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COMMUNICATIONS

CONGENITAL MIOSIS OR PINHOLE PUPILS OWING TO DEVELOPMENTAL FAULTS OF THE DILATATOR MUSCLE*

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Part I.—Clinical.—By S. HOLTH

THE pupil must be considered abnormally small when its diameter is less than 2 mm. by diffuse daylight, the eyes looking at a distant object. A pupil of 1.5 mm., or less, under the same conditions is in a pathological contraction, *miosis* ($\mu\epsilon\iota\acute{o}\omega$ = I diminish); the spelling "myosis" is wrong (J. Hirschberg). This marked contraction of the pupil, most frequently appearing after a local application of eserine or pilocarpine, is a well known characteristic also of certain intoxications (especially of opium or morphia); it appears in several diseases of the brain or the spinal cord, and is seen—mostly unilaterally—in paralysis of the cervical sympathetic nerve.

These conditions are excluded in the three cases of congenital extremely contracted pupils observed by me, and which I will describe below.

Congenital miosis must be extremely rare; during thirty years I have only seen these three cases. As shown below in other cases

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the condition has been mistaken for spinal miosis or for the sequelae of iritis.

In the ophthalmological literature very little is to be found concerning congenital miosis and in the neurological literature nothing.

W. R. Wilde's essay of 1862 on "Malformations and Congenital Diseases of the Organ of Sight"¹¹ seems to be the work on which the references in English writings on the subject are based. Wilde clearly differentiates between congenital miosis or microcoria and "Sinizesis congenita" due to closure of the iridial aperture produced by persistence of the pupillary membrane. He saw "Myosis congenita" in both the eyes of one patient, unilateral miosis in two patients, one of whom had been considered elsewhere as suffering from "*syphilitic iritis*" and had been treated for this non-existing disease; a third unilateral case is called "Microcoria" but Wilde adds: "It was almost similar in appearance to the preceding cases." None of these patients could tell of any similar cases in their families. There was no microscopical examination. Neither does Wilde speak of the effect of mydriatics nor of treatment in these cases.

X. Galezowski² saw four cases of "myosis," without any disease of the optic nerve; there was nearly always at the same time a contraction of the accommodation muscle. Galezowski gave no information as to whether the cases were congenital; nor do Truc and Valude when they say³ that the pupil may be a pin point, responding comparatively weakly to mydriatics.

In Collins and Mayou's work¹⁰ the condition is twice mentioned, and the probability of a feeble development of the iris musculature is stated. In cases of congenital cataract the authors have often seen that the pupil will not dilate well on the application of atropin. I have made the same observation myself in such cases; but I have never seen the pupils so small here as in my three cases of miosis congenita.

In the *American Encyclopedia and Dictionary of Ophthalmology*¹⁴ Dr. D. T. Vail gives thirteen lines about the matter. He says that the condition must be a fault of development, and states that it may appear in consanguineous marriage of the parents, but there are also cases without consanguinity. It may be combined with microcornea or other signs of microphthalmus; there is no treatment to be suggested.

Kurt Saupé¹⁷ has observed a case of unilateral congenital miosis; the previously consulted oculist considered the pinhole pupil as Argyll Robertson's spinal miosis, and wrongly diagnosticated tabes. Saupé considers the miosis in his case as congenital, but does not know if the defect is in the dilatator muscle itself or in its innervation. The author had not the opportunity to make a microscopical examination.

Since the autumn of 1897 I have had under observation two sisters and a brother whose pupils have all their lives had a diameter of about 0.5 mm.; after several days of instillation of mydriatics the pupils were dilated to 2.5 mm. at the utmost, but generally less. Addition of cocain 5 per cent. did not further dilate the pupils, but the palpebral fissures were enlarged (some exophthalmus); this fact shows that the cervical sympathetic nerve functions well. The three patients never squinted, and had good horizontal and vertical muscle balance for the distance of 5 metres; for reading distance (30 cm.) only the few normal degrees of exophoria, no hyperphoria. Only one of them—case (a)—had spasm of accommodation.

