man the centers are not arranged simply in a row, but suggest that they may be situated in the manner shown in this scheme (see opposite page). Both series of observers at any rate believe that the ciliary muscle is preceded over by the most anterior nucleus, and next follows that for the iris. Hence and Voelckers place next that for the Internal Rectus.
Quoted by Sturge, doc. lit.
but Kühne and Rick separate the two by that for the Elevator Palpebrae Superiores, we shall see that for certain reasons the theory—supported by experiment—suggested by the former is more likely to be correct in this particular than that pronounced by the latter observers. That part of the general place of origin of the III nerve which is situated just at the junction of the Abducent of the inferior and the III. Ventricie subserves the actions of Accommodation, Convergence and Contraction of the pupil and may be called the Combined centre for the three actions so closely bound up together.

The impulses resulting in contraction of the pupil as the result of light striking the retina, reach the centre through Magenta’s fibres, which connect the floor of the Sphenoid Abducent with the Corpora Quadrigemina.

Adanit found a spot in the Corpora Quadrigemina irritation of which produced upward movement of the eyes; a little behind that and to the right and left centres for lateral movements of both eyes together and behind these a centre for downward movement and convergence.

The pupillary fibres of the III nerve on leaving the
The centre, pass first through the Lenticular ganglion. They, along with the motor fibres to the ciliary muscle, leave the trunk of the nerve along with the branch to the Superior Oblique muscle, branch off from this trunk and join the Lenticular Ganglion as its motor root, whence they proceed to the eye. They pierce the Sclerotic above and below the nerve in 8 to 12 or 16 bundles called the Short Circular Nerves, and run to their terminations in iris and Ciliary Body along depressions between the Sclerotic and Corneal coats.

I do not mean to deal with the Centres for the other muscles of the eye.

My reason for entering so fully into the arrangement of centres will become apparent when I come to the Pathology.

The Symptoms of Paralysis of Accommodation must now engage our attention. They vary according to the static refraction of the eye and the completeness or incompleteness of the paralysis.

In the Emmetropo, or person of normal refraction, paralysis of accommodation does not in any way affect the distant vision.
but if there be, as there often is, coincident dilatation of the pupil, there may be so much dazzling in consequence of the abnormally large supply of light as to cause confused vision. But the patient is unable to read, to sew, or to do any work which necessitates the correct and accurate view of small objects near at hand. If the paralysis be not complete, and his ciliary muscle retains some power, he may be able to make out print held at a considerable distance from his eyes, in exactly the same fashion as a presbyope. A pair of convex lenses of a strength not exceeding 4 dioptries, worn by the patient will enable him to read as well as ever, and at once make the diagnosis unmistakable.

In the case of a hypermetrope, or person of abnormally low refractive power, who requires that the ciliary muscle should lie in an active state, to enable him to see well at a distance even, distant vision is rendered bad by the occurrence of paralysis. Gowers is quite under a mistake when he says that the distant vision of hypermetropes remains good. A reference
to the cases I shall mention later will at once settle the matter. The defect is at once corrected by a convex lens, and the condition is then obvious. It is necessary to enter here a little more into detail than I had meant to do, but as this is a point of which observers so accurate even as Dr. Gowers go as far, it is worth while to do so; I shall, however, be brief. Under ordinary conditions the presence of a small amount of hypermetropia is no bar to good vision; indeed, it is a curious fact that savage nations which depend for their livelihood on hunting, and therefore on the accuracy of their distant vision, are, like the lower animals, hypermetropic. The North American Indians, for example, whose power of vision is proverbial, are all hypermetropic. If then a young person, let me say a girl of the age of 10 (see case 40) presents herself, whose vision when tested, is found to be only 3/60, which is raised to the normal standard by the use of a lens of +1.5 D. (20 times convex), we at once conclude that the case is one of paralysis of accommodation, since so trifling an amount of hypermetropia would under normal circumstances produce
no symptoms whatever. The defect which would otherwise occur is overcome by the constant utilization of a small amount of power of the ciliary muscle. If so great a defect of vision was produced in a child of that age by Hypermetropia, the degree of manifest Hypermetropia would be, not 4 dioptres, but certainly not less than 6. The near vision of Hypermetropes is even more interfered with than that of Simetropes, but is precisely the same way, and will be corrected, and brought up to normal by the use of a lens of +4 D (dioptres) added to that lens required for distant vision.

In both cases, if the paralyses be only partial, a singular complaint is sometimes made, viz. that all objects near at hand appear too small. This Micropia is produced by the necessarily increased endeavour to contract a paralytic ciliary muscle giving rise to the impression of a greater contraction than has really occurred; and since one ray at least in which the edge of the eye of object is by the unconscious estimation of the degree of contraction of the ciliary muscle necessary to focus it, the sensation of greater contraction...
Berry. Subjective Symptoms in Eye Disease. p. 57.
gives an impression of greater proximity, and 
"as its image is of course of the same size as it 
would be were its distance more correctly estimated, 
it appears smaller." This erroneous impression 
of size is thus analogous to the "false projection" 
of objects which is found in cases of paralysis 
or paresis of the external ocular muscles.

Myopia can only occur when some power is 
retained by the ciliary muscle, and is not 
present when the paralysis is complete.

In the case of a myope, or person whose 
refractive power is greater than normal, it 
is important to remember that paralysis of 
accommodation may give rise to no symptoms 
whatever. If the patient be so far myopic as 
never to use his ciliary muscle even 
when reading, it is obvious that paralysis 
will not affect his vision, and the condition 
can only be made out by careful measure-
ment of his "range of accommodation." This 
may be much reduced or may be entirely 
absent according to the degree of the paralysis.

If he is in the habit of using a certain amount 
of accommodation when reading, the lack of 
it will be shown exactly as in the other cases.
Parkinies Images.
A. During Rest.  B. During Accommodative Effort.
by the recession of his "near point."

Another means of diagnosing paralysis of accommodation, but not so satisfactory as that given above, was made use of by Shelley-Brock in his Series of Cases. He observed that when a near object was looked at by one of his patients, the plane of the iris was unaltered from what it had been when he was fixing a distant object, and the anterior chamber remained of the same form and depth as before. Since the iris rests on the anterior surface of the lens, and the anterior surface of the lens advances during the act of accommodation, thus reducing the depth of the anterior chamber, it was manifest that as these changes did not occur, accommodation did not take place. The absence of all change, during the effort to look at a near object, in the size and position of the images of外部的 formed upon the anterior and (roughly speaking) posterior surfaces of the lens, also indicated the absence of accommodation.

The degree of loss of sight, then, varies considerably. The Hylopi may be unconscious of his defect; the Emmetropes in whom paresis rather than paralysis has occurred, may be able
Scheby: Buck. loc. cit.
able with some difficulty to read, while
the high Hypermetrope, whose ciliary
muscle is completely paralyzed, can hardly
distinguish the letters of the largest print.

We now come upon a fact which one, a
priori, would hardly have expected to be the case.
It was observed by Schenkendorf, "Bemerkens-
werth ist dass sich unter allen Patienten nur
zwei Myopen fanden; theils erklärt sich dies
wohl aus dem jünglichen Alter, als dem
"der Diphterieis am meisten verfallenen (die
"meisten Patienten waren unter 14 Jahren), theils
"auch wohl; weil bei kürzlichdigen die Be-
"schwerden am geringsten sind." On re-
ference to my cases (page 59) it will be seen that
all the patients were Hypermetropie; and I
think that this is a point of some interest.
That the condition may, in cases of high Myopia,
be entirely overlooked, is not wonderful; but it
could hardly escape observation in the Emmetropie
if it were present; since their near vision is
deriously affected by loss of Accommodation.
Of course, a less complete degree of paralysis, which
would only put an Emmetropie to some inconvenience by removing his "near point," and might
by Wagner, for example, in Lexicon Sceptrum, 1790.
thus he easily overlooked, would have a
much more serious effect upon a Hyper-
metropia and could hardly be passed over.
Still it seems probable that Hypermetropes
are more liable to this form of paralysis
than Emmetropes or Myopes;—it may be
that their constant use of the ciliary muscle
pre-disposes to its paralysis. I do not know
in what other direction one could seek for
an explanation of this fact, but I confess
that the one I have suggested appears very
unsatisfactory. It is not uncommon
for a similar thing to happen after fevers,
though to a much less extent; Hypermetropia
which before was latent, becoming temporarily
manifest. This is produced in such cases
by conjunctival weakness of the eye, the same in
nature and cause as that of the arms and
legs.

What cannot but be regarded as a
serious mistake has crept into numerous
books and papers dealing with this subject,
viz: it is said that in a few cases Myopia
has occurred. Now if Myopia has really
been wrought about by Diphtheria, it can
only
only have been by the occurrence of an attack of Atrophying Chorioiditis during the asthenic state of the patient; and such My- 
opsia will be permanent. What is much more probable is that a false Myopsia has 
been brought-about by paralysis of accommodation, 
as it is obvious that such a condition cannot possibly produce a true Myopsia, since no para-
lysis could cause an increase of refractive power. 
High Hypermetropes even in health sometimes behave like Myopes. Their distant vision is bad, 
and since no strain of accommodation could bring their vision up to what it should be, they 
give up the painful efforts alike at Convergence and accommodation, and holding the print 
close up to their eyes are able to read wonder-
fully by means of the large but very blurred images formed thus upon the retina. A moderately 
high Hypermetrope whose accommodation has 
become suddenly paralysed, may, by acting 
thus, simulate Myopia. In no other way 
are I able to account for this mistaken state-
ment, which believe myself to be correct in 
stdating, no oculist has ever confirmed. 
Some observers, principally those of France, 

School Buck. loc. cit.

Weber. loc. cit.

Mary. loc. cit.

Medical Times and Gazette. 2. 1875. 374.
have noted that in Diphtheritic paralysis of accommodation they have observed a deterioration of vision more severe than can be accounted for by simple loss of the action of the ciliary muscle. This Schepis-Bouch can account for in no other way than by supposing it to be due to "Lento-Astigmatism", as Volcker suggested it is not produced by dazzling from a dilated pupil, since it occurs sometimes when the pupil is not dilated. Weber also observed this condition and thinks it may be owing to a diminished "receptivity", or sensitiveness (Empfänglichkeit) of the nerve elements, similar to that of the common sensory nerves in other parts of the body.

But I cannot but agree with Wang that there is no such condition as that spoken of, and if one places before the patient's eyes suitable convex lenses, his vision rises at once to normal.

One must, however, be aware of concluding that bad vision following Diphtheria is necessarily due to paralysis of accommodation, and thus be betrayed into giving a prognosis which may unfortunately be too sanguine. Mr. Bouchut it was who pointed this out. He examined
twentysix cases of general diphtheritic para-
lysis, and found neuro-retinitis double, in
twenty-two of them. I cannot help thinking
that M. Boulebort has been deceived in some of
his cases by the apparent, though perhaps partly
real, congestion of the jugular which is one
of the signs of Hypermetropia, and is very
apt to delude one into the idea that an in-
flammatory affection of the jugular is present
in its early congestive stage. His results are
entirely exceptional. I do not suppose that
any other observer has met with one case of
Neuro-retinitis in fifty of Diphtheritic paralysis
or perhaps much more; though Bright's disease,
and therefore perhaps Neuro-retinitis, has been
known to follow this singular disease in a
very small number of cases. In the conclusion
concerning the pathology of Diphtheria which he
draws from this phenomenal series of observations
and into which it is not necessary for me
now to enter, I do not see any way to agree;
but M. Boulebort has done us a service
by warning us against the hasty adoption of
an incorrect opinion.
Paralysis of Accommodation, it must be remembered, is a condition which occurs physiologically as well as pathologically, but not in the same class of patients as that in which Diphtheria is most frequently found.

Under physiological conditions it is present in all persons who have reached the age of 65 years, that being the age at which all power of accommodation has ceased. If then a person aged 65 or more should acquire Diphtheria, we could not tell whether his ciliary muscle were paralysed or no, for at that age it has no power over the lens, which is then so hard as to be unaffected by accommodative efforts. Even if its muscle were paralysed he would in no way suffer as regards his vision from any other than of 65.

Pathologically, apart from post-diphtheritic paralysis, Cycloplegia occurs under several conditions:

1. From the use locally of a mydriatic such as Atropin or Belladonna, or internally of such a drug as Belladonna. In these circumstances the pupil is always widely dilated. In the case of Atropin as in the case of the opposite
action of scirrux, the effects on the two
dimensions, ciliary and sphincter iridos, are
not simultaneous, that is to say, do not begin
at the same moment and cease at the
same moment, but are both present at the
same time and are inseparable. There
is no known drug which locally applied or
administered internally, will dilate the
pupil without paralysing the ciliary muscle,
or vice versa. This fact as we shall see
has an interest in connection with the causa-
tion of Diphterie paralysis.

