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COMMUNICATIONS

COMPLETE AND INCOMPLETE FORMS OF THE BENIGN DISORDER CHARACTERISED BY TONIC PUPILS AND ABSENT TENDON REFLEXES

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IN earlier papers on this subject it has been suggested that certain apparently dissimilar abnormal pupillary reactions formerly described as occurring in unrelated conditions are manifestations of the same disorder. The abnormal reactions are, on the one hand, the tonic pupillary reaction (syn. pupillotonia, myotonic reaction, tonic convergence reaction of pupils apparently inactive to light, Marcus's peculiar pupil phenomenon, non-luetic Argyll Robertson pupil, pseudo-Argyll Robertson pupil) and, on the other hand what may be called atypical phases of the tonic pupil. In the atypical phases tonic reactions are absent or difficult to detect and the state of the pupil in cases that present them is usually designated by the term fixed pupil, ophthalmoplegia interna, iridoplegia or partial iridoplegia, of unknown origin.

The disorder referred to, if the views expressed here are correct, may manifest itself in the following clinical forms:

A. The complete form characterized by the presence in one or both eyes of the tonic convergence reaction in a pupil apparently

inactive to light and by absence or diminution of one or more of the tendon reflexes.

B. Incomplete forms with (1) Tonic pupils alone. (2) Atypical phases of the tonic pupil alone. (3) Atypical phases of the tonic pupil with absence of reflexes. (4) Absent tendon reflexes alone. (This form will not be considered here.)

The evidence on which this synthesis was based is briefly as follows. Many cases have been described presenting the features of each of the forms just named. Apart from some of the pupillary signs the features of each group are identical in every respect. The high proportion of females affected, for example, is exactly the same in each group. In all of them too the eye signs are unocular in a proportion of about four to one, that is in a proportion that differs from the incidence in any of the better known forms of abnormal pupillary reaction. No known hereditary constitutional or acquired disorder plays any part in the production of the abnormal signs; the course in each group is benign.

Considerations of this kind led me to the conclusion that the features that seemed to distinguish the groups, namely the different states of the pupil and the presence or absence of abnormal tendon reflexes, were fortuitous. This belief was strengthened when patients were encountered with tonic reactions in one eye and fixed or partially fixed pupils without tonic reactions in the other, some of them with and some without tendon areflexia. Further evidence was supplied in descriptions of cases that presented iridoplegia at one examination and tonic reactions some years later, or the reverse.

It is interesting to note the steps in the development of our knowledge of this subject. The tonic or myotonic pupillary reaction was first described in 1902 by Saenger¹⁹ and by Strasburger²⁰ independently. Among the earlier records of cases presenting this anomaly I found nine in which absence of one or more of the tendon reflexes was mentioned; this aroused no curiosity in those who observed it but was passed over without comment or misinterpreted as evidence that the eye signs were luetic in origin; "probably congenital tabes," "incipient tabes dorsalis," "as yet no other signs of tabes or G.P.I." are representative of the comments on these cases.

In 1921 Behr⁵ described eight new cases of pupillonia and concluded that the presence of this abnormality did not justify a diagnosis of syphilis. In one of his patients the ankle jerks were absent.

In 1924 Foster Moore¹³ published his first paper on the "Non-Luetic Argyll Robertson Pupil." His cases were characterized by "the complete or substantially complete inaction of the pupil to the light stimulus, the leisurely manner in which it contracts

with convergence and in which it dilates again after relaxation of convergence; the frequency with which it is unilateral; the integrity of accommodation; the absence of syphilis; the presence of knee jerks and the absence of signs of any nervous disease even after many years." His clear description of the pupillary phenomena leaves no room for doubt that he was describing the tonic pupil of other writers.

At this date then it was known that abnormal reactions superficially resembling the Argyll Robertson phenomenon might occur in non-syphilitic subjects. The occasional association of absent tendon reflexes with those signs had given rise to no comment.

In 1927 and in 1931 Morgan and Symonds^{15, 16} published papers with the titles "A series of cases with rapid onset of unequal pupils and failure of accommodation: a *forme fruste* of encephalitis lethargica" and "Internal ophthalmoplegia with absent tendon reflexes." These papers are of great historical interest for our subject. Among earlier accounts of similar cases, the first of which I have knowledge was described in 1906 by Edwin Bramwell and Sinclair⁶ in an article on "Ophthalmoplegia interna unilaterale," but Morgan and Symonds published the first *series* of cases presenting abnormal pupils and absent tendon reflexes of unknown but *certainly of non-luetic origin*. They described the pupils in their cases as being fixed or very sluggish to light and on convergence. In their second paper they mention Foster Moore's group but emphasise the differences between their cases and his.