(a) **Miss Ingeborg B.** (the youngest of them, born in 1876) came to consult me on September 4, 1897. On my inquiries she

FIG. 1.

The pupils of Ingeborg B. in her 21st year on September 5, 1897 (before application of mydriatics). This illustration as well as Fig. 2 and Fig. 3 are reproductions of pencil sketches by Asta Nørregaard, the artist; the sketches are on a somewhat reduced scale, the horizontal diameter of the corneae being in reality 11.5 mm. instead of 10 mm. as drawn. But the same artist painted the originals of Fig. 4 and of Fig. 5 in natural size.

informed me that as long as she could remember, her eyesight, like that of her elder sister Anna and of Axel the twin-brother of the latter, had been very bad in twilight. The parents had observed in all of them pinhole pupils since their birth. A younger brother, born in 1881, has normal pupils, and his eyesight is always good even in twilight. The parents were cousins; the father was stated not to see quite well, and his pupils were rather small (I estimated them to be between 1.5 and 2 mm. when, one day in 1897, he came in his 60th year for a prescription for his daughter; I proposed an examination of his eyes but he refused; some years later he died). Ingeborg complains of shortsightedness and of daily headaches, a feeling of pressure in and above her eyes with pains in the back of her head, all of which she has had as long as she can remember. Her pupils are extremely small, the right one 0.3 mm., and the left one 0.5 mm. in diameter (Fig. 1); they show a slight eccentricity upwards (Fig. 1 and Fig. 3).

The pupils do not react to light or to convergence. The colour of the iris is mainly diffuse chestnut brown, in some places however greenish grey; the radial fibres are close and stretched, and never undulating; circular folds do not exist. The pupils, after instillation of 1 per cent. atropin thrice a day for four days, were dilated to 2.5 mm. (Fig. 2); this diameter was kept for many years by instillation of scopolamin 0.2 per cent. every morning; even during this slight mydriasis, no circular contraction grooves were seen in the periphery of the iris.

In her 21st year the myopia was ascertained to be -2.75 in the right eye and -1.25 sph. $\ominus -1.5$ cyl. $.40^\circ$ in the left eye with $V = 5/4$ in both, while before the atropinization sph. -10.0 to -12.0 D were required to obtain nearly the same vision ($V = 5/5$).

FIG. 2.

The pupils of Ingeborg B. in her 21st year on October 9, 1897, after instillation of atropin 1 per cent. thrice a day for four days.

After the atropinization the normal fundus could be seen both in erect and inverted image, which was impossible through the pinhole pupils. The visual fields were good for white and colours (Ole Bull's colours—"invariable" in the periphery), for colours however somewhat more narrow before the dilatation of the pupils than after the dilatation. The tension is normal and is minus after the lapse of 26 years—still so.

After the atropinization, the patient's headaches disappeared, but in the course of a few weeks the atropin caused conjunctivitis. I then prescribed scopolamin 0.2 per cent. for instillation every morning, which did no harm to the conjunctiva. She has continued this treatment without interruption for the last 26 years; if the instillation is omitted a single morning she has her old headaches later in the day, whilst at the same time the refraction increases, though not to the same degree now as in her youth. The static refraction under mydriasis is also lower now than in her youth; the degree of myopia has decreased. Thus I found the following

refraction on October 23, 1919, at 11 a.m., after the usual instillation of scopolamin at her home three hours previously :

Right eye : $-1.75 \text{ sph. } \ominus -0.75 \text{ cyl. } 90^\circ = 5/5$.

Left eye : $-0.75 \text{ sph. } \ominus -0.75 \text{ cyl. } 60^\circ = 5/4$.

On February 23, 1921, I showed the patient to my colleagues in the Medical Society in Christiania¹⁵; to show the difference in the pupils without the slight mydriasis, and with it, I told the patient to omit the instillation of scopolamin in the right eye for a whole week beforehand. The refraction of the right eye in the patient's forty-fifth year on February 23 was: $-1.0 \text{ sph. } \ominus -0.75 \text{ cyl. } 90^\circ = 5/6$. After resuming the scopolamin application the spherical correction, as in 1919, was -1.5 and $V = 5/5$. During the week, when the right eye was without scopolamin, the patient again had her former dull pains in and over the eye and at the back of her

FIG. 3.