2. In Glaucoma. This came may be briefly
dissolved, occurring as it does in 99 cases out
of every 100, in persons much older than the
usual Diphterie patients, and being often uni-
lateral, or at least more advanced in one
eye than in the other. Here also there is
in uncomplicated cases — semidilatation
of the pupil. The cause of the paralysis
in Glaucoma is generally considered to be
pressure on the ciliary nerves as they pass
forwards from their place of entrance into the
globe round the optic disc to their termina-
tion in the Ciliary Body and Haustral Fossa.
3. In paralysis of the III nerve, the cause of which may be syphilis, brain tumour, rheumatism or what not. In such cases, even though all the branches of the III nerve are not affected, if the twigs supplying the ciliary muscle are paralyzed, so are always those supplying the sphincter iris. I think I am justified in saying "always." I find no trace in literature of any case to the contrary; indeed such an idea is never mentioned as the ciliary and not the iridic branches being palsied in a case of partial paralysis of the III. It seems never to have occurred to anyone. Paralysis of the III. is moreover most frequently unilateral and Diphtheritic paralysis never shows itself in this form; one, two, or three of the muscles supplied by it may be paralyzed, and these not always quite symmetrically, but paralysis of the III. nerve either unilaterally or bilaterally, consequent on Diphtheria, has never been observed.

4. Rarely but occasionally paralysis of accommodation occurs in Locomotor Ataxy. Paralysis of one or other of the external ocular muscles...

Hutchinson. Transactions of Medico-Chirurgical Society of London. 1878.

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**Analysis of Accommodation**

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Meyer. Practical Treatise on Diseases of the Eye, p. 152.
developed on both sides, and he "cannot but regard it as highly probable that in almost all the cases the affection is due to syphilis."

6. Injuries of the eye may give rise to cycloplegia, accompanied by iridoplegia; however, without it may be, any further damage to the organ. Such cases rarely recover, if ever, and it is not quite easy to say what the precise pathology of them is. Of course they are unsymmetrical.

7. Paralysis of accommodation and pupil movement has several times been remarked upon as the forerunner, it may be for some years, of mental derangement. In such cases it may, for a time at any rate, be unilateral. Evidently some obscure central changes, perhaps of a vascular nature, have been going on, which produced these early symptoms.

8. Meyer believes that paralysis of accommodation may be occasionally of rheumatic origin. He does not mention the state of the pupil in such cases, the existence of which I very seriously doubt. I shall have to refer to this matter later.

9. Paralysis of accommodation has also been seen under certain other conditions: — in diabetes, poisoning,
Hutchinson: Ophthalmic Hospital Reports. vol. 37.

Neben Accommodations-Beschränkungen bei Zähnebleiden.

page 120.

poisoning by raw meat, sausages, unwholesome fish &c., 
i.e. Trichinosis, and as the result of severe nervous shocks. It has also been observed 
to follow strain of the ciliary muscle, work close to a hot fire in the case of glass blowers, and 
as a consequence of Herpes Zoster.

10. Hermann Schmidt found paresis of Accommodation sometimes to a high degree, but 
never reaching actual paralysis in consequence and during the course of tooth ache. 
So this matter also I shall have to revert later. 

With the exception of Hermann Schmidt's observations there, in almost all, if not all the 
cases of Cycloplegia mentioned, whether the condition of the pupil-movements is stated, 
we find also induceplegia, usually with a dilated pupil.

Hughlings Jackson says: "So far as I know, 
paralysis of the ciliary muscles on the two sides 
is only found in a high degree after Diphtheria."
Manigault. Loc. cit. 84.
It is now my duty to discuss the question:
In what relation does the subsequent paralysis stand to the antecedent Diphtheria? There are three possible answers:—

1. That the Paralysis is simply a coincidence.
2. That the Paralysis is not related to Diphtheria more than to any other acute disease.
3. That the Paralysis is caused by Diphtheria and is peculiar to it.

The first of these answers, though formerly not doubted, and still in many cases entirely agreed upon, is certainly untenable. As I have said, it is well-nigh impossible to obtain an accurate estimate of the frequency of the condition, but the "progressive fréquence" of paralysis of some organ or other is certainly well-established. "Hypothe" une relation evidente de cause et effet entre la "présence des fausses membranes ..., et une variété de "paralyse qui mérite le nom de paralysis diphtherique."

It was at one time suggested to explain the occurrence of paralysis of Accommodation by saying that when weak during convalescence the patient had "caught cold," and thus acquired a "rheumatic" paralysis; but bilateral simultaneous rheumatic paralyses are very rare; indeed I
Medical Times and Gazette. 1. 1861. 523
I may say that double paralysis of accommodation never is found in consequence of rheumatism, while this bilateral form of that paralysis is, as we shall see, the rule, quite invariable, in Diphtheria.

If the second of these answers be the correct one, one must look for a cause of paralysis common to all acute diseases; and this can only be — is by those who uphold this theory stated to be — Asthenia, Anaemia, Debility produced by the virulence of the original malady.

That the third answer is the true one I am quite certain, and it is partly upon the truth of it that the theory which favours of the connection of the subsequent paralyses is based.

Dr. Ebert showed in the Medical Society of Berlin in 1861, a case of Diphtheria in which there was paralysis of the palate and the eye. In the discussion which followed, von Graefe went the length of denying all connection between paralysis and Diphtheria. He considered that paralyses were frequent after every grave disease of the nervous system, lymphoid, erysipelas, etc. Every disease which was
Sermack: Serrin's Dictionary of Medienie - Art: Diphteria.
was accompanied by fever was also accom-
panied by paralysis of accommodation and
a semi-paralytic state of most of the muscles
of the eye. He also stated that he considered
the proportion of cases given by Davinault
(30 of paralysis of accommodation, and 60 of para-
lysis of the palate) as very small compared
with what might be found after other acute
diseases.

Sir John Rose Larmack writes: "It is necessary
to call attention to the fact that paralysis, in
various forms and degrees, occurs associated
with, or as a sequel of, Typhoid Fever, Relapsing
Fever, Cholera, Diphteria, Smallpox and Pemmican,
and that in all they are attributed by Guibler
and others to an essentially similar state, in-
herent to most fevers and acute diseases.
Though their common phenomena present
points of difference, they have common funda-
mental characteristics, namely, they seem all
tobe as a rule of peripheral origin; they
all manifest a natural tendency to recovery;
and when they become protracted, they are apt
to become permanent from atrophy of the muscles
causd by long disease."

Guibler
Hubler, Archives Générales de Médecine 1860-61, quoted in Medical Times and Gazette. 2. 1865.
Gubler states that he has known cases of various paralyses occurring after Measles, Scarletina and Variola — in the case of the last fever the paralyses having affected the palate. He holds that all such paralyses including diphtherial, are due to debility and nervous exhaustion.

The materials of discussion of the three theories, or answers to the question I have propounded, I place under 10 headings which I think exhaust the various aspects of the subject:

1. The remarkable frequency with which attacks of Diphtheria are followed by paralyses of some organ or set of organs.

Of 37 cases of Diphtheria admitted to the City Fever Hospital between 1 Nov. 1888 and 1 April 1889, ten died in the early stages of the disease. Eight of the cases which recovered were known to have suffered from paralyses, and six of the fatal cases also. Possibly more of the fatal cases were affected, for as some were admitted almost in a dying state they were not examined. And of the cases which recovered sixteen were never heard of after leaving hospital, and
And of these some may very probably have become paralyzed later. However, taking into account only those known to have been paralyzed, we have fourteen of thirty-seven, a percentage of very nearly thirty-eight. [see also page 14].

2. The comparative rarity of paralysis after other acute diseases. If, as has been stated, the cause be the anemia and general debility produced by acute disease, one ought to find paralysis quite as frequent after other severe diseases in which the anemia is great as after Diphtheria. Recognizing the difficulty or rather the impossibility of maintaining this statement, Ernack falls back on the most fallacious assertion. He says "During the course of, and in the convalescence from, no other acute disease, are anemia and anemia so constant and so profound as they are in Diphtheria." Is it so, is the Anemia, is the Asthenia more constant and profound after Diphtheria than it is after Smallpox, Syphilis, Cholera, Pneumonia? I think not. And what of the later stages in cases of Phthisis, Cancer, and Pernicious Anemia? The vitat
Itcher, Véichonu Archiv, vol XXVIII.
posts could hardly be lower than in these diseases, and yet we never hear of a true localized paralysis occurring, far less being frequent in them, apart from the direct effect of such a cause as a haemorrhage or growth of a node.

Of the acute diseases mentioned above take Acute Pneumonia as an example. In how many cases of persons who recover from this disease, and pass through a long convalescence, is paralysis of any organ found? Not one in five hundred, perhaps not one in a thousand. Convalescence is seldom longer and more tedious, debility and asthenia are seldom more profound than they are after a severe attack of Enteric Fever, and does the percentage of cases followed by paralysis even approach one, the lowest estimate recently made of that of Diphtheria? How paralytic and other nervous disorders are very far from being unknown as a consequence of Typhoid Fever, and bare tell the same is true of Cholera. Weber cannot accept the theory that weakness and anaemia are the cause of the paralysis; he says, "Es ist zwar wahr, dass in der Mehrzahl der Fälle eine gewisse..."
It is an interesting criticism, though not an available argument, on the alleged anaemic cause of the paralysis and its connection with all acute diseases, that the majority of writers on pneumonia, fevers, etc., have never mentioned the subsequent occurrence of paralysis, while I suppose that no one has within the last twenty or thirty years written even a pamphlet upon diphtheria without reference to subsequent nervous phenomena.

There is one other point in this connection deserving of our attention. It is known that diphtheria occasionally complicates typhoid fever—a fact of great pathological interest which I must refrain from commenting upon—and from the somewhat "unperspicuous" statement of Jenner already quoted (page 12) as well as from observation, one gather also
also run its course at the same time
with scarlatina. Now, if the general
symptoms are severe, the throat symptoms
maybe overlooked or thought little of;
and, should paralysis follow, the case will
probably be considered one of nervous
sequelae of enteric or scarlet fever as the
case may be.

3. The curious selection of the seat or seats of
paralysis, and

4. The fact that while paralysis of one part, etc.,
other parts are in a state of healthy activity.
The two commonest seats of paralysis by far
are the throat (soft palate and pharynx) and
the eye. The remarkable predilection for
these special situations is not what one
would expect from simple anaemia. So
far as I have been able to discover, paralysis
of the palate alone, or of the ciliary muscle
alone, or of one followed by the other is
practically unknown as a sequel of any
other disease. Speaking of paralysis following
typhoid fever, Drury says:—“In very rare
instances paralytic weakness after typhoid
has been associated with paralysis of the
soft..."
"Soft palate, but even these cases do not closely resemble Diphtheritic paralysis. There is never the paralysis of accommodation or the slow progress of paralysis from one part to another. Epilepsia, though occasionally, and Variola though not infrequently followed by paralysis, does not seem ever to be complicated with palsy of the soft palate or the ciliary muscle.

After Diphtheria it is not uncommon to see a patient who is able to take a walk of from a few miles without difficulty, but whose ciliary muscle is completely or almost completely inactive and down whose nose perhaps fluids flow on any attempt to swallow. Jonasen mentions a case where a man affected with paralysis of the palate was able for some time to perform his duties as a "dischargers of barges." Donders—it is sad to have to tell him now the late Prof. Donders—in his great book says: "After Diphtheria we often observe the want of position in this respect. If individuals are sometimes overtaken, there are others who feel perfectly well, have resumed their work, and run for hours, while the paresis of accommodation con-
"times. They complain of nothing else than their vision of vision. This regeneration of strength in one set of muscles during a time of almost complete paralysis of another set is very remarkable, and very little suggestive of an asthenic condition. Not only so, but there is one symptom which is apt to remain long after all the depressing effects of the original disease have been recovered from, and which can by no stretch of imagination be referred to simple asthenia: viz. the persistence of absence of the (correctly or incorrectly named) tendon reflex of the knee jerk. While the paralytic affections have departed, as a rule, by the end of the second or third month, this loss of reflex may be found still existing in the eighth, eighteenth, or even the twelfth month; the lower limbs, if they had ever been affected at all, having long before returned to their normal condition in every other particular.

