REMARKS

ON

OCULAR SYMPTOMS OF GENERAL PARALYSIS
OF THE INSANE, WITH SPECIAL REFERENCE
TO ITS CLINICAL GROUPINGS.

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THE following remarks are devoted to the consideration of the more natural clinical groupings of that intractable disease, general or progressive paralysis of the insane. We have here in the oculo-motor anomalies the bulbar and spinal symptoms, and in the indications of cortical implication a very complex symptomatology presented, the mutual dependence of which symptoms demands a very close inquiry ere anything like a clear insight can be gained of the disease as a whole, or of the several natural groups constituted by the mode of evolution and march of symptoms.

The oculo-motor as the more constant group of symptoms will claim special attention here; and a preliminary view of the nature of such anomalies and the sources of fallacy involved in their investigation may not be out of place ere we attempt the synthesis of morbid phenomena constituting the distinctive features of the several groups.¹

The reflex loop for the contraction of the sphincter of the pupil is constituted by the retina, optic nerve and tract, and Meynert's tract connecting the latter with the centre as the incident, and the third nerve as excitator or motor. The centre is constituted by the motor oculi nucleus lying along the floor of the Sylvian aqueduct, and the short ciliary nerves emerging from the lenticular ganglion are regarded as the myotic fibres emanating from this centrum (Gaskell, Jessop). The dilatation of the pupil is effected through the agency of a far more extensive tract. As incident, we have all the sensory nerves from the fifth cranial to the last spinal. The centre for dilatation is, according to Hensen and Voelckers, just external to the nucleus of the third nerve beneath the corpus quadrigeminum. The motor fibres from this centre pass down to the cilio-spinal region of Budge,² emerge with the anterior roots of the second dorsal nerve (Gaskell)³ to join the cervical sympathetic, and, losing their investing medullated sheath in the superior cervical ganglion, pass on to the cavernous plexus, Gasserian ganglion, ophthalmic division of fifth, its nasal branch, and, finally, as the long ciliary nerves are distributed to the iris as its dilating or mydriatic fibres (Gaskell).

The existence of radiating dilating muscular fibres for the iris in man is not now admitted, although in certain animals its existence has been confirmed, for instance, in the cat (Heese),⁴ in birds, the otter, and the rabbit. Iwanoff's assumed dilator iridis has been amply disproved by Schwalbe, Fuchs, Jessop, and others. Some attribute the dilatation entirely to vasomotor influence, inducing a contraction of the iridal blood vessels (Grünhagen); others regard the phenomenon as due to muscular and vasomotor fibres, whilst Gaskell in great measure attributes it to inhibition of the sphincter iridis through the agency of the sympathetic.⁵ His views point to the existence of none but radiating elastic fibres, in the only portion of the iris free from folds, and this phenomenon is, he believes, entirely due to this elastic agency during relaxation of the sphincter pupillæ. Gaskell regards the fibres