According to the view expressed here the differences are immaterial and presented themselves fortuitously but at the time they seemed to justify a separation of the two groups. In one group, by chance, the tendon reflexes were normal in every case; in the other they were abnormal. In one group tonic pupillary reactions were present, in the other they were not observed. In one group accommodation was unaffected and the signs were found on routine examination, in the other visual symptoms came on suddenly and were found to be due to defective accommodation. Hence the identification of these groups as different manifestations of the same disorder was not suspected.

During the last year the number of cases available for study has been greatly increased in papers by Foster Moore,¹⁴ Gordon Holmes,⁹ and myself.^{1, 2} Foster Moore reported 15 cases presenting the non-luetic Argyll Robertson pupil; whereas the reflexes were normal in his first series, diminution or absence of one or more of them was noted in three of his later cases:

Among 54 cases of partial paralysis of the pupil of unknown aetiology, Gordon Holmes found 19 in which the paralysis of the pupil was associated with signs of other nervous disease, chiefly

loss or diminution of the reflexes. In ten cases tonic pupillary reactions were detected. Holmes remarks that his group is allied to, possibly identical with, that described by Foster Moore, and while emphasising the non-syphilitic nature of his own cases, he quotes Gehrcke in support of the notion that tonic pupillary reactions may be found in a number of dissimilar conditions.

Our knowledge will be advanced if the views I have expressed are accepted as correct, namely that (1) all the groups mentioned above are certainly manifestations of the same disorder, and (2) that the tonic pupillary reaction, as here defined, is peculiar to this disorder.

So far we have been considering cases with tonic pupils alone, and tonic pupils or "atypical phases of the tonic pupil" with absent tendon reflexes. There remains for consideration the hypothetical form in which the disorder is manifested solely by the presence of an abnormal pupil which does not present tonic reactions. It will be more convenient if, before dealing with this group, the tonic pupil is described and attention drawn to some of its variants.

The Tonic Pupil

In its most characteristic form, the tonic pupil is unilateral, and considerably larger than its fellow. To the usual bedside tests the reaction to light, direct and consensual, is completely or almost completely absent. After a sojourn in a dark room for an hour or so the pupils usually dilate to an equal size; thereafter, on exposure to bright diffuse light the abnormal pupil, perhaps after some delay, contracts slowly and may continue to contract until it is smaller than it was before it dilated in the dark; after some delay it dilates slowly to its original size. The most consistent, the most easily recognized, and for diagnosis the essential feature of the true tonic pupil is its behaviour during and after convergence. While the patient gazes at a near object the pupil, often after some delay, contracts slowly and with increasing slowness through a range sometimes greatly in excess of the normal, often down to pinhead size; having been larger it almost always becomes much smaller than its normal fellow.

After the effort to converge is relaxed one of several things may happen; the pupil continues to contract (rare), or remains fixed for several or many seconds (the rule) or begins to dilate at once (common); dilatation once begun proceeds at a rate even slower than that of the preceding contraction and many seconds or even minutes elapse before it regains its usual resting size.

The ciliary muscle may be affected in the same manner; the resulting disturbance in accommodation is noticed most often during relaxation; after fixation of a near object several seconds

elapse before distant objects become clear. The movement of the ciliary muscle is slow but not reduced in range; it always corresponds to the age of the patient; there is no constant relation between the rate of contraction of the iris and of the ciliary muscle in the same case; tonic accommodation has never been observed apart from tonic convergence reactions (Axenfeld)³.

The tonic pupil dilates well to mydriatics and contracts under eserine. Repetition does not facilitate movement as it does in myotonic skeletal muscles.

In the cases I have analysed the eye signs were unilateral in a proportion of just under five to one. There are very few exceptions to the rule that the abnormal pupil is the larger one. It is sometimes irregular in outline or oval with the long axis horizontal or vertical. Changes in size from time to time often through a wide range are common. (This is an important diagnostic feature.) Behr⁵ saw marked hippus confined to the abnormal pupil in three of his cases.