5. The paralysis does not occur at once.
This rule is not universal, I am aware, but it is certain that in the great majority of cases no paralysis is noticed, with the exception (not always to be found) of that of

The...
The soft palate, until two or three weeks after the healing of the ulcer, and until the patient is up and going about, or has at any rate for some time begun to put up strength again. "C'est presque toujours à un épisode assez éloigné de moment où la fausse membrane a disparu deux ou trois semaines après l'excision de tout phénomène mobile du côté de la gorge, qu'on voit survenir les premières signes de la paralysie. Les malades respirent moins fortes, on les croit en pleine convalescence, lorsque apparaissent de nouveaux phénomènes morbidès." Of the history were that the patient had had a sore throat; that when he tried to get up, he was found to have his lower limbs paralysed, that his palate and accommodation were also in a state of palsy; and that from this condition, he slowly and gradually recovered;—then one might suppose the condition to be a result of asthenia. But the history of real cases is far different. The patient is convalescent, is up for several hours daily, takes his food well, and is becoming stronger day by day, when he begins to "stutter", to
"Splutter" in taking his food, some of which is apt when fluid to be returned through the nose; and that a week or ten days after that, he complained of inability to read. Some time after, when his voice is already recovering its natural sound, weakness in the legs begins to be troublesome and Anaphrodisia may remain for many months.

It has, I believe, been sometimes observed, that while the disease was at its height the palate was uninsensitive, a condition which became ameliorated with the improvement in the ulcer, but which subsequently returned as one of the truly paralytic effects.

The two of Jenner's cases are very instructive in regard to this delay in the recurrence of paralysis. (a) Mrs. J., aged 12, took the disease on 8th February and was convalescent on the 22nd of the same month. She was "well" on 7th March. On 17th March she looked in tolerable health; there was no enervation, but slight pallor. She was then quite unable to walk a step; there was also some loss of power in the arms, and fluid food regurgitated through the nose. On 23rd March she

Dostoevsky quoted by Jacobst.

Weber. loc. cit.

Jacobst. loc. cit.

Greenhow. quoted by Weber.
she was no better, became powerless, and
died in a few days. 6. Boy of 22
months. He recovered from Diphtheria in three
weeks and could then run about and seemed
well. Ten days later he was noticed to stagger
and paralysis came on; he was pale but not
at all emaciated. Ten days later, the
right side of his face and neck were paralyzed; he became paraplegic
and died in a fainting state two days later.
Do these cases, which are not exceptional, but
quite typical, read like histories of an acute
condition?

6. The paralytic symptoms are frequent after mild
or comparatively mild primary attacks.
Wagner and Dietl have indeed stated their
belief that such cases are unreliable than are
the severe forms like followed by paralysis; and
Keypart of Birmingham, who had great experience,
wrote to Weber that he thought great severity of
the original disease diminished the likelihood
of secondary symptoms. Jacob is seems to have
entertained a similar opinion. Others, however,
believe such symptoms to follow more frequently
in the severer cases. Greenland's opinion was, that

*This is considered by many to be a very bad sign.
Though the opposite no doubt does occur, yet as a rule severe paralysis followed severe attacks of Diphtheria more often than a mild one. However this may be, whether paralysis follows severe primary attacks or mild, most frequently, it is quite certain that in an unknown proportion of cases, paralytic symptoms do follow throat affections so mild, that either they have been forgotten or at all events were barely suspected of a Diphtheritic origin; that though a bad primary attack is likely to be followed by nervous symptoms, palsy in such cases which are not especially severe, is scarcely fully recognized. This fact, he says: "Diphtheritic paralysis bears no relation to the intensity, extent, or continuance of the characteristic local manifestations of the disease. It is no doubt most commonly as a sequel to the severe forms of Diphtheria that paralysis occurs, but on the other hand, it is by no means unusual in the present day for chronic disorders of universal or local nature to show themselves in persons who have had Diphtheria in apparently its mildest form. P. Flanigan has mentioned 2 cases.
Jacobi, loc. cit. 1497-98.

Edinburgh Medical and Surgical Journal, May 1870.
a certain number of cases of this kind—cases in which paralytic affections were more or less general, and more or less persistent, followed peculiar disease situated or the pharynx and occupying a very limited surface. Jacobi says "Not infrequently it is just these cases in which neither fever nor local phenomena were of marked intensity, which are followed by paralysis;" and again: "It is the fact that diphtheritic paralysis frequently appears as the severest case while existing the apparently mildest." (Italics are Jacobi's). See in this relation also an interesting case published by Graniger Stewart of rather severe and widely distributed paralytic symptoms which followed an attack of sore throat, which was only known to be of diphtheritic origin by the fact that the patient's brother had, immediately before his (patient's) own attack pharyngia, had suffered from diphtheria. The patient suffered from paralysis of accommodation, among other symptoms, such as paralysis of palate, anaesthesia larynæ and numbness of the legs, and from "an almost complete paralysis of the circular fibres of the iris"—a somewhat unusual condition.
Hermann Weber. Friedens Archiv: XXX.
as we shall see. My case A.M. (No. 3) also illustrates this matter of acute diphtheria followed by severe paralysis, so do the cases of J.M. (No. 1) and J.R. (No. 2).

Hermann Reuter gives a case of widely distributed paralysis affecting palate, eyes, legs, and arms, with sensory as well as motor symptoms, and associated with impotence, which followed an attack of diphtheria, of which the primary symptoms were so mild that I should not have given the name of diphtheria, if I had not known that often this disease affects some members of a horse so slightly as to remain almost unnoticed, while in others it assumes a fearful severity, quite analogous to the great differences which have been observed in whooping-cough, scarlet fever, typhus, and other epidemic fevers.

So far as I have been able to make out, there appears to be no rule of frequency in the subject; there seems to be no special liability of one variety or another of diphtheria, but the special form of paralysis is just as conspicuous in selecting its victims as the original disease is. I should certainly

[Signature]
not go the length of making any comparison of frequency in the two circumstances. It is an interesting fact that not only do some families show a special tendency to diphtheria, but even also to diphtheritic paralysis.

7. The fact that pathological changes have been found in the nerve centres, nerves, and muscles. That such changes have been found no one can deny, but the present is not the place to discuss them. They come in more naturally when I am writing on the pathology of the paralysis.

8. The changes observed in the electrical reactions of the paralysed muscles. A very curious and apparently inexplicable fact in connection with this matter is that so many different observers have obtained such diverse results, and even the same observer obtains different reactions in different cases of the same disease. Therefore one cannot use this change in electrical reaction as anything but a very secondary argument until we understand the meaning of the variations. If the poetic state of the muscles were really a mere academic weakness, one would certainly not
Gouven. loc. cit. II. 533.
Expect to find the marked alteration in electrical reactions which is sometimes noticed. That there is even sometimes a changed relation of effect to stimulus is an indication that some tissue change has occurred, and that mere asthenia is not the only cause of the paralysis.

So the galvanic current Lasèpas and Rumpf found the reaction normal, while Leube found it diminished; to Faradic electricity Krehl-Ebing found no response, Rosenhain found the reaction diminished, and Jaffray found it increased. Jacobson, from whose useful little book this summary is taken, found a very uncertain reaction, at times normal, occasionally diminished, and sometimes increased. Forner found no change when the affection was slight, but if it was severe and lasted for two or three weeks, he then found diminished irritability to Faradisation, or even entire loss of response. The senses still respond to Veltasium, but their reaction to it sometimes becomes altered so as to resemble that found in peripheral paralysis. It is not necessary
Donners. loc. cit. 560.
necessary to quote more authorities to show the varying results.

9. The Symmetry of bell-Diphtheritic paralysis is, it must be admitted, distinctly more suggestive of an asthenic than of an organically paralytic affection. So far as I have myself seen, and so far as I have been able to discover in literature, accommodation paralysis is always symmetrical. But it must be remembered that the action of the two ciliary muscles is quite as uniformly and necessarily bilateral as that of the two parts of the diaphragm. In a case of Anisometropia, for example (inequality of the two eyes as regards refraction) it is known that one eye is quite unable to accommodate to a different degree from its fellow in order to make up its deficiency. One eye is (unconsciously) selected, either constantly or merely for the time being, and the ciliary muscle of each eye contracts to a degree suitable to the requirements of that eye without any reference to the needs of the other. "Even a slight difference in refraction," says Donders, "we are not able
To adjust by accommodation, so inseparably is the tension of accommodation in one eye associated with that in the other. This fact is an extremely important one to note, says Ladd, and places the statement in italics: "An anisometrope almost always makes the same effort of accommodation on both sides." So that it is quite possible that if cases of unilateral paralysis of accommodation occur, they cause practically no symptoms whatever, because one eye does the whole work till the other recovers. I do not think it at all probable that any of the so-called cases of unilateral paralysis of accommodation occur, quite the contrary, but I say that if they should, they would probably not be diagnosed without great care being taken in testing; that indeed they would be as difficult to verify as those of half a diaphragm. This may explain the invariably (?) bilateral occurrence of the paralysis. One must bear in mind also the closely contiguous positions of their centres in the brain.

It is right to mention, however, that some have observed a greater degree of para-

Gull. Quoted by Greenhow loc. cit. 225-232. See also my case No. 6, p. 65.


Weber. loc. cit.
lysis on one side than on the other, a condition that Buzzard believes to be more rapid than the other.

In the case of the palate, exceptions to this rule of symmetry are certainly not unknown. It is not at all infrequently observed that one side is more paralyzed than the other. In the case of the limbs also, although paralysis is infinitely more common than any other form of palsy, the hemiplegic type has been observed; and not seldom the patient who is not exactly paralyzed but only "mildly paretic," spontaneously complains of greater weakness on one side. Duchenne has seen even hemiplegia, in a case in which the only part paralyzed was one of the lower limbs; and Hermann Weber has seen a somewhat similar condition.

When the external muscles of the eye are affected, which is rare, they are usually affected unsymmetrically; a muscle or nerve of one eye being paralyzed. Symmetrical palsies and symmetrical paralysis.
paralysis of the VI nerves have been observed. Anatomically, paralysis of both External Recti muscles is "symmetrical". Functionally, however, it is not so. The functionally-complementary muscle to the Left External Rectus is not the Right External Rectus, since the two are never used together, but the Right Internal Rectus, which always acts along with the Left External. This, however, is only true so far as lateral movements are concerned, for the Internal Rectus has the duty, not less important, imposed upon it of assisting in the act of Convergence. The combined action of the two Internal Recti in Convergence, which I have observed paralyzed or at least in a paralytic state, as a consequence of Diphtheria, is a true functionally symmetrical action.

10. It used to be stated, I do not know whether anyone still holds the opinion since the publication of the results of post-mortem examinations of cases of Diphtheritic paralysis, that the paralysis must be of a simply dystonic nature, that no organic changes could exist, since spontaneous recovery almost invariably took place. Our present knowledge of nervous diseases
deasures, however, is not sufficient to enable us to state absolutely what lesions may and what cannot be recovered from. Most writers on this subject dismiss the question of prognosis by saying that some cases die from a affection of what they may perhaps be allowed to call "Vital" nerves, nerves, functions, such as those regulating the action of the heart and lungs, nerves, that is, on what functional activity life is dependent. They say, or tacitly leave it to be understood, that all the rest recover; but the facts are not so, for there have been recorded a few rare examples of permanent paralysis resulting from Diphtheria—so we shall see shortly.

Consideration of the subject under these ten headings above, I think, that the paralysis is not simply a result of Diphtheria; that, in short, the third answer to the question proposed on page 36, is the correct one.

The next few pages will be occupied with an account of the cases I have observed.
1. J.M. A girl aged 3 yrs. well grown. While there was much diphtheria in the neighborhood, patient became ill of headache and severe elevated sore throat, which, patient's mother informed me, the doctor in attendance had stated was not diphtheria. The child was only confined to bed a week or two; and about 6 weeks after she was able to rise, patient was sent back to school. It was only then discovered that she could not read.

Pupils of moderate size, not dilated, equal, and mobile to light; convergence good, but pupils do not contract during the effort.

V. R. E. = \( \frac{20}{20} + \frac{1.50}{20} \). Near vision - only very large print. +3.50 gives vol. 1. - futility read at the proper distance for her age.

In this case until 14 days after she came to consult about her eyes, there had been no other paralysis, but palsy of the palate then appeared and, however, to last long. She was quite well again in 3½ months from the time when she was first seen, during the midst of which time she was upon hygienic treatment.

2. M.S.
In the light of Mr. Bouchult's "observations" given on another page, I think it right to say here, that in this case and that of No. 3, it would have been very easy to diagnose retinitis, although none existed; the injected appearance, which at once suggested the condition, being entirely due to Hypermetropia. One had to take a second look to be sure that such was the case. I ought to add that Mr. Bouchult's reputation as an observer is not high.
2. Miss a girl aged 14 years, tall, but not strong-looking. Had an extremely severe attack of diphtheria commencing 5 weeks before I saw her. Eye symptoms were noticed a fortnight before I saw her, at which time also there was slight throat-paralysis. There was no albuminuria. Fundi injected and red.