The pupils of Ingeborg B in her 45th year on March 4, 1921, at 1 p.m., after the usual 0.2 per cent. scopolamin instillation in each eye at 8 a.m. on the same day and after the application of an atropin crystal to the left eye at 10 a.m.

head—but on the right side only. The scopolamin application, which in 1897, like the atropin, effected a diameter of the pupils of 2.5 mm. (Fig. 2), now caused a diameter of somewhat more than 1 mm. only (Fig. 3); the addition of an atropin crystal did not further dilate the pupil.

It is the spasm of accommodation and its consequence, the headaches, which have necessitated the scopolamin application daily all these years; it has moreover highly improved the eyesight in twilight: on account of the moderate myopia, reading and close work, in spite of the paralysis of the accommodation, have been easy for the patient. If she had been emmetropic or hypermetropic without spasm of accommodation, a small iridectomy upwards would have been the best thing—in order to spare the accommodation.

On January 12, 1923, examination of both eyes under slit lamp illumination with binocular microscope $\times 24$: no trace of pupillary membrane in the pupillary area, no synechiae.

(b) **Miss Anna B.**, Ingeborg's sister, born July 28, 1867, has been under my observation since December 1, 1897. She has never suffered from headaches or spasm of accommodation. Right pupil formed a vertical slit 0.25×0.5 mm., left pupil a nearly horizontal slit 0.5×1 mm. (Fig. 4).

FIG. 4.

The pupils of Anna B. in her 30th year on December 2, 1897, before application of mydriatics and also in her 50th year in spite of continual daily application of mydriatics since 1897.

FIG. 5.

The pupils of Anna B. in her 30th year on December 9, 1897, after instillation of atropin 1 per cent. thrice a day for four days.

The iris in both eyes was blue slate colour, with a central sphincter zone yellow as raw sienna earth (a similar iris colour was seen in one of W. R. Wilde's cases): closely stretched and not undulating radial fibres, no circular contraction grooves in the periphery. The right pupil after instillation of 1 per cent. atropin thrice a day for four days became nearly round with 2 mm. diameter, while the left pupil became a horizontal oval with diameter 1.5×2 mm.; as shown in Fig. 5, both pupils have a somewhat polygonal

outline, but by focal light and magnifying lens+40 D. no posterior synechiae are seen.

Under atropin the refraction was the same as before:—Right eye—7.0 V.=5/6. Left eye—8.0 V.=5/5. The eyesight in twilight was much improved. Owing to atropin catarrh I continued the mydriasis with 0.2 per cent. scopolamin every morning for 22 years. But when she approached the age of 50 the mydriatic effect decreased. When I saw her on October 6, 1919, in spite of scopolamin the pupils were exactly of the same form and size as before the beginning of the treatment (Fig. 4), nor did atropin effect any change. The myopia was much increased in both eyes, and vision diminished by retinal changes at macula lutea (visible after iridectomy): Right eye—15.0 V.=5/30. Left eye—16.0 V.=5/15. Both eyes: tension normal and visual fields of good extent for white objects in full daylight; but in twilight very contracted. I made an iridectomy upwards first on the right eye, then on the left; the small pieces of the iris removed by iridectomy were sent to Dr. Berner for microscopical examination. I stretched the pieces out on paraffined cork plates, but they curled so much in the fixation liquids that orientation in the sections became impossible. As a result of the division of the sphincter by the iridectomy the horizontal diameter of the pupils became again 2 mm. just as after mydriatics in the patient's youth; she could now again see much better in twilight.