The reactions often vary in the same eye at different times. In most of my cases I was able to confirm the observations that the pupil dilates in the dark (Dimmer)⁷ and then contracts in bright diffuse light (Lerperger)¹⁰ sometimes to a size smaller than it was before (Behr)⁵. Relaxation after convergence always proceeds more slowly than the tonic contraction. Both movements become slower as they proceed. Very rarely contraction is normal, dilatation only being abnormally slow. The tonic convergence reaction may easily be missed owing to the long delay that may occur before contraction begins. Some patients have difficulty in sustaining the effort to converge and must be persuaded to persist for 20 seconds at least; five or more seconds may elapse before contraction on convergence is detected which once begun may continue slowly through a wide range. On one occasion, in a patient whose pupil usually began to contract in about five seconds, ten seconds elapsed before any movement was noticed.

The more of these cases I examine the more firm is my belief that complete absence of contraction of the pupil on convergence is uncommon and that the tonic reaction would be found more often if it were diligently sought for in cases of "fixed pupil," "iridoplegia" and "partial iridoplegia" of unknown origin. Nevertheless the pupil in this disorder may rarely be so stiff that no reaction to light or on convergence can be obtained. One patient with a tonic pupil and absent tendon reflexes showed a typical convergence reaction in one pupil; in the other which had recently dilated suddenly no response was obtained to light or on convergence. In two others with no history of sudden dilatation, but like the one just mentioned with some difficulty in accommodating, one pupil was tonic the other fixed. It is possible that I was not

persistent enough in these cases but they prove that in some cases, certainly examples of this disorder, the pupils may be or appear to be completely inactive. Some of these fixed pupils present tonic reactions later; a pupil known to be tonic may dilate suddenly and then become fixed.

Isolated Ophthalmoplegia Interna and Iridoplegia of Unknown Origin

We can now consider the cases that abound in neurological and ophthalmological literature of ophthalmoplegia interna, ophthalmoplegia interna unilaterale, iridoplegia or partial iridoplegia for which no cause can be found.

Since 1878 when Jonathan Hutchinson introduced the term ophthalmoplegia interna many papers have appeared giving descriptions of series of cases with an analysis according to the supposed cause. In every series there is a large residuum after cases attributable to third nerve palsy, syphilis, diphtheria, encephalitis lethargica, herpes, atropine, and all other known causes of iridoplegia have been excluded. In many of these cases eye signs persisted for many years without the appearance of other evidence of nervous disease. In a few, tonic reactions appeared during the period of observation. Cases with tonic reactions in one eye and pupils of the kind now under consideration in the other have already been mentioned. The abnormal reactions are usually detected in early life and females preponderate.

With a full appreciation of the paucity of the evidence but impressed by the fact that the tonic reaction is not always sought for, that even when sought for it is easily missed, that the state of the pupils in these cases is exactly that seen in cases where the diagnosis is made certain by the associated abnormal tendon reflexes and that the incidence as regards sex and age is the same, I submit that many cases of ophthalmoplegia interna or iridoplegia of unknown origin are examples of the disorder described here. This matter will be mentioned again under diagnosis.

Relation of the Tonic Reaction to other Abnormal Reactions of the Pupil

Behr discussed this aspect of the subject and concluded that the tonic reaction differs essentially from the abnormal reactions seen in the Argyll Robertson pupil, fixed pupils and ophthalmoplegia interna. The Argyll Robertson pupil as described by its discoverer is small, usually bilateral, constant in size and unaltered by light or shade; it contracts promptly on convergence and dilates again promptly when convergence ceases; it dilates slowly and

imperfectly to mydriatics. It thus differs from the tonic pupil in every essential point and can hardly be confused with it by anyone who is familiar with the distinctive features of both anomalies. But because the tonic pupil seems to be inactive to light and does not react on convergence, so conforming to the current incomplete and misleading definition of the Argyll Robertson pupil, patients with tonic pupils especially if the tendon reflexes are also abnormal are almost always thought to be suffering from syphilis of the nervous system. The tonic reaction is unilateral in about 80 per cent. of the cases; the Argyll Robertson pupil in about 5 per cent., the fixed pupil in less than 10 per cent. and ophthalmoplegia interna in about 20 per cent.