Pupils were equal, moderate, and movable, though perhaps a little sluggish in their tendency to light and accommodation. Convergence was good.

\[ \frac{20}{40} +1D = \frac{20}{20} \] Near vision. Some degree of farsightedness.

Under tonic treatment, the accommodation-power returned completely in a month, by which time the palatal palsy and the rather staggering gait had much improved. The pupils remained sluggish for some time longer.

3. Miss a girl aged 8 years, pale and delicate-looking. This was a mild case of diphtheria, but throat-paralysis was present, as well as very weak pulse, sometimes disappearing, clammy skin, vomiting, fainting and "gastric symptoms." There never was albuminuria.

Pupils widely dilated, right being rather the
larger.
larger. React very slightly to light, the right remaining always the larger of the two. Convergence is very imperfect and, along with it, the pupils contract only very slightly and imperfectly.

\[ V = \frac{25}{250} + 3.0 = \frac{25}{250} \] Near N. Normal types. +7.0 = 10/11.

Easily up to 6 inches, where the convergence fails and diplopia occurs.

Findings: Leds injected, and the disc is indefinite but I think I believe to be entirely due, as in the last case, entirely to the hypermetropia.

Six weeks later, the child became unable to walk. Pupils react better to light, but were unchanged on convergence.

Four weeks later, under home treatment, the child can walk, though not steadily, swallows perfectly and speaks well. Accommodation and Convergence seem quite restored, pupils acting at the same time, though they are still sluggish to light. Jordan reflex still absent.

4. KS. a girl aged 10, healthy looking.

Two weeks before I saw her, Diphtheria had attacked her, but not very severely. She was confined to bed about a fortnight, during the second
Second half of which and for a week after fluids returned through her nose. When she came home there was no palsy of the palate; she had spoken and swallowed well for more than a week. No other paralyses had occurred besides throat and eye. Tendon reflex present.

Pupils median, mobile to light (left is larger). Convergence, during which movement the pupils do not act, is only possible up to 15 niclæs, within which distance it fails and diplopia occurs.

\[ V = \frac{20}{70} + 10 = \frac{30}{70} \]

Near V, largest of small types +3.5 = 11.

A week later patient was distinctly worse. Has had a slight soreness of the throat. The voice has again become quavering and fluids return through the nose. \[ V = \frac{20}{70} + 15 = \frac{35}{70} \] Koll. Convergence impossible within 20 niclæs.

Patient was ordered Strychnine. Two months later she was able to converge to 6 niclæs and to read half into either eye unaided at 8 niclæs. Made the patient for some weeks use Pilocarpine Solution locally to the eye, but it produced no permanent contraction.
5. G. M. a man, aged 30, employed as a hotel "books." Was ill of "altered sore throat" three months ago, which he believes he caught by a cold. Was ill, but not confined to bed, for 3 weeks. The voice at that time and for 6 weeks after was quite lost; the patient could only speak in a whisper. He never had any difficulty of swallowing, no diplopia nor any other paralysis than that of accommodation. There had been no diphtheria among the servants or visitors in the hotel as far as he knew. A doctor who happened to be in the hotel gave him the diagnosis above stated and recommended a gargle which he used. He had had diphtheria 5 years ago. Pupils moderate, left rather the larger, sluggish tonight, but act freely when conjugated, which is perfect.

V = 30 R. 42° + 10 = 30. H. 45 V. 5°/5 is 2. 3-5 D. near

V 41 at 6 inches. Hypermetropia estimated by direct examination at 1.5 D. The "near point" for a hypermetrope of 41/5 D. at the age 2.
of 30 is 6 inches. (For an emmetropia of the same age it is 3½ inches). He required a lens of +3.5-D to bring him up to this standard; his accommodative power was therefore to this extent defective. The full range of accommodation at this age being equal to 7D; he possesses at present only half that amount. He says he is not getting better. He was ordered an internist solution, and I instilled a drop of scopoline into one eye which acted well upon both pupill and ciliary muscles. As he continued to progress only very slowly I ordered him to use a drop of pilocarpine to one eye daily. This enabled him to see well to read for some hours at a time, but that eye did not appear to recover anymore rapidly than the other. At the end of 3 months he was only slightly better.


6 weeks previously this child had "mumps." She had felt only a little sore throat below the swellings caused by the congested parotids. She was confined to bed for three weeks and returned.
returned to school a fortnight later. When she went there it was found she could still see. Distant vision was said to be "as good as ever." There was a little doubt about whether any scar was of the palate existed or not.

Pupils are equal, of normal size and mobile alike to light and to convergence which is good.

\[
V = \frac{20}{70} \text{ (compare patient's own statement above)} + 1.5 = \frac{20}{30}. \text{ Near V, normal letters +5.5 = 100/10.}
\]

But not perfectly. Edge of the disc is perhaps not quite well defined, Ordered Sininen and a generous diet.

A fortnight afterwards patient returned having had regurgitation of fluids through the nose since her first visit. Right arm was thought to be distinctly weaker than the left, a fact of which the patient spontaneously complained. Ten weeks later she was quite well.

7. A.W. a girl aged 9. Had had right-shoulder a severe sore throat from which she had suffered a month previously. \[ V = \frac{20}{70} + 20 = \frac{30}{20}. \]

Dr. McBrude, having found a quantity of fat over

[Signature]
growth in the pharynx and of very marked
fistula tissue in the nose, removed these
growths, and healed the nasal condition.

At the time when she had her loose tooth
none of the other children in the house, of
whom there were several, had any such disease,
and no doctor saw her. Four weeks later
the caries was much less and a fortnight-
afterwards had entirely recovered.

NB. This case did not occur to myself, but have
the kind permission of Dr Berry under whose
care the girl was, to refer to it.

8. A. M. a boy of 11. pale, bronchitic and asthmatic.

Six or seven weeks ago he was ill, his
mother being severely ill, of a bad sore throat,
which attacked several members of the
family at the same time. No doctor was
summoned to attend him.

Pupils moderate and mobile both to light
and to convergence. Equal in size.

\[ V = \frac{20}{70}, \quad +1 = \frac{20}{70} \]

Near vision +5. Dreads not.

This case I saw for the first time only a few
days ago, so that there is no progress to report.
Also Lucet of 9th March 1857.
Of these eight cases then, there is little room for doubt that six were certainly of diphtheritic origin, notwithstanding the negative diagnosis given in case No. 7. In case No. 6, a diagnosis of diphtheria was given, but when the patient returned the second time she told us that the doctor had come to the conclusion that her illness had been diphtheria, presumably as a consequence of his having observed some other cases arising at the same time.

In regard to case No. 5, however, there is a little more difficulty. G. M. had an acute ulcerated sore throat, but he had been subject to sore throat for some time after his infection with syphilis, and was quite confident as to having been exposed to cold just before this attack came on, when rising from bed to open the hotel door to a late visitor on a damp, cold, winter night. There is reason to believe, on the other hand, that a truly diphtheritic sore throat may be set up by exposure to cold, see Graniger Stewart's case of an engine driver who acquired diphtheria apparently in this way. The very slow progress...
From the patient's description of his own voice during the acute stage of the disease, the peculiarity does not seem to have been due to paralysis of the palate. See page 77.
progress towards recovery on the part of this patient is also to be noted. This may have been due to the greater age of the patient than is usual in diphtheria. In Graniger Stewart's certainly diphtheritic case of the Eugnie driver just referred to, the paralysis was also more prolonged than is usual. It may be that this matter of the age of the patient is a point to be remembered in giving a prognosis. Cases in which a person affected with diphtheria is able to go about and do his work cannot, I think, be very frequent, though there is no doubt they do occur. If this were a case of diphtheria, then, it had a certainly unusual course. Further, it was, so far as can be made out, apoplectic, and it was followed by a very paralytic history. There have not been very many cases recorded of paralysis coming the palate and attacking other parts, though they have certainly been seen. A concatenation of unusual occurrences naturally makes one suspicious of the truly diphtheritic nature of the disease, but what else can it have been? The paralysis
Of accommodation can hardly have been in any way due to syphilis, I think; it is possible of course that it might be the first indication of syphilitic neuropathy, as such as Hutchinson describes, and which he considers to be always or almost always syphilitic; but for several reasons into which there is not necessary now to enter, I don't think it was. The patient, too, was quite sure that until the throat affection had come and gone, his sight was perfect. Any other form of throat affection producing paralysis or paresis of accommodation, if it ever occurs, which I doubt, is certainly even more unlikely than the diphtheritic; so I think we are bound to conclude that it was of this nature.

In the case also of No. 7, we are left in doubt of the truly diphtheritic nature of the acute throat affection.

These cases open up the question, Can paralysis follow other throat diseases? How, here we must distinguish between two very different things, (1) paralysis of the palate alone, and (2) paralysis of other

Marigny l'oe. eit.

Jacobi l'oe eit.
requires including or not including the palate. That temporary paralysis of the palate may follow an acute inflammation of it is quite easy to be understood. Irons and Lacèque, in their first paper upon this subject, issued before the general occurrence of paralysis as a sequel of diphtheria had been noted, ascribed the palsy of the palate simply to the effect of the plastic inflammation, and Manigault at first considered this state to be analogous to the paralysis of the bladder and intestine, in consequence of pyelitis and enteritis. Jacobi believes that it is so, and says that "in some cases the local paralysis, particularly when it begins early depends first on the edematous infiltration of the whole tissue, and further on a direct implication of the nerves, or on the compression by the inflammatory products." It may be that such is the case, and that, when palsy of the palate occurs during or immediately after the ulceration, it is produced in this way. It will not however explain the later occurrence. Duchenne entertained no doubt that other throat discases might produce paralysis of
Trousseau. loc. cit. II. 563.
the palate. He says, "I have seen it follow a simple pharyngitis." Frohseur related a case of paralysis of the palate (and other parts) following a sore throat diagnosed by Gubler as "one of common membranous sore throat—glandular herpes." Commenting on this case and others which he gathered from authors of eminence, he says we are entitled to ask: "whether sore throats of apparently the most simple character may not give rise to paralysis of the veil of the palate.... Do not these cases of apparently simple sore throat originate in the same cause as severe Diphtheria, especially when they occur during diphtheritic epidemics? If it be so, we can quite understand how paralysis of affective may supervene after a simple just as after diphtheritic sore throat. I do not wish you, however, to believe that simple sore throats never bring in their train paralysis identical with that which occurs as a sequel of Diphtheria. Facts observed by able clinical physicians show that irrespective of the epidemic inflammatory influence of Diphtheria, simple sore throats may be the starting point of that peculiar form of general paralysis.... But while I admit this, I wish to state most positively, that though it is very common to
met with paralysis as a sequel of diphtheria,
it is exceedingly rare to see it following a simple sore throat:"

General paralysis following a simple sore throat I do not see any way to accept as a fact. Those who believe in the occasional occurrence of this, weaken their argument very much by admitting that some of these occur during diphtheria epidemics, as will be seen from the quotation just made from Homnan. The fact is, I think few will deny it, that cases of mild sore throat occur during a diphtheria epidemic which, if seen at another time, would be thought of little if, but which are in reality (abnormally?) mild cases of diphtheria; and if seen during a diphtheria epidemic, why not at other times, since the disease is by no means purely epidemic?

Remember too, the thousands of cases of pharyngitis tonsillitis, etc., which are never followed by any symptoms, and you will see that if paralysis be a consequence of simple throat, inflammation it is a vastly more rare one than even it is of erysipeloid fever, which very seldom had this sequel.

Case No. 6. presents an interest of a slightly different type. When the patient was...

Dörfy. Progrès Médicale 1886. 47.

Towers. loc. cit. II. 525.
first seen, it was believed that she had been suffering merely from an attack of Mumps; Diphtheria had not been suspected. Now paralysis of accommodation following Mumps has been recorded by Boas, and paralysis of the larynx with loss of Electrical irritability, and a reflex action by Jeffrey. Commenting on these two cases, Governors says "It is possible however that in each of these cases there may have been undiscovered Diphtheria; in the second case indeed slight angina was present." Here then is a case in point. The girl, who was believed to have had Mumps, returned stating that the doctor now believed she had had Diphtheria. This case illustrates how easy it is in a matter such as this, to go astray. The swollen glands being the prominent symptom, the case had been put down as Mumps by a skilful surgeon in large practice; it suggests forcibly that many cases in which diphtheria is a prominent symptom may be ascribed to Mumps, and many in which the larynx is marked, and particularly if the redolent rash described by various authors as occasionally to be seen, is obtained, may be considered to be Scarlet Fever.
Jacobi, Towers, Lade, Drousseau, Mang, &c. loc. cit.
St. Peter Sade. Notes on Diphtheria.
What then is the essential nature of post-diphtheritic paralysis of various regions?