On March 31, 1920, the patient died suddenly of apoplexia cerebri. A post-mortem examination of the eyes was allowed on April 3—61 hours after death. Before the enucleation I injected 1 c.cm. of Bouin's fluid (saturated solution of picric acid in water 75 c.cm., formol 25 c.cm., concentrated acetic acid 5 c.cm.) in the vitreous of both collapsed globes till the tension was felt normal, and preserved the enucleated globes in the same fluid; rabbit eyes were used as prostheses. Both globes oval (with staphyloma posticum verum, axis antero-posterior in R.E. 28 mm., in L.E. 28.5 mm.) were sent to Prosector O. Berner, M.D., who could now orientate all the microscopical sections of the iris of these eyes.

(c) **Mr. Axel B.**, the twin brother of Anna (Case *b*), I have seen once only, on October 29, 1919, when he was more than fifty-two years old; his sister Ingeborg (Case *a*) had asked him to come. Like Anna he never suffered from headaches or spasm of accommodation. He said that in his youth, when he was driving on the high road by twilight, he had difficulties in steering the horse and they both often ended in the ditch. Now that he lives in town he is mostly indoors, and does not like to go out except in full daylight. He is emmetropic in both eyes with V.=5/5 and reads without glasses Jäger No. 1 at 25 cm. (pinhole pupils: minimal circles of diffusion). The iris in both eyes is an even cinnamon brown, with

the radial fibres closely stretched and not undulating; no circular grooves in the periphery. The right pupil is nearly round, 0.75 mm. in diameter; the left pupil forms a horizontal oval 0.5×0.75 mm. in diameter. After two instillations of homatropin 1 per cent. in the course of three hours the right pupil was dilated to 2×2.5 mm. and the left pupil to 1.75×2 mm.; he is still emmetropic with $V. = 5/5$, but to be able to read Jäger No. 1 at a distance of 30 cm. he must now use + 3.0D; he cannot read without convex glasses on account of larger circles of diffusion. The patient, though admitting the advantage of being able to see well also in twilight, preferred to be without mydriatics in order to do his office work and his reading without spectacles as hitherto.

On March 6, 1922, Axel B. died of apoplexia cerebri after a few days' illness. Post-mortem enucleation of the eyes was allowed and made six hours after death; protheses, rabbit eyes. The enucleated globes were preserved in Bouin's fluid and immediately sent to Dr. O. Berner.

Part II.—Anatomical.—By O. BERNER

(Abridged account by S. HOLTH)

The transverse diameter of the pupils in the enucleated eyes of Anna B. (case *b*) is 2 mm., as after iridectomy in vivo; in those of Axel B. (case *c*) 2.5 mm., as after homatropin and cocain in vivo (less a 10 per cent. deduction because of the corneal magnification; the normal post-mortem diameter of the pupil of man at the age of 50 years is 5 mm.

In a sector of each iris some sections—meridional and transversal—were made before depigmentation. As depigmentation of the sections is a method in which much time is wasted, Alfieri's method was tried in the iris in toto from normal persons of all ages, and with excellent result; then the iris and the ciliary body from the four globes with miosis congenita were also depigmented in toto by Alfieri's method. Normal irides (for comparison) were also depigmented in toto by a concentrated solution of hydrogen dioxide (from E. Merck) to which was added a few drops of ammoniacal water. By both methods the bleaching was made in a short time under an ordinary electric incandescent lamp. Imbedding in paraffin, serial sections of 5μ , staining with different methods: van Gieson's liquid, Böhmer's haematoxylin and eosin, Heidenhain's iron-alum-haematoxylin and counter-staining with eosin. This Heidenhain's staining is superior in clearness, and has the great advantage of making the sections preservable for any length of time.

Case B (Anna B.).—The microscopical examination of the sections revealed no difference between the two eyes; they will therefore be

described together. The iris (Fig. 6) is somewhat thin, with the exception of the central part, where the sphincter muscle is very distinct.

On examining *non-depigmented sections* it was at once noticed that the membrane of Bruch, or, as it is often called, the inner limiting membrane, to a great extent was defective. In a few sections and especially with regard to the intermediary and peripheral part of the iris, in front of the pigmented layer there was only a faint red rim where the membrane of Bruch should have been; but it extended only a little way in (Fig 6.)