The tonic pupil is almost always larger than its fellow; the unilateral Argyll Robertson pupil is always the smaller. If we call a small pupil one with a diameter of less than 3 mm., then the tonic pupil is hardly ever small, whereas the Argyll Robertson pupil is small in 80 per cent. of cases, the fixed pupil in 9 per cent. and the pupil of ophthalmoplegia interna in 10 per cent. (Behr.) The tonic pupil often varies in size from time to time, and may show hippus. In the dark the pupils become equal. These things the unilateral Argyll Robertson pupil never does. The behaviour of the tonic pupil that has dilated in the dark is peculiar to it; slight changes with alterations in the illumination may occur in other conditions but not the full or even excessive movements of tonic pupils. The movement on convergence differs from that of the Argyll Robertson pupil; in the latter this is typically prompt and dilatation is equally prompt.

The fact that the tonic pupil can usually be made to contract through a normal range on exposure to light after a long sojourn in a dark room and on convergence if this is long enough sustained suggests that there is no true paralysis but some qualitative change in its mobility. The same is true of accommodation; the movement is slow but not reduced in range; this is essentially different from the findings in ophthalmoplegia interna where the diminished range of movement is decisive for diagnosis. Syphilis is an important factor in the production of other abnormalities of the pupil but here, I believe, it plays no part.

All the evidence seems to me to support the notion that the tonic convergence reaction in pupils apparently inactive to light is a thing apart. The peculiar extra-ocular phenomena (symptomless areflexia) that are frequently associated with it also suggest that we are confronted by a unique condition.

On the other hand it has been stated that tonic reactions have been observed in hereditary syphilis, tabes, general paralysis, disseminated sclerosis, diabetes and other conditions and that Gehrcke⁸ collected 20 cases of this kind. The alleged syphilitic

cases are most numerous and most easily disproved. Gehrcke accepted the diagnosis of the original authors. Their mode of reasoning was that tonic reactions are a sign of syphilis of the nervous system, therefore syphilis is one of the causes of tonic reactions. His list is headed by three cases of general paralysis described by Piltz. These cases are ruled out by the fact that the reaction on convergence was normal. Markus's case is included, presumably because the knee jerks were absent. Markus's own comment on this case was that the signs "might be considered sufficient evidence of congenital syphilis. I am, however, inclined to take a less serious view." The cases of Nonne,¹⁷ Lerperger¹⁰ and Axenfeld⁹ were labelled ? tabes dorsalis, tabes incipiens and lues congenita? respectively because tendon reflexes were absent. In none of the cases of supposed luetic origin was there any real evidence of syphilis. In 1921 when Behr studied the relation of the tonic reaction to syphilis he had to contend with the fact that many of the authors who preceded him believed that the tonic pupil was a variant of the Argyll Robertson pupil. Now, with close on a hundred cases available for study, we are able to form a very definite conclusion.

A large proportion of the patients with tonic pupils are healthy young females; the eye signs they present are not observed in known syphilitics; tendon areflexia when present is not accompanied by other signs of organic nervous disease and is symptomless; patients giving evidence that the eye signs have been present since childhood have lived many years without the appearance of other symptoms. In 37 cases to my knowledge the blood and cerebro-spinal fluid were examined, in 18 the blood only, with completely negative results. These tests have been done on a much larger number of cases but I find that I have not always made a note on this point when looking up the recorded cases. I can say, however, that in one case only (Barkan)⁴ the Wassermann reaction was positive in the blood of a patient with no other sign of nervous disease except a tonic pupil. It is hardly likely that a form of syphilis of the nervous system exists which is commonest in healthy young women and is characterised by ocular and reflex phenomena peculiar to itself and never seen in known syphilitics, by the constant absence of a history of family or personal infection, by the absence of all other manifestations of syphilis and by its uniformly negative findings in the blood and cerebro-spinal fluid. It is more reasonable to suppose that syphilis plays no part in the production of this disorder.

I think the following conclusions may now be drawn with safety: the tonic convergence reaction in pupils apparently inactive to light is, in all probability, never a manifestation of syphilis of the nervous system: the combination of this sign with absent tendon

reflexes has never been observed in syphilis of the nervous system and is not one of its manifestations. The evidence for the alleged incidence of tonic reactions in disseminated sclerosis is confined to the case, a boy, aged 17 years, labelled by Strasburger²⁰ in 1902 “? sclerosis multiplex.” The only case, so far as I know, of the association of tonic reactions with diabetes was recorded by Nonne in 1902. The patient, a man aged 40 years, presented glycosuria, tonic convergence reactions and absent ankle jerks. It is more than likely, as the case is an isolated one, that the associated conditions were unrelated.