The seat of infection of Diphtheria being the throat, the most common consecutive paralysis being also situated there, and that being naturally the first place traced to Diphtheria as its cause, a local origin was of course the explanation which first presented itself. Most authors agree that the palatal paralysis is the most frequent; a few, however, consider the ciliary nerves to be that most frequently affected. Marmar's table shows an immense preponderance of the palate (70) over the other (39).

Jacobi, Gowers, Kemeyer, Ede, Imonsen, Marx, Schumacher and others of the best authorities all agree to this. Still, though I do not wish to cause doubt on what they say in this matter, it is but right to remember how obscure the one paralysis is, and how obscure the other, how easily passed over. I think we may take it for granted that few or no cases of palatal paralysis are overlooked, the symptoms are so definite and striking as always to attract attention. The return of fluids through their nose, the swellings, puckered voice,


Schroty-Buch, loc. cit.
voice, are "positive" symptoms which at once cause the alarm of friends; but such is not the case with paralysis of accommodation. The patient's inability to read is referred simply to weakness, particularly if, as is often the case, the paralysis is only partial, and he is able by training to make out a few lines at a time; or it may never be noticed at all during the time that the patient is supposed to be ill. Cases 11 and 6 illustrate this fact. Nothing was noticed to be the matter with their sight until the girls were sent back to school some weeks after the throat affection, and when I inquire no doubt the condition had existed for some time. I refer on another page (28) to the fact that in the case of Mrs. Jones the condition may never be discovered.

Buhl says that paralysis of accommodation is the commonest form of post-diphtheritic paralysis. Entrace Smith that it is as frequent as that of the palate. Selby-Brock is quite sure that affection of the accommodation is commoner than one is disposed to think, and that many cases...
cases if it either do not come for examination or are overlooked.

Granting then, as I am quite willing to do, that palatal paralysis is the common form, though the disproportion between it and that of the accommodation is perhaps less than is generally supposed, will a cause confined to the spreading from the seat of the original infection suffice for explanation? I think not. If the palate were always affected by paralysis (when paralysis of any region followed diphtheria) and were always first affected; if the palsy spread from there to various parts of the body in regular sequence, this theory would be more tenable; and if, when traumatic diphtheria occurred on a limit, let us say, paralysis spread from that limit to various other parts of the body after a similar fashion, that would be an additional argument and a powerful one. Such, however, is not the case. Where paralysis follows throat diphtheria, the palate is not always affected at all (see case 5. J. M.) and even when it is paralyzed, may not become
All three are mentioned by Schelby-Buch.

Jacobi: loc. cit.
became so tells after other parts of the body have been attacked (see Cases 1 and 26). Out of 22 of Scheel's cases of paralysis of accommodation, paralysis of the palate was absent in 10. Dicke, Paget, Hunter, and Bartels have also seen similar cases.

The fact that some patients die of paralysis of vital nerves, while others have never the slightest affection of these organs; that some are affected with paralysis of the upper and some of the lower extremities; that some have paralysis limited to sensory nerves (Duhamel) or almost limited to them, while others have only their bowel functions interfered with—all combine to make me agree with James that it is characteristic for the paralytic phenomena to follow a certain order of "unjustifiable." I have abundant evidence that there are many cases in which precisely the contrary holds true, that is, that it is characteristic of diphtheritic paralyses that they follow no certain course, passing by certain parts of the body and attacking others.

The same is true in cases of hemorrhagic diphtheria.
Marecot and Vulpian. Comptes rendus de la Soc. de Biol. 1862.
There are five situations in each of which the lesion has been supposed by some to be situated:—The muscles themselves, the nerve terminations, the nerve trunks, the sympathetic system, and the brain and cord. Excluding for the present the theory that the symptoms are produced simply by passing apart altogether from any necessary demonstrable lesion.

1. The Muscles. A favour of this theory is the mentioned fact that undoubtedly the degenerative changes have been found in the muscles, principally of the palate, but also of other parts of the body, such as the heart (Millen). These were first observed by Lacassot and Vulpian in 1832. There are, however, certain difficulties in the way of adopting the muscle theory. These are:

a) The degenerative changes suggest rather an atrophy secondary to disease of the nerves than a primary muscle change (Gowers).

b) The fact that the muscles react to other stimuli, electrical and therapeutic. Thus the dilatory muscle responds to Berinn, which is believed to act directly upon the muscle elements.
Iesop. Transactions of Royal Society of London. 1886.
Bericht über die VIII. Ophthalmolog. Congress 1888.

Leesez. Die Pupillarbewegung. p.70
This is certainly a very important fact, and
is rendered more so by the knowledge that
the paralysed muscle reacts as powerfully
to Eserin as a normal one does. [Jcossop
days that Eserin acts on the muscular tissue
of the sphincter pupillae, and that it probably
acts in the same way as on the
pupillary muscle. Lecser, however, following
Schoeler, believes that it acts on the endorgans
of the III. nerve.]

1. The frequent occurrence of sensory paralyses.
   This, he it remembered, is not an exceptional
   occurrence, but being less obvious than the motor
   is frequently left undetected. Duhamel indeed
   says that he has seen sensory paralysis occur
   without motor, though more frequently with it.
   d) The difficulty of accounting for the uncertain,
      wandering, paralyses from various parts of the body
      on the sole ground of this theory.
   e) The fact that the part affected with the
      original disease is not always attacked by
      paralyses, and is sometimes not paralysed
      until after some other part or parts of the body.

2. The nerve-dominatian. I do not think
   that there are any very definite facts pointing
Pathological Attuatures of Nerve fibres in the muscles have been seen by several observers.

Lauder Brunton. Materia Medica. 222.
to this situation, and the nerve endings have been supposed to be affected simply because there was no visible lesion, in some cases, anywhere else. But certain of the objections to the muscle theory apply to this also, e.g., for example, if also, though to a less degree, i.e. Again, pilocarpine, in dose too great at any rate, causes the ciliary muscles to contract, and pilocarpine acts upon the nerve endings (Sander-Brunton). There is a danger however in accepting such an argument as this; for it is easy to see that an organ, such as the end-organ of a motor nerve, may be affected by the unusual, abnormal stimulus of a drug, when it is in a state of insability to appreciate the normal stimulus conveyed to it by the motor nerve from its centre in brain or cord. I have observed that in many works on subjects such as this it is taken for granted that if an organ is to be in a state of irritability to an abnormal stimulus, it must therefore be irritable to a normal one—a position which is quite unwarrantable.

3. The Nerve Trunks. Degenerative changes have been seen in the nerve trunks from anterior.
Gowers. loc. cit. II. 837.


Bizzard. loc. cit. 118.
Anterior root to motor division, proportionate to the degree as well as to the extent of the paralysis. The nerve sheath undergoes an increase in the number of nuclei, the axis cylinders become broken up, and even disappear here and there. Such were the changes first seen by Charcot and Balguere in 1862 in the palatine nerves. Buhl, in a case in which, however, there was no clinical history of paralysis, found the nerve sheath infiltrated with small nuclear bodies. Greenland was one of the first to write in favour of a peripheral neuritis, and Bland-Sutton is strongly of the same opinion. He says: Moreover, the sensory disturbance which is so often present in diphtheritic paralysis, cutaneous anaesthesia, tenderness of muscle and nerve-tendons, and darting pains, cannot possibly depend upon lesion of the ganglion cells of the cord, whilst they point so strongly as possible to affection of nerve fibres. He considers that diphtheritic closely resembles alcoholic paralysis, only that in the latter disease is more severe and the sensory symptoms are more marked, but he has seen cases of diphtheritic paralysis.
The frequent occurrence of sensory paralysis is also distinctly in favour of this theory.

Aitken. Diseases of the Nervous System. 1877.

Benson. Transactions of Ophthalmological Society.
with no affection of palate and accommodation.

with "dropped" hands and feet, severe lancinating
pain, tenderness of muscles, more or less power-
lessness of upper and lower extremities, absent
tendon reflex, cases only distinguishable from
Alcoholic paralysis by the absence of any
mental disorder. He relates, too, the case of a
lady aged 22, whom he saw seven months
after being affected with a moderate Diphtheria,
and who when he saw her had atrophy of the
right half of the tongue, weak action of the lips,
probably weakness of the right external palpebral
muscle, and a mobile palate. He thinks this
limitation distinctly in favour of a neural
rather than a central lesion.

Levy and Althaus hold to the theory of a
"migrating neuritis," as the latter calls it. But as
Benson points out in his valuable paper, if this
were the cause, one only out of many muscles
supplied by a nerve could hardly become
paralysed, as occasionally happens, particularly
in the case of the eye. Prism for example is
not very uncommon. Now, how could prisms
- the levator palpebrae superioris being only one of
several muscles supplied by the III nerve - if the

Cause
cause were a neuritis of that nerve? He adds: 'how is it the short root of the ciliary ganglion which is affected [in paralysis of accommodation] for the pupil continues to act to light.' It must be noted however that in one or two of my cases the light reflex could hardly be obtained.

A much more serious objection is one which is well brought out by several of my cases, viz.: (1) the occurrence of paralysis of convergence-myopia, that is, during an effort at accommodation and convergence, the pupil remains stationary though it reacts well to light. (2) The occurrence of a modified degree of the converse of this, viz.: paralytic of the light reaction, while the convergence-myopia takes place with ease. (3) The occurrence of paralysis of convergence while the internal root retains full power. No neuritis can possibly account for any of these conditions. It is impossible that a neuritis which prevents convergence-myopia should leave intact the mobility of the pupil to light. It is impossible that a neuritis which prevents light reaction should leave intact the convergence-myopia, unless it might be a retrobulbar or other
Hughlings Jackson. British Medical Journal 1877. 577. Lecture
form of neuritis of the optic nerve interfering with the conduct of light, which is in the highest degree improbable and for which there is not the slightest evidence. It is impossible in the third place that a neuritis should prevent the two internal recti muscles from acting together and yet allow them full power to act separately, each along with the external rectus of the opposite side, as was the case in Cases 3 and 4, in which each internal rectus retained full power.

4. The Sympathetic System. This theory arose chiefly from the misunderstanding of a remark made by Hughlings Jackson in his lecture on Ophthalmology in relation to General Medicine. He said: "The peculiarity of diplophthric neuritis is that the paralysis is of parts supplied through a ganglion of the sympathetic chain. There is not paralysis or paresis of the III. nerve but of parts of it which are supplied through the ciliary ganglion. In other regions we see the same thing. The defective articulation is owing to paralysis of muscles of the palate supplied through Meckel's and the otic ganglia. The very slow pulse which, as Jenner and Francia..."
Hutchinson. Transactions of Medico-Chirurgical Society. 1879.
have pointed out, is found in some cases, may be explained by affection of the cervical ganglia of the sympathetic. Let anyone should mistake his meaning, for such an origin, because paralysis occurs in regions such as the limbs which, as regards their movements, are quite out of sympathetic control. There are various other objections to the theory which, however, has some able upholders. Some of these objections are, the occurrence of sensory symptoms and the absence of vasomotor or other essentially sympathetic symptoms. If Hutchinson is right in referring his cases of phthisical ophthalmo-scleritis to the central ganglion, (whatever he is), the difference in regard to the pupil between such cases
D'Herpin. Archives de Physiologie norm. et pathol. 1878.
cases and those in which paralysis of accommodation is the result of Diphtheria is very striking. In all Hutchinson's cases, there was lido plegia, which is rare in Diphtheritic cases. (see page 109.)

5. The Brain and Spinal Cord. At present, the region considered by the majority of writers to be the most probable seat of the disease in the central nervous system, but the exact nature of the lesion is not at all agreed upon.

Buhl, in the case I have previously mentioned, found extravasations of blood in almost all the organs of the body, including the pia mater and cerebral cortex, in the central ganglia, crus cerebri, pons, medulla oblongata, and cord. The account of what he discovered is interesting, but in the absence of a clinical history of paralysis cannot be considered of any great value in clearing up the question of pathology. Valpiani, Loret, and Dejerine divide the honour of having earliest described changes in the situation where one would naturally expect to find them (if one believes in a demonstrable central lesion) 197: in the anterior cornua of grey matter of the cord. Loret discovered multiplication of
Hidol. Transactions of the Medico-Chirurgical Society of London 1883.
the nuclei in all parts of the cord, but particularly in the anterior horns. Dejerine found some of the nerve cells of the cornua swollen and others shrivelled, and considered that they were fewer in number than they ought to have been. The neuropil also showed "signs of irritation." Dr. Abercrombie read a paper before the International Medical Congress of 1871, in which he stated that he had seen similar changes. He had observed no haemorrhage.