FIG. 6. A general view of a radial section through the iris of Anna B. (Case B). It will be seen that the pupillary part is thick and contains the well developed sphincter. The intermediary and peripheral parts of the iris, on the other hand, are very thin; *sh. f.* indicate the short connecting fibres between the sphincter and the dilator, and it will be seen how the sphincter draws the dilator to itself, so that corresponding little depressions are formed upon the posterior surface of the iris.—X indicates places where small quantities of dilator have developed.

This absence of the membrane of Bruch is indeed so marked as regards the middle and peripheral parts of the iris, that it may be said to be the rule. The most that is seen is a faint reddish colour at the anterior margin of the pigment layer between this and the stroma. But in the central part of the iris, i.e., behind the sphincter, a distinct red rim, answering to the membrane of Bruch in normal sections, is constantly seen.

The absence or defective development of the membrane of Bruch between the ciliary body and the periphery of the sphincter is still more marked in the *depigmented sections*, where the epithelium continues right up to the stroma; the boundary between the separate cells in the front layer, the "muscle epithelium layer," appears as a rule, but not always, clear and distinct, although the greater part of it has not developed into contractile elements. These cells may be

fusiform (Fig. 8), or in any case somewhat pointed at the ends (Fig. 7). The majority of these cells are certainly long enough to cover 4 or 5 cells in the hinder layer; the cells in this layer are

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FIG. 7.

The appearance of the pigment epithelium from the intermediary part of the iris of Anna B. (Case B) after depigmentation; in front a flat epithelium with well marked cells. ($\times$ about 800).

FIG. 8.

A view of the depigmented epithelium from an intermediary part of the iris of Anna B. (Case B): there are well marked spindle-shaped cells in the front layer. ($\times$ about 800).

cubical, while those in the front layer—the muscle epithelium layer—are long or at any rate oval.—These long flat cells not infrequently contain 2, and sometimes 3, nuclei, which are occasionally rounded, but generally elongated.

In those places where the dilatator has developed, several types of it may be found. Behind the sphincter for instance, the normal conditions are seen. In those patches in which the dilatator is more or less poorly developed there are in front of the undifferentiated front layer of epithelium spindle-shaped epithelial cells which in form resemble plain muscle cells but in which no myoglia fibres are developed.

In most cases however, there are myoglia fibres in these spindle-shaped cells, and there is therefore no reason to doubt that these are cells of plain muscle lying in front of the anterior epithelial layer.

FIG. 9.

A drawing of the posterior surface of the iris of Anna B. (Case B), made under a binocular microscope and bright light. ($\times$ about 12).

In transverse sections are here sometimes seen—but few and far between—plain muscle bundles projecting into the stroma.

It is also due to the poor development of the dilatator that the back of the iris, under the microscope—preferably binocular—and a bright light, only very few of the radial folds are visible (Fig. 9).

In man, the dark brown, almost black, back surface of the normal iris appears smooth to the naked eye, and it is only under the microscope—preferably binocular—and a very strong light that the two groups of radial folds appear in distinct relief. As no illustration of this condition is given in ophthalmological, and only very rarely in anatomical, textbooks, we have thought it desirable to give an illustration of the posterior surface of a normal iris, from a girl of 17 years, as seen under the binocular microscope (Fig. 10), in order to show how entirely different are the conditions in the case of miosis congenita of Anna B. (Fig. 9).

Fig. 10 shows the two systems of radial folds well developed, both Schwalbe's contraction folds behind the sphincter and the structural folds which begin 1.5 mm. from the margin of the pupil, and become broader and flatter towards the ciliary margin. Here, too, there are some fine circular folds which are only formed when the pupil is dilated. In miosis congenita the circular folds are therefore entirely absent. There are only indications of a few shallow structural radial folds on the posterior surface of the iris of Anna B. (Fig. 9). Notwithstanding the strong binocular magnification the surface here appears almost smooth.

With regard to the appearance of the stroma it was stated that in the pupillary part it was best developed, while in the intermediary

FIG. 10.

A drawing of the posterior surface of a normal iris, from a girl of 17, made under bright light and binocular microscope ($\times$ about 12). The structural radial folds have the same character in the normal iris of a man of 65.

part of the iris it presented a peculiar appearance, resembling the foetal mucoid (or gelatinous) tissue. In front of the sphincter the connective tissue contained an abundance of fixed cells, but there were no signs of inflammatory conditions in it; several cells were here pigmented, some of them "clump cells"—in vivo this region was sienna yellow. Behind the sphincter the tissue had a more sclerotic character with fewer cells and more numerous fibrils. In the mucoid stroma of the middle and peripheral part of the iris a peculiar ground substance was seen and in this scarce fibrils and cells of which very few were pigmented; in vivo this region had a blue slate colour. There was nothing remarkable about the stroma cells as regards their shape; in the Heidenhain coloured sections there were a good many large eosin stained cells with numerous dark granules in the protoplasm. The blood-vessels, too, presented no peculiarities.

We have thus shown that in Anna B.'s eyes the dilatator pupillae is defective in its development, and we must assume that the anomaly is due to an inhibition that has asserted itself in embryonal life. This inhibition has not affected that part of the dilatator that lies behind the sphincter, for this part is developed normally. We have, then, all the developmental stages from this normal condition

FIG. 11.

A general view of a radial section through the iris of Axel B (Case C). Short and long connecting muscular fibres are seen to go from the sphincter to the dilatator, which is well developed not only behind the sphincter but in most places, also in the intermediary part of the iris, where it ends in a thickening with muscular fibres unravelled and projected into the stroma (compare Fig. 14). In the thin periphery (compare Fig. 12 and Fig. 13) no Bruch's membrane or muscle cells are seen.

behind the sphincter down to the complete inhibition represented by pure epithelial cells in the intermediary and peripheral parts of the iris.

Case C (Axel B.).—Here also the microscopical examination of the sections revealed no difference between the two irides. But the dilatator muscle is better developed here than in the irides of the

FIG. 12.

Microphotograph (about $\times 13$) of a radial section through the iris of Axel B. The thin dilatator-free part of the iris is here $1/5$ th of the distance between the pupillary and the ciliary border.

twin-sister Anna (Case *b*) ; as twins of different sex they originate from different ova.

In Axel B.'s irides the dilatator is well developed not only behind the sphincter, but also in the intermediary zone. The dilatator muscle fails only in the periphery ; this dilatator-free zone may in many places be measured to be $1/10$ or $1/8$ of the distance between the pupillary and the ciliary border (Fig. 11) ; in other places to be $1/5$ (Fig. 12, enlarged in Fig. 13).

Between the intermediary and the peripheral zone the iris forms a thickening in which the dilatator ends (Figs. 11, 12, 13 and 14).

In the peri-pupillary region the dilatator receives short muscular connecting fibres from the sphincter, and in the intermediary part long ones (Fig. 11). In the intermediary region Bruch's

FIG. 13.

Enlarged microphotograph (about $\times 45$) of the thickening and thin dilatator-free iris periphery in Fig. 12.

FIG. 14.

Microphotograph ($\times$ about 150) from a radial section of the iris of Axel B. through the thickening in the intermediary part where the dilatator ends unraveled in muscular fibres, which are projected into the stroma diverging in all directions.

membrane is clearly seen also without depigmentation, but in depigmented sections usually small spots are seen in which the dilatator is completely lacking—a finding known also in the normal iris (von Szily). The front layer of the epithelium is here in immediate contact with the stroma, which—as in the thin dilatator-free periphery—has an extraordinary lax structure like foetal mucoid

tissue. But in most places the stroma is normal and contains pigmented cells of which several are "clump cells"; in vivo the whole iris had a cinnamon brown colour. In the stroma in front of the sphincter there are perhaps more fixed cells and behind the sphincter perhaps more fibrils than in a normal iris, but nowhere

FIG. 15.

A drawing of the posterior surface of the iris of Axel B. made under bright light and binocular microscope ($\times$ about 13). Radial structural folds are seen, but in less pronounced relief than in a normal iris (Fig. 10). During the fixation in Bouin's fluid the lens capsule became partly adherent to the pigment epithelium, which during the preparation followed the removed capsule in some places (the light spots in the drawing).

(To colleagues desiring more details of the microscopical anatomy of the iris in congenital miosis, on application to the authors, reprints of Lit. 16 (and later of Lit. 18), which contains more text, more microphotographs, and several drawings reproduced in four colour print, may be sent.—Figs. 4, 5, 9, 10, and 15 in this article are printed with blocks chosen among those made for four colour print in Lit. 16 and 18).

signs of or sequelae of inflammation. In the above-named thickening the stroma is richer in fibres and is more red stained with eosin than elsewhere in the intermediary zone; the dilatator is unravelled here in fine muscular fibres which are projected into the stroma diverging in all directions (Fig. 14).

The epithelial layers have also an increased volume (Fig. 14) on the posterior surface of this thickening which is found in most of the section series; but in some series the thickening is completely

lacking and here the dilatator-free part advances much more towards the pupil than elsewhere.

When one sees the sections of the thin dilatator-free iris periphery, one receives the impression that this peripheral part has been strongly stretched by the continuous contraction of the sphincter. One might be tempted to believe that the above-named thickening may be the dilatator's normal site of origin, which has been loosened from the ciliary region. In opposition to this surmise is the fact that the muscle fibres are unravelled from the dilatator end and projected in all directions into the stroma here. Some epithelial processes into this stroma point towards the periphery. Also in the iris of the twin sister Anna (Case *b*), a few similar thickenings in the intermediary part are seen. With both twins a congenital inhibition of the dilatator development must be presumed.

According to the better development of the dilatator the posterior surface of the iris of Axel B. is less smooth than that of his twin sister Anna (Fig. 9), the posterior surface of his iris shows radial structural folds (Fig. 15), although in less strong relief than in a normal iris (Fig. 10).

Part III.—Epicrisis.—By S. HOLTH

From my clinical observations I drew the conclusion that these congenital pinhole pupils could not be explained by a primary spasm of the sphincter muscle which nearly always—as after an instillation of miotics—is accompanied by spasm of the ciliary muscle. But spasm of accommodation was only found in one of the three patients, the still living Ingeborg B. (Case *a*), whose static myopia is of a low degree; the permanent spasm of accommodation is observed to cause a considerable increase of her myopia even in her forty-fifth year. This spasm of accommodation is accompanied by frontal and occipital headaches, which always disappear after a local application of mydriatics to the eyes. The patient has till now never suffered from any disease of the central nervous system; I believe therefore that this spasm of the ciliary muscle must be considered to be due to a kind of irradiation from the m. sphincter pupillae, which having no antagonist is always contracted. But the reason why only Miss Ingeborg suffers from spasm of accommodation and never her emmetropic brother Axel or her strongly myopic sister Anna, I confess I cannot explain.

Another explanation of miosis is paralysis of the cervical sympathetic nerve: but my patients had no sign of this; e.g., no ptosis; furthermore the application of cocain produced a slight exophthalmus with enlargement of the palpebral fissures. The sympathetic miosis, however, is nearly always unilateral and never shows the pupil so narrow as a pinhole. For these reasons I believed the most natural explanation of the lack of dilatator

muscle from birth, to be that the *dilatator muscle itself* was *not developed or very incompletely so*. I think that Prosector O. Berner's fine preparations give decisive evidence of the truth of this surmise.

I will, however, draw attention to some more peculiarities in the iris of the four anatomically examined eyes with congenital miosis. In the dilatator-free parts of the iris the stroma has the character of foetal mucoid or gelatinous tissue. This is also the case with the thin peripheral dilatator-free part of the iris of Axel B. (Fig. 13), although the dilatator is well developed in the intermediary part, the muscle being destitute of peripheral anchorage—in other words, having no functional origin—cannot act as the sphincter's antagonist. In the normal iris the stroma is much denser in the peripheral than in the intermediary zone.

In congenital miosis the stroma in the sphincter region, too, may perhaps be said to have a peculiar character: in front of the sphincter perhaps some more fixed cells than in the normal iris at different ages, and behind the sphincter perhaps some more fibres. The deviations of the pupils from the round form in cases (b) and (c)—compare Figs. 4 and 5—may perhaps be explained by meridional variations of the elasticity of the peripupillary stroma. These conditions are, however, difficult to interpret, because also in the normal iris the peripupillary as well as the peripheral stroma is denser than the intermediary stroma. At any rate the qualities of the stroma must be much less important for the explanation of the congenital miosis than the discovered considerable deficiencies in the dilatator muscle.

Finally I wish to say that the use or the elimination of the accommodation does not seem to have influenced the static refraction of the six clinically examined eyes.

In Case (a) Ingeborg B., in spite of continuous spasm of accommodation since her early childhood, the static myopia was very moderate at the age of 21, viz.—2.75 and—1.25 D. The diminution of the myopia at the age of 43, to—1.5 and—0.75 D, was, in my opinion, not caused by the mydriatic elimination of the accommodation during 22 years; I believe that the refraction of the lens itself is slightly decreased with age. During these 22 years she has never used convex glasses for close work because she can easily read without glasses Snellen 0.5 at distances between 20 and 50 centimeters (narrow pupils: small circles of diffusion). From the age of 21 she has had daily instillations of mydriatics; before that time she had been obliged to converge strongly during reading and close work because her myopia, by spasm of accommodation, was increased to—10 or—12 D.

In Case (b) Anna B. I would repeat that the patient never suffered from spasm of accommodation, and her myopia at the age of thirty

years was -7 and -8 D, and was increased at the age of fifty-two, to -15 and -16 D in spite of daily mydriatic elimination of the accommodation during 22 years. She could never be persuaded to use strong or weak concave glasses, and did much reading and fine close work under strong convergence and with bent head.

Anna's emmetropic twin brother Axel B. (Case c) was until his thirtieth year a country gentleman and did not do much reading or close work during his youth; he never suffered from spasm of accommodation.

Treatment of Congenital Miosis

Presbyopes with exclusively indoor life may prefer no treatment at all [e.g. Case (c) Axel B.]: they can read for a long time without glasses. But in outdoor life the twilight blindness is very embarrassing and can be relieved in two ways.

1. By daily application of mydriatics to the eyes, when myopic, as long as the pupils by this means can be dilated to 1.5 or 2 mm. diameter; this treatment is also indicated in other refractions when the miosis is complicated by spasm of accommodation. It appears, however, that a local habitude to or resistance against mydriatics develops after a lapse of many years, so that their dilating power on the pupil gradually decreases [Cases (a) and (b)]. It is scarcely age which affects this, for in Case (c) Axel B. at the age of 52, a few instillations of 1 per cent. homatropin dilated the pupils to 2 and 2.5 mm. as other mydriatics did with his sisters at the age of from twenty-one to thirty years.

2. By optical *iridectomy*—as a rule upwards and not broad (vertical application of the blades of the iris scissors); indicated in all refraction conditions where the accommodation should be left intact, but also if mydriatics appear to be injurious, e.g. by increasing the tension (which I have not seen in my cases). Iridectomy may also prove necessary in elderly—mainly myopic—persons, when the dilating effects of mydriatics after daily applications during many years are at last insufficient or nil (Case c).

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BINOCULAR CHOROIDAL TUBERCULOSIS WITH DETACHMENT OF THE RETINA IN TWO KITTENS.*

ADDENDUM

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DR. STANLEY GRIFFITHS reports on his bacteriological investigation of specimens from the second kitten as follows:—

"An emulsion of the mesenteric gland produced general tuberculosis in a rabbit, and cultures obtained from the lesions of the cat were typically bovine in cultural characteristics.

"Undoubtedly the infecting tubercle bacilli are bovine in type."

* See BRITISH JOURNAL OF OPHTHALMOLOGY, July, 1923, p. 305.