The discovery of tonic pupils in patients with some other disorder naturally gives rise to the belief that the eye signs are of recent onset and a manifestation of the disease that is producing symptoms. Thus Roemheld¹⁸ saw tonic pupils and absent reflexes in men with gunshot wounds of the head and labelled them “traumatic pseudotabes.” Mayer¹² saw the same signs in a man who had been struck by lightning and published his case under the same heading. Apart from cases of this kind there is no evidence that tonic reactions may result from trauma. Equally unconvincing are the cases in which migraine, neurasthenia and measles are mentioned as evidence of the variable aetiology of tonic reactions. My conclusion is that the tonic convergence reaction in pupils apparently inactive to light is *sui generis* and that no known morbid factor plays any part in its production.

General Features

Taking all the forms together about 80 per cent. of the cases are females. The female preponderance is slightly higher in the group characterized by tonic pupils and absent reflexes. It may be therefore that females are not only more susceptible to this disorder but are also more likely to present tendon areflexia. The signs are frequently detected in the course of a routine examination and it is not possible to determine when they first appeared. Some patients say the pupils have “always” been unequal; this was noticed at the age of six months in a patient of Foster Moore’s with tonic reactions at the age of 5 years. Sometimes the onset of symptoms is sudden; some discomfort is felt in the eye, vision becomes blurred and examination reveals a dilated pupil. Most of the patients who give this history are in the twenties or thirties but this gives no certain indication of the age of onset because we know that a pupil already tonic may dilate suddenly and then give rise to symptoms for the first time.

Apart from discomfort in the eye and difficulty in accommodation, both uncommon, the disorder runs a benign symptomless course. There was slight drooping of the lid in several of my cases.

Among 44 cases of tonic pupil described by ophthalmologists, absence of tendon reflexes was noted in nine. I suspect that the incidence of areflexia is higher than this. Neurologists on the other hand have reported groups of cases in which the reflexes were abnormal in every case; but this was their reason for including the cases in their series. Once it is recognized that the disorder may present itself in various forms reports on these artificial groups will cease and it will soon become possible to give the relative numerical incidence of the different forms. This cannot be done at present.

The most frequent extra-ocular sign is diminution or loss of one ankle jerk. I have never seen loss of any other jerk with both ankle jerks still present and equal. Asymmetry is not uncommon. In two of my cases the knee and ankle jerks were lost on one side only. The arm jerks rarely go before the knee jerks and ankle jerks are all lost. It is improbable that jerks once lost ever return.

The general health of all of my patients was remarkably good. There was nothing to suggest that they belonged to a neuropathic stock and nothing in the history recurs with sufficient frequency to deserve mention as a possible aetiological factor.

Diagnosis

The diagnosis of the complete form presents no difficulty; the combination of the tonic convergence reaction in a pupil apparently inactive to light with absence of one or more of the tendon reflexes is pathognomonic.

With regard to cases with abnormal pupils alone it must be understood that slowness of movement by itself does not constitute a tonic reaction; that is seen in retinal disease, in traumatic iridoplegia and in many other conditions. The typical tonic pupil shows delay as well as slowness in its responses; it usually responds to light after a sojourn in a dark room; it retains its new size after the stimulus is removed and relaxation is slower than contraction to light, with convergence, and when present, with accommodation. In most of the cases that appear on superficial examination to present fixed or partially fixed pupils appropriate tests will reveal tonic reactions. To elicit the reaction on convergence persistence is required on the part of the patient and the examiner. In difficult cases the most obvious sign is the relative smallness of the originally larger pupil when the patient looks into the distance after converging powerfully for half a minute or so. I am in the habit of allowing the patient to confirm this observation for herself in a mirror; it never fails to elicit an exclamation of surprise. If accommodation were tested in the patients with "ophthalmoplegia interna" who complain of misty vision it might be found that

accommodation though slow is not limited in range and that relaxation is slower than contraction. This would give the diagnosis, as tonic accommodation is peculiar to this disorder.

Where tonic reactions cannot be obtained the diagnosis may still be made with some confidence if the eye signs are unilateral and the patient is young and free from other signs of organic nervous disease. A history of longstanding inequality of the pupils with recent sudden increase in size of the larger one, of wide variations in the size of the larger pupil, or the discovery of hippus confined to the larger pupil are important diagnostic points. Occasionally tonic reactions in the smaller pupil give the clue to the nature of the larger "fixed" pupil. The combination of pupils of the kind under consideration with absent tendon reflexes, without other signs, in a young person, is strong presumptive evidence of the disorder. If persistent efforts to elicit tonic reactions have failed and a careful consideration of all the features of the case, age, sex, family and personal history, still leaves room for doubt, the appropriate tests to exclude syphilis must be made.