Dr. Percy Pidd, who gives a useful résumé of former observations, produces also the results of a very careful post-mortem examination made by him on the body of a boy aged 13 who died paralysed and with severe bronchitis two months after an attack of diphtheria. In the anterior cornua of the dorsal region of the cord he found the large nerve cells atrophied, some of them had lost their processes, had become globular, and were pale and vacuolated or granular. He never found them swollen. He considers that his results and Abercrombie's substantially agree, the differences between them being due to the difference in the duration of the condition. Abercrombie's
Bußgeld. loc. cit., 116.
cases were rapidly fatal, apparently indicating lesion.

but the change which first occurs may be a
swelling up of the follicles, followed by a later
stage of shrinking and degeneration. Field
concluded in his case to obtain permission to
extend the examination to the trunks,

so there is not one of the condition of those
structures. Burghard says that Mendel

conducted a post mortem examination of a
case. Fatal ten days after the commencement

of paralytic symptoms. There had been

paralysis of limbs and of several sphincters.

Under the microscope the ganglion cells of the
dep over of the oculomotor nerve were

found to be large and to appear swollen. In

the nerve trunks he saw interstitial and

parenchymatous necrosis, and small haemorrhages

in parts of the intracranial centres. Discussing

the pathological alterations found in this
case, Burghard says he thinks that the nerves

were affected first, the central changes

being secondary. Mendel concludes, and

Burghard thinks he does so with justice, that

the "Diphtheritic poison may attack not only

nerve sheaths and neuromyema, but also..."
Greenhow. loc. cit.

Quoted by Percy Hiddle.
The walls of blood-vessels and that we have no
right to assert that affection of one or the
other of these tissues represents exclusively
the pathology of diphtheritic paralysis. But
it seems to me that one should be suspicious of
the truly primary character of a neuritis,
and the truly secondary character of a central
change which are accompanied by haemorrhage
into the nerve centres. If the vessel walls
are also diseased as a result of the diphtheria,
good and well, but if not, one cannot well
dee how peripheral neuritis can produce
central haemorrhages; and I am aware from
the plan of requiring to accept two theories
combined where one simple one will suffice.

Greenhow has frequently found haemorrhage
spreading into various tissues, but at any
tale in most of his cases the urine was
albuminuric, so that it is right to receive this
statement with a reservation.

It is proper also to say that H. Weber,
and Perret found no changes whatever,
and in some of their cases Bertet and Vulpian
were unable to discover anything abnormal.

A difficulty meets us here. Can such
changes
Another argument for the central character of the lesion occurs to me. I am not aware that anyone has yet used it. It is that at or about the time of the occurrence of the paralysis, the mental attitude of the patient changes in some cases. In one case of Paroxysms, though he made no use of this observation, it is stated that the change which his friends observed was that of certain patient was a change in his character. Full of life and cheerful and vociferous, he had, after it became unpatient and difficult to manage.
changes as have been described as occurring in the nerve centres in fatal cases, be present in the cases which survive? Is the statement correct which is made in Dr. Poire's translation of Duchenne's Works, that it is probable that the repair of motor cells once lost is impossible?

If so, then one of three conditions is correct: either the fatal cases differ fundamentally in pathologista from the non-fatal, which is in the highest degree unlikely; or the very marked changes which take place and which certain appearances destructive, are not really so, which is not improbable; or, as I think, these central and neuritic changes are not perhaps necessary to the pathology of the paralysis.

If paralysis spreads from one part to another, beginning at the throat, by what path could it spread? Could some influence inducing paralysis spread from part to part of the body travel by way of the lymphatic system? Beyond the fact that the neighbouring lymphatic glands to the ulcerated portion are inflamed and swollen, I know of nothing which
Which would indicate this, and no one has ever suggested it. When first general paralysis was known to occur after diphtheria, some evil influence was considered, spread along the nerves. In the case of diphtheritic paralysis, which Ebelt showed to the Medical Society of Berlin, he ascribed the simultaneous affection of the palate and the eye to the circumstance that "the nerves of the palate, which arise from the sphenopalatine ganglion, are connected with the optic nerve by some fibres which proceed from the ganglion just named, through the fissa divisa inferior to the optic nerve." What is, or might be thought to be, of much more importance is that Telemann dissected out nerve fibres connecting the sphenopalatine with the ophthalmic ganglion; but as we shall see, there is good reason to believe that the ophthalmic ganglion has nothing to do with the condition, and most certainly the optic nerve has not. Of course, the cause of Dr. Ebelt's papers, the cause of the "Amans's disease" was not fully understood. In a similar way the first few cases were explained, but...
but when more were recorded, it was found impossible to apply the theory in its original form. Think what a long journey the neuritis (if it were a neuritis; the evil influence, at any rate) would have to perform if it spread round to the 11 nerve in one case, to the cardiac nerves in another, to the lower extremities in a third. Such journeys are performed by peripheral neuritis under other conditions, but what I mean to point out here only is, that if there be no general or centrally-acting cause, it is unreasonable to think that neuritis will spread in this way over the whole body, omitting large tracts and only "dropping out" here and there.

The most probable means by which the pain is spread is not the (organic) one of the nerves, but that of the circulation. It is probable that the "evil influence" is absorbed, either from the ulcer, or directly from the atmosphere without the occurrence of any ulcer (see Boisrobert's cases, page 125), and thus affects the system generally, through the medium, as I believe, of the central nerves.
A vaso-motor change might be suggested as a theory, but the suggestion is too indefinite to be useful, and the next question would be, "What causes the vaso-motor change? A poison?"
Rerum exempli.

Here, then, we are in this position. Persons die from or during paralysis consequent on Diphtheria, and in the nervous and muscular tissues there may be found (1) Nothing, (2) Degeneration of Muscle, (3) Degeneration or inflammation of Nerves, (4) Haemorrhages in the Cord, etc., and (5) Cellular changes in the nerve-cells in these regions.

This state of things suggests to my mind the existence of some form of poison generated by or dependent on Diphtheria and acting on the nervous system as the cause of the paralysis. From this, when once satisfied that general paralysis was dependent on Diphtheria, at first endeavoured to explain this circumstance as being in some way connected with the occurrence of albuminuria, a condition which he had found in certain cases, and which was first demonstrated by W. of Birmingham. It was pointed out, however, (1) that only in a small proportion of the paralysis cases is there or has there been any albuminuria, (2) that the nervous symptoms which are produced by albuminuria, are rather by that

state
Jonnelli: mentioned by Farre. Cruceet 2. 1862. 583
state of the kidneys which produces albuminuria, are of a totally different character. The symptoms and the form of defective vision in the two cases are entirely different. The indistinctness of vision sometimes occurring in Bright's disease is produced by one or both of two causes which are quite dissimilar viz. Retinitis, neuritis or neuroretinitis on the one hand, and trauma, without any fundal changes, on the other; neither has any analogy to the condition present in Diphtheria. Need not go further into this subject, the differences are obvious. - Tomsenian, when he recognised that the albuminuria theory was not able to account for the conditions, suggested the poison theory which I have referred, and which, in a modified form, he considers to be the correct explanation of the symptoms. M. Tomaselli of Sicily also in the year 1862, expressed a similar opinion to Tomsenian. Two important points must be noted:

1. Spontaneous Recovery is the almost invariable rule. Although it is quite true that in some, perhaps in nearly all cases of post-mortem examinations, degenerative changes
have been found in some part of the nervous system; and although it is also quite true that we do not know from what degree of degeneration it is possible for tissues to recover, one may be able here to draw a distinction between a primary alteration of structure in the nervous tissues, and the disorganization produced by the temporary action of a poison. Is it not more probable that the latter may be entirely, entirely removed, uncovered from than the former? Kihl's observations led him to the conclusion that the changes in the motor areas were to all intents and purposes indistinguishable from those of poliomyelitis anterior acute. Now we cannot remember the fact that an attack of poliomyelitis is followed, I suppose invariably, by rapid atrophy of some of the muscles, and permanent deprivation of function of a limb, or group of muscles at least, whereas such a result is infinitely rare in Diphtheria. It is on the other hand noteworthy, that a widespread infantile paralysis generally becomes localized to one limb, or one group
Peterson, loc. cit.
of himself, so that of nerve cells which
are presumably affected in the same
way, though not to the same degree, some
become permanently paralysed while
some entirely or partially recover. Still
receiving the matter as a whole, I feel bound
to reject the necessary occurrence of such
decisions as Dr. Kidd has seen in the cord,
and Mendel in the similar cells of the
region of deep origin of the III nerve. I
think also that the small-haemorrhage
theory which Benson favours, comes under
the same condemnation. It seems unfairest
that in either of these cases, the vast majority of
all the persons affected should entirely recover.

On a superficial glance at the subject
one might say: yes, but the cases which are
available for examination must be the
worst cases of paralysis, for they die. But
this would be quite an unfair and untrue
contention. These cases die because the
paralysis has "happened" to affect vital
nerves, while other cases do not die in which
the paralysis, though quite as severe, and
sometimes affecting organic reflexes, has
avoided
such as St. Helen. Vechos' Archiv. xviii
Avoided the cardiac and respiratory nerves, or affected them to a less degree.

It is rather suggestive that Brousson and others mention quite inadvertently, if one may use the expression, a peculiarity of diphthetic paralysis which is also a peculiarity of symptoms of poisoning, viz. the great variability from time to time of the patient's condition. A limb may be almost paralyzed one day, much better the next and the day following, and as bad as ever two days after. In an exactly similar way patients often alternately rally and sink under the effects of a poison.

2. Analogies exist.

a) The symptoms produced by lead poisoning appear to have been neglected as an analogous condition. Lead has among other affinities a tendency to produce paralysis of certain groups of muscles, - the extensors. It is not yet a settled point how this action is produced, but some good authorities believe the poisoning may take place in the central nervous system. I am quite aware that by many lead paralysis is considered to be merely a form
Schub- Bueh. Loc. cit.
of peripheral neuritis; but there are certain facts about it which may lead one to believe that it is something more than that. If it be so, that iscombe the more in favour of the resemblance.

1. Cases of poisoning by putrid meat, stale fish, and some forms of poisonous fishes occasionally happen. In some of these cases paralysis of various organs has been observed to remain for some months. Schelley Brown cites five cases of paralysis of accommodation following "Würstvergiftung" without any other paralysis which recovered in from seventeen days to seven weeks.

c) Paralysis of accommodation has also been noticed in certain parasitic diseases, particularly in trichiniasis, although the paralysis is never found in unstriated muscle. The same condition is seen in a few cases also of Diabetes Mellitus, in which disease also the "tendon reflex" is lost.

These analogous conditions are, however, not very strong arguments. Still they do indicate that there follow the introduction
Handfield Jones. Semleian Lecture on Some points in the Pathology of nervous diseases. Medical times and gazette 2, 1825.

Edinburgh journal of Medical Science, 1826. Hamilton.
of certain persons into the system, paralyses which select favourite seats and neglect others.

Handfield Jones is of opinion that Diphtheritic paralysis affords one of the best examples of a true primary paralytic. Hence the word primary to indicate that "the affected part is that which is first acted on by some modifying cause, whether that be a poison in the blood, or some insensible influence, or whether it act in a negative way inducing simple exhaustion." No whole history seems to me to demonstrate that it is not dependent on any organic lesion, but on some depressing influence.

I found in the Edinburgh Journal of Medical Science of date 1826, an interesting paper by Dr. James Hamilton Jr., Professor of Medicine, Midwifery, and the Diseases of Women and Children in this University, entitled "Observations on a peculiar modification of St. Vitus' which occasionally affects Children." After describing some cases in which death occurs by distress of breathing, without either difficulty or wheezing — with sudden sinking of the living powers, in cases which, up till the appearance of this symptom, looked very favorably.
Eade. Notes on Diphtheria. Lancet 2. 1887. 56

Bretomeraud. Traité de la Diphtherite.

the author proceeds to discuss the manner in which this may arise. He uses the analogy of diarrhoea occurring suddenly in cases of suppuration of the hip joint, and shows that neither the explanation of a direct communication between the cavity and the bowel, nor that of direct absorption of pus into the blood, can explain it, and adds, "so it was more reasonable to suppose that there is a very partial absorption of the matter of the abscess, which excited, through the medium of the nerves, a diseased action on the surface of the intestinal canal." Referring again to the throat, he says, "The explanation which occurs to the author is, that the matter secreted by the ulcer being evidently of the nature of a morbid poison, may act by paralysing or otherwise influencing the par vagum."

Sir Peter Cole considers the paralysis due to the presence in the blood of a morbid poison whose special affinity is for the nervous tissues.

Bretonneau, and more recently Gowers, have compared Diphtheria and Syphilis. The latter considers them analogous in their actions on the nervous system by means of an organic virus. We may note also that, the poison of Diphtheria sometimes
Weber. Virchows Archiv für pathologische Anatomie; XXVIII. 123.
sometimes so closely resemble those of the
person of Syphilis, that cases of Diptheritic
paralysis have been repeatedly mistaken
for tubercle, and we have seen that Syphilis
has, like Diptheria, a special tendency to
arrange an intracranial lesion. He
considers that the resemblance is not destroyed
by the great difference in time of action of
the two diseases on the nervous system, since
the primary maladies are so unequal in
duration. But there is possibly even more
than an analogy between the effects of the
two diseases. I have seen a few cases in
which there was evidence of a persistent and
even a progressive lesion of the spinal cord
after Diptheria, and lately saw a woman
in whom true primary atrophy of the optic
nerves with partial Ophthalmoplegia was a
distinct sequel to a true Diptheritic paralysis.
Hence we may hope that the discovery of the
mechanism by which Diptheria affects the
nervous system will take us a step nearer to the
elucidation of the mysterious sequelae of Syphilis.
Hermann Weber regards Diptheritic
paralysis as somewhat analogous to the nervous
symptoms.
Aphasie arising from traumatic tetanus.

Der traumatische Tetanus und die diphtheritische
Nervenstörungen haben mit einander das gemein,
dass sie genossen nicht stets gleicher Zeitraum
zwischen dem Auftreten Anfang der peripherischen
Verletzung oder Veränderung und der Auftreten der
zentralen Störung liegt; dass ferner ein nicht alle
sondern nur selten Fälle von Verwundungen zu
Tetanus führen, so auch nur auf einzelne Fälle
die fraglichen Nervenstörungen folgen; und dritten
das wie die kleinsten Wunden Tetanus erzeugen
können, so auch die leichtesten Fälle von Diphtherie
ihre eigentümlichen Nervenstörungen.

They do not have these characters in common.

But I am at a loss to perceive the importance
of the fact, the affection of the nervous system
in the one case being so entirely different from
what it is in the other.

Another theory yet is that of Steffen, who
suggested that Diphtheria may be allied to
Herpes Zoster, the seat of origin in each case
being the Medulla, and each having a favorite
external seat of election. I do not know if
anyone agrees with him in this, and as
we have been, it is not necessary that the

Throat
According to Fedorovitch it was Guénée de Mussey and Barthéz who first observed paralysis after wound diphtheria in 1857. Two years later Desjardins described the first case of paralysis of accommodation following wound diphtheria. Barthéz has seen it follow diphtheria of the vagina consequent on gonorrhoea. Diphtheria of wounds being practically unknown in this country I have had no opportunity of personally studying the condition.
Throat be affected, since paralysis may follow Diphtheria of wounds or ecchymosis in any part of the body.

I regard it then as highly probable that the paralysis is due to poisoning, and that the visible lesions found post mortem are non-essential; they are merely a probably temporary result of the poisoning, and cannot be considered as essential, since they are so frequently entirely absent, not being found even by those who were very anxious to discover them, and who, having seen such lesions at other times and in other cases, could probably search very carefully. In lead poisoning, to which I have referred, changes in nerve trunks, muscles, have been found, and so in Diphtheria, but on consideration of the points which I have brought forward in reference to pupil movements and the action of the internal recti muscles, it seems to be incontrovertible that the poison acts on the cerebral centres, whether it may produce cortical neuritis in various parts or no.

A point which should not be omitted.
de Watterville. quoted by Buzzaud. loc. cit.

Jacobi. loc. cit. 98.
to the suggestion made by de Watteville, that peripheral neuritis may be secondary to a functional affection of the nerve centres in the cord. Unless this be so, then these cases would come into yet closer affinity with those of diptheritic paralysis, where the functional affection is replaced (or produced) by the action of a poison.

Dr. Jacobi holds — a matter which it would require very large statistics to prove — that in cases where the original disease has been severe and the temperature high, paralysis is less likely to occur, because the poison has been more likely to have been all rapidly eliminated; that in cases where the general symptoms and local affection have been slight, paralysis comes on more certainly, more slowly and more insidiously, since the poison has not been dispersed through the system so rapidly, nor eliminated so thoroughly. He excludes from this system the cases of rapidly occurring paralysis of the palate, which he ascribes to directly local cause; and it is possible this may be quite correct. He describes the lesion as consisting in
Gowers. Diseases of the Nervous System. II. 813.


Handfield Jones. cit. cit.

see Lancet of 9th March 1887.
in a tropic affection of the motor system, almost always peripherally in nerves and bundles, seldom, if ever, in the centre.

Kempe, writing on this subject says that an extraordinary series of cases was observed by Boissonade, may even suggest that paralysis may occur without antecedent diphtheria.

Boissonade saw several examples of paralysis of palate, accommodation, and joints, occurring during an epidemic of diphtheria in Paris, in the persons of some who had not suffered from diphtheria. A few of these subsequently became affected with sore throat.

Handfield Jones in the Lumbian Lectures for 1865, refers to two such cases in which the patients were affected with paralysis of the palate, extremities, and gullet, with anaesthesia, they having been exposed to diphtheria but not having suffered from it. It is very difficult to accept such statements as correct, when we know how very slight and evanescent may be an attack of true diphtheria, and also that in exceptional cases, even moderately severe sore throat may cause not the slightest pain or inconvenience. If the facts stated
be established, they are very remarkable and suggestive.

But though I adopt the poison theory of the causation of paralysis, producing its effects by infection of the nerve centres, I think that the adoption of that theory does not necessitate the acceptance of a bacterial origin of the disease. I am one of those persons, daily, year, becoming fewer, who look with disfavour on the attempt to refer all diseases to micro-organic causes. It may be that Diphtheria is produced by such a cause; I am not able to prove the contrary, but at present at any rate there are peculiar difficulties in the way of proof. It would be an interesting study in view of the recent development of this and kindred subjects, to enquire into the alleged epidemic origin of Diphtheria; but it would lead me too far from my subject at present.

I am aware that there are objections to the poison theory; such as the occurrence of paralysis at a time remote from the occasion of absorption of the poison. It must be said, however, that there is a tangible poison which

Need
Taylor's Medical Jurisprudence. I.
need not produce any action for some days subsequent to its administration; refer to phthisis; and there are two diseases at least whose behaviour is analogous. I suppose that there is no limit practically to the time at which Syphilis and Malaria may exert their effects, during the intervals of which there may be apparently perfect health. I know no theory which explains so well as the one I have adopted all the peculiarities of Diphtheritic paralysis; the others all fall short in some point or other.

To what is already known with regard to the time of onset of paralytic symptoms I have nothing to add. P Fahr gives the time of onset as generally from the third to the sixth week after commencement of the throat ulceration. Lormalet gives eight to twelve days after complete recovery. The paralysis of palate, which in the great majority of cases is the first paralysis to occur, may be found to begin as early as two days, or as late as three months from the
Magie. des Paralysies Diphteritiques. Restitut by Jacob.

Démon. Gazette des hôpitaux. 1877.
commencement of the illness. There is an impression, which James sternostomically denies, that the later the onset the severer the palsy, and there is some ground for the assertion. Magne believes that the sense of taste is lost before any other paralysis occurs. Cases Nos. 6, has the very unusual occurrence of paralysis of palate being delayed till after that of accommodation has occurred, and Case No. 5 exhibits the peculiarity, also very rarely found, of paralysis of accommodation being the only palsy which apparently ever occurred. Cases similar to No. 6 have been recorded by Schelby-Brun as well as numerous ones resembling No. 5. Such have also been seen by Dr. Dagenstecher, Bartels, and others.

It is quite in accordance with what has been observed by others that all my cases with one exception were to be found in persons under 15 years of age. This is the case in all probability merely because Diphtheria is vastly more common at that age than later. I do not lay much stress upon the fact that six of my
on page 25.

Donders. loc. cit. 592
patients out of eight were girls. I regard this simply as a coincidence, as it is believed that sex has no influence on Diphtheria.

Besides the fact, previously mentioned, that all my cases occurred in Hypermetropes, another fact is brought into prominence by that series. In only one case were the pupils dilated, and even then they were not immovable. Now Donders says:—

"Uncomplicated paralysis of accommodation has only one objective symptom: dilatation and immobility of the pupil," but he adds, "the connection between paralysis of the pupil and accommodation cannot be called absolute; once formed satisfactory accommodation is still consistent with absolute immobility of the pupil. In one instance, too, paralysis of accommodation disappeared without a change of the mobility of the pupil, and on the other hand, with perfect or almost perfect loss of accommodation, the motion of the pupil may be but little disturbed." He does not indicate under what circumstances he observed these conditions.
1864.

Greenhow quoted by J. Roberts. Practice of Medicine.
book was published our knowledge of the subject has, however, greatly increased, all we can say now of the relation of paralysis of accommodation to dilated pupil is that one is a frequent accompaniment of the other. Very various have been the statements made with regard to the pupil in post-diphtheritic paralysis of accommodation. Pagnozzi, however, never saw a case in which the pupil was dilated. In his 24 cases, Schulte-Bexpérience found only one in which the dilated dilatation and sluggish reaction of the pupil could be observed; in all other cases the pupil moved itself normally. He quotes Jacobson, Tacon, Manigault, and Leguen as agreeing substantially with this statement; they consider dilated and sluggish pupil decidedly the exception, and with their statements my cases agree. Weiler, on the other hand, and Greenhow, but considered the dilated pupil to be the rule. In only one of my cases were the pupils markedly dilated, but in this and two other they were decidedly sluggish in their reaction to light, although some movement was distinctly present.
Manz. loc. cit.
present. I have never seen it entirely absent. Schubert, Buch and Marx regard mydriasis as a complication of cycloplegia just as any other paralysis might be, and remark that it does not have the same course as cycloplegia, but sometimes precedes, sometimes remains behind it. In the thirteen cases which the former observer saw of non-diphtheritic paralysis of accommodation, it is interesting to note the difference, since in five of these mydriasis was present. He says that Voelckers concluded from the rarity of the combined accommodation and papillary paralysis that the lesion must be peripheral and not central (i.e. either must be in the muscle itself or in the nerve terminals), an interesting conclusion, since the development of the question has for the same reason led me to a precisely opposite opinion, viz. that the lesion is central and not peripheral.

In no paper or book on the subject of diphtheria and its subsequent paralyses have I found any observation on the
Subject of the power of Convergence. In one of my cases (No. 3) convergence could not be maintained within six or eight inches' distance from the eye; in another it could not be maintained within eighteen inches. To show that this was a true paresis and not merely an effect of weakness, it is only necessary for me to point out that in the former case there was widely distributed paralysis of the palate completely inco-ordinate, the gait staggering etc., while in the latter, the one in which convergence was most interfered with, there had been incomplete paralysis of the palate, which had entirely passed off, but no other paralysis.

Now the important point in both of these two cases was this, that there was no deficiency in action of either of the two Internal Recti. This was particularly noticeable in case 3; the girl wished to look at an object at either side, the eyes moved freely; there was no "hanging fire" on the part of either internal rectus; the eye could be turned completely in to the nose. Thus there was perfect movement of either internal rectus, but not of the two combined.
continued... This I regard as a very strong indication indeed of the central origin of the paralysis.

Again, this failure of convergence is of a nature entirely different from that observed in cases of defective vision on the part of one eye, which is usually seen in myopes or those affected with astigmatism, and which is described under the somewhat misleading title of Insufficiency of the Internal Recti. In such cases, when the object looked at approaches within a certain distance of the eyes, one eye continues to be directed straight up or, while the other, after a moment's hesitation, slowly or quickly turns outwards and becomes misdirected. In these true cases of paresis of the function of convergence, on the contrary, when the fixation object approaches within a given distance, neither eye remains fixed upon it, and neither rotates outwards; the effort to converge simply fails, and both eyes maintain a position of fixation of a more distant object; diplopia, contrary to the former case, at once resulting. Thus the two conditions are
dieser. lcb. eit. p. 20.
are entirely dissimilar, are not even analogous.

In the case in which the pupils were dilated and sluggish to light, they moved only slightly during attempted convergence and accommodation. In two other cases, in both of which there was free reaction to light, the contraction of the pupil associated with accommodation and convergence was lost; and one of these cases was that in which the function of convergence was most interfered with. This fact also I regard as strongly indicative of a central lesion.

It used to be thought that the contraction of the pupil which accompanies accommodation was also dependent on it, but it has more recently been shown that the Mydriasis is rather to be ascribed less to the accommodation than to the simultaneous act of convergence. Leser says: "Die die Accommodation für die Kähe, begleitende Pupillenverengung, ist also nicht so wohl an diese als an die gleichzeitig einstretenden Convergenzbewegung gebunden."
American Journal of Medical Science. Edited by Leeser.
pupillary does not agree in point of time with that of the ciliary muscle. There are various other proofs: Albers demonstrated that the size of the pupil was proportional to the convergence-angle. Adamietz and Worrino have shown that when a high myope causes his visual axes to converge upon an object, which, though near, is yet beyond his "far point," and therefore to accommodates, his pupils contract; that is, when one converges without accommodating, his pupils contract. The same phenomenon is exhibited when one endeavours to overcome the effect of a person whose face is turned outwards; one has then also to converge without accommodating and the pupils then contract. When one accommodates without converging, as when one endeavours to overcome the effect of concave lenses, pupil contraction does not occur. Leake, an American observer who states that by long practice he has acquired the power of converging without accommodating, and vice versa, agrees in the same results. The pupils also of aphatic eyes contract during convergence although
The case has not yet been published.
although there is no power of accommodation, but it must be remembered that the impulse to accommodation is still present although the power is not.

Probably the truth is that neither convergence nor accommodation is the cause of the Myopia, but that all these movements, which habitually occur simultaneously, are so associated that though under certain circumstances one can be separated from the others, there is a very close relationship among them.

Hastie saw a case which has an interesting bearing on this subject; there is not the slightest reason to think it is of Diphtherial origin, although the history is decidedly against any such idea. It is that of a little girl, pale and quiet, but apparently quite healthy, who complained ofAsthenopia. Found her to have for all distances, divergence of one eye, but no Diplopia. V.R.T. 20 of J. No 1, which she reads with ease. She has 1-5 D of Hypermetropia. She never makes any attempt at convergence. When urged constantly to fix the eyes with all the energy she possesses upon a small object held at a distance of ft.
Quoted by Swansy, Handbook of Diseases Feb 24th from Graefe-Sieweck, vol II.
lies that five inches from the eyes, she can by a great effort converge to it. As a general rule however, she does not converge at all but merely fixes with one eye. The pupils react to light freely but to accommodation not at all. Here there is a case of active accommodation, with less or congenital deficiency of convergence, in which the pupil is mobile to light but immobile to accommodation. I should have said that the internal recti are perfectly active at natural movements. Albert thinks there is probably a centre for the three actions of accommodation, convergence, and contraction of the pupil; at any rate, Jensen and Wielckers showed that the three centres were close together. It is on account of the closeness of association of convergence and contraction of the pupil that Speil disposed to favour the arrangement of centres suggested by Jensen Wielckers rather than that of Kahler and Rich (see page 17), who separate these two centres by that for the Levator Palpebrae Superioris. In my cases, there were found: one case (No.3.) in which convergence, convergence-reaction, and light-reaction, were all
And one (No. 8.) in which all movements were active.
all feeble; two (Nos. 6. & 8.) in which these
movements were perfect; two (Nos. 2. & 5.)
in which Convergence and Convergence
paralysis were good, while light reaction was feeble;
one (No. 1.) in which Convergence and light
reaction were good and Convergence paralysis lat;
and one (No. 4.) in which convergence and Con-
vergence-paralysis were almost lat, while the light
reaction was normal.

It is interesting to observe that when
the accommodation is paralysed, one may
have a condition of pupil exactly the opposite
of that described by Argyll Robertson, namely
a perfect action of the light-reflex, along
with a paralysis of the {Convergence-paralysis}.

Will such a condition be found after difflusion
without paralysis of Accommodation? Does
convergence paralysis ever occur without
that of accommodation; does paralysis of
light reaction, or of Convergence reaction,
ever occur without the others under the
same circumstances? These are questions
which I cannot answer yet.
A line of distinction sharp and clear ought to be drawn between those nervous symptoms consequent on Diphtheria, which I believe to be truly paralytic, and which I have endeavoured to show are so, and those symptoms superficially resembling them, but in reality totally different, which occur after severe acute illnesses or as a result of prolonged drain on the physical resources. I mean the weakness of limbs, the enfeeblement of accommodation, and other symptoms following fevers, pneumonia, prolonged debility, etc.

These are simply weaknesses resulting from overwork. The nervous energy having been used up in the struggle with the disease or in the endeavour on the part of nature to supply nourishment for two persons out of a supply of aliment which is perhaps insufficient for one. In order to carry on the work of life one requires to have a certain stock of nervous energy and power, and in order to meet extra demands this must be in excess of the daily need. If too much of this is absorbed and used up, in fever, when particularly the nutritive processes are acting...
Hermann Schmidt. Graefe's Archiv. XIV. 1. 106.
badly and the fresh supply of energy is thus curtailed, this want of nerve power shows itself in weakness for weeks or months afterward, even in health. The same thing takes place. How is it that one forgets pain, mental or physical, when hard at work? Is it not that the surplus nerve energy, some of which has been being directed to the seat of pain, has now become diverted into the channel of work, and the demands of the former have to give way to the latter? To illustrate my meaning, I shall refer to a paper written by a Dr. Hermann Schmidt "über Accommodation, Beschwerden bei Zahnleiden." He observed 92 cases of toothache in which the range of accommodation was reduced, in some cases slightly, in some enormously. He looked upon this diminution of accommodation as a symptom of increased tension of the globe brought about by stimulation of the sensory fibres of the V nerve, resulting from the reflex stimulation of vasomotor nerves. The circumstance that the younger the patient was, the worse and the more certainly occurring
was the symptom, he explained as due
to the greater elasticity and the less rigidity
of their vessels. I cannot agree with him
on the subject of causation. I believe the
loss of accommodation to have been an
instance of the demand made by the abnormal
condition of the brain, absorbing some of
the energy which should have been directed
to the normal act of accommodation, which
thus became impaired. I may add that
Priestley Smith of Birmingham examined several
cases with his tonometer, with a view to verifying
this statement of Schmidt, and could discover
no rise of tension whatever. I make no
doubt—Schmidt was entirely in error in the
matter.

When the active supervention of the
mind, so to speak, is withdrawn, as when
sleep occurs, a somewhat analogous event
takes place. If one falls asleep over a
book which he has been reading, and
notices his symptoms as he does so, he
will observe that the print becomes blurred,
that diplopia occurs, and that he loses
his place on the page. These symptoms
are produced by the relaxation of the ciliary muscle, and of convergence, and by the upward
of the eyes which occurs during sleep, and indeed whenever the eyes are closed. The
advent of sleep has prevented the declaration
of nervous energy to these functions, which
then become abrogated.

In a similar fashion after fevers, so I
have been endeavouring to explain, the
ciliary muscle is weakened, and when
an endeavour is made to read, it often
refuses to act because it is unable.

And in this lies the difference between
a true paralysis such as occurs after
diphtheria, and the asthenia of fever.
The diphtheria patient, or rather he who
was the diphtheria patient, whose other
muscles are active, finds one or two
of them useless, and does not understand
his condition. The fever patient's muscles
are all weak; while he cannot read, neither
can he walk, and he rightly attributes his
condition to weakness. I do not say that
true paralyses do not occur after fevers, but in
the majority of cases the asthenic condition is quite distinct
the true paralysis of diphtheria.

To the subject of prognosis it is not necessary for me to enter. I have never heard of an accommodative paralysis becoming permanent, though, as we have seen, other forms of diphtheritic palsy occasionally persist. My cases are all improving or have entirely recovered, with the exception of Case No. 5. Improvement in this case is advancing so slowly that it is almost imperceptible, but I think he will eventually recover. Some one or two of the cases, I have observed, after remaining almost stationary for a time, recover so rapidly that it is almost sudden.

Holding the views which I have now given expression to in this thesis, I reluctantly regard the treatment as a matter of little importance. I believe that fresh air, particularly sea air, tonics administered internally, and above all, time will be found sufficient treatment for all diphtheritic paralyses of accommodation.
Kingsford. Lancet. II. 1888. 488.
accommodation. For the general paralytes, raw meat is strongly recommended by Blacke, according to Mariagault.

Belladonna and quinine have both been much praised. Kingsford, whose knowledge of the disease was very extensive and who believed, as I think I mentioned previously, that it was closely allied to Syphilis, administered iron in large doses. A good deal of difference of opinion exists as to the use of Skalphine. It is recommended by Sidney Ringer, Edouard Smith, Handfield Jones, Froboseau, Sketch Dorée and others. Moynier says in reference to it: "Il faut autant que possible pour obtenir un effet appreciable, arriver à produire des démangeaisons à la peau". Leotard adds: "by all means the employment of Skalphine and Veronia in the form of Skalphine. Extensive experience has, he says, taught him to "look upon Skalphine as the most valuable remedy in Diphtheritic paralyzes, and he employs it, as do Bonnie and some others, substantively and near the seat of palsy. Others, again, of whom Bertel is the chief, deny the usefulness of Skalphine. This differs
Vertet. Eeinosen's Cyclopaedia. I. 698.

Sorrens. loc. cit. II. 846.
On this matter is worth quoting, especially in contrast to the statement of Morpier just quoted. "These preparations [Styphnic] cause the desired muscular contraction by acting upon the central organs, and if given in these cases in sufficient amount to produce this contraction, it will not only be difficult to avoid the effects of poisoning by Styphnic, but we shall be pretty certain to cause such an irritation in the Medulla Oblongata and Spinal Cord as will almost necessarily aggravate the morbid changes which are the foundation of the paralysis." Between these two different veins Gowers occupies a middle position. Styphnic, he says, "sometimes seems to be of actual service, but it is certainly powerless to neutralize the morbid process in its early stages and seems to be without influence on the spread of the disease. Moreover, it is not wise to give large doses of a drug that stimulates the nerve cells so powerfully."

Wade of Birmingham, whose name is associated with the observation of Alluminuria in Diphtheria, recommends the use of Alluminants, such as Acids of Allumin, or...
of Iron, Bichlorides, Mercury and Bichromate, and the idea is good, particularly if one holds the opinion of the continued existence of poison, but I cannot agree with the view when he condemns tannin as of little use. My own opinion with regard to Strychnine is that it is useful, partly perhaps, simply as a powerful irritant to the nerve centres, and keeping up their "tone," but I should never think of pushing the use of it to the extent advocated by Kropin and feared by Perot.

In the department of local treatment, there are various opinions as to the use of Myrtus to stimulate the Ciliary muscle. Bichromate thought the less persistence of this case, which he believed lasted a shorter time than the average, was due to the use of Calabar Bean. Henry Williams of Harvard also considered Exerin useful in abbreviating the duration of the paralysis. Exerin readily in these cases contracts the pupils and excites the ciliary muscle to contraction.
action temporarily, but whether it can produce any lasting benefit is very questionable. It is not a point on which one would have any right to give a decided opinion without seeing a long series of cases, since the duration of the paralysis is acknowledged to be as uncertain as its onset.

Should the patient wear correcting spectacles? Dioscorides forbade the use of them, others have recommended them. No doubt a patient ought to tear all work alone until after all paralysis have disappeared, but we frequently see patients who, being otherwise in good health, will not close, and see no harm in allowing them to wear glasses of a strength rather insufficient. Being insufficient, in order that the ciliary muscle may have a certain amount of stimulus to contract. It would, therefore, be wrong to order fully correcting glasses, and thus relieve the ciliary muscle of all duty.
Summary.

We have seen in the preceding pages proved or endeavoured to prove, concerning Diphtheritic Paralysis, that the lesion can be neither peripheral nor intermediate; it must therefore be central.

That in all likelihood, since recovery is the almost invariable rule, no serious damage can be done to the cells of the nerve centres; that therefore the symptoms are probably to be ascribed to the action of a poison.

That accommodation may be paralyzed without affection of either convergence or pupil movement.

That in certain cases, convergence is paralyzed along with accommodation, without implication of pupil movements or of lateral movements; paralysis of the light-reflex of the pupil without loss of convergence-paresis; and that in other cases, loss of convergence-paresis is associated with the action of the light-reflex.
I had not originally intended that this paper should have extended itself so far, but the subject interested me, and it has grown under my hand.

First that the observations which it has been my good fortune to have had the opportunity of making upon the variable motility of the pupil and other secondary circumstances attendant upon the paralysed state of the accommodation as produced by Diphtheria, maybe of service in guiding us to a true notion of the pathology of this most interesting condition.

William George Sym.