Diphtheria was suggested as the cause of the signs in some of my cases. Tonic reactions have never been recorded in this disease. The internal ophthalmoplegia of diphtheria is peculiar to it; there is true paralysis of accommodation, perhaps always bilateral and apart from very rare exceptions always isolated; it appears about ten weeks after the onset of the disease and recovery is always complete in a month or two. It never passes over into tonic accommodation (Axenfeld). True tonic reactions have never been recorded after encephalitis lethargica; no patient with tonic reactions has ever presented the early or late symptoms of this disease. A review of other conditions in which pupillary and tendon reflex signs may be found reveals none with which this disorder could well be confused.

Nature of the Disorder

We have seen that the tonic reactions are probably the expression of a unique kind of perversion of pupillomotor activity. Behr attributed them to a peculiar disturbance of the function of the vegetative portion of the third nerve nucleus. This notion receives some support from a consideration of the peculiar symptomless areflexia. Areflexia occurs in a number of disorders of the vegetative portion of the nervous system. Details are out of place here but it may be mentioned that in dystrophia myotonica and other myopathic atrophies the knee and ankle jerks are often lost in patients with powerful unwasted lower limb muscles and intact sensibility. Areflexia is also seen in Graves' disease, myasthenia gravis, family periodic paralysis and cataplexy, disorders in which

the peripheral nerves and central nervous system apart from the vegetative nervous system are so far as we know intact. When we know more of the functions, interrelations and affections of the ductless glands and the different portions of the vegetative nervous system we shall be on the way to an understanding of many disorders of obscure origin, including the one that concerns us here. In the meantime all we can say is that the ocular and reflex signs we have considered seem to be the expression of a kind of perversion of nervous activity of which, at present, we can form no conception.

Conclusions

The disorder characterized by tonic pupils and absent tendon reflexes manifests itself in several clinical forms; these can be referred to conveniently as complete and incomplete forms. The complete form is characterized by the presence of the tonic convergence reaction in a pupil apparently inactive to light and by absence or diminution of one or more of the tendon reflexes of the lower limbs. The incomplete forms present :

1. Tonic pupils alone.
2. Atypical phases of the tonic pupil alone.
3. Atypical phases of the tonic pupil with absent tendon reflexes.
4. Absent tendon reflexes alone.

In the atypical phases reactions are absent or difficult to detect. Most of the cases encountered in ophthalmological and neurological practice of ophthalmoplegia interna, ophthalmoplegia interna unilaterale, iridoplegia and partial iridoplegia for which no cause can be found are probably examples of an incomplete form of this disorder.

No known hereditary or acquired morbid factor plays any part in its production. In particular it has no relation to syphilis. It runs a benign course without the addition of further symptoms and is compatible with long life.

I take this opportunity of again thanking my colleagues at the Royal London Ophthalmic Hospital for their kindness in allowing me to examine cases under their care.

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CONTACT GLASSES

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RECENT interest in contact glasses would suggest that they are an innovation, but this applies only to the technical excellence of their manufacture for the conception of their clinical possibilities was perceived at least fifty years ago.

Historical

About the year 1879, Rähmann, concerned with the treatment of conical cornea, became dissatisfied with the crude lenses at that time in use and turned his attention to lenses having a hyperbolic curve. His improved results stimulated interest in the optical correction of conical cornea and in 1887 came the experimental work of Fick, which culminated in the production of the earliest contact glass.

Fick asked Professor Abbey, at that time a director of the Zeiss firm, if he could provide a contact glass having a corneal radius of 8 mm. and a scleral radius of 12 mm. by grinding. Four glasses were provided, but in 1889 Himmler pointed out that these glasses were not ground but had been skilfully manufactured by blowing.

Quite independently, in 1889, Müller, in his inaugural dissertation at Kiel, described his work with blown contact glasses which he had carried out in conjunction with Himmler.

Fick then obtained ground glasses with the corneal and scleral parts cemented together; the one piece glass appeared in 1892, manufactured by Benvist, Berthiot et Cie, and, almost at the same time, by Strubin of Basel. Müller himself had a myopia of 14 dioptres, which was reduced by contact glasses to 1.5 dioptres. He could not, however, wear them for longer than half an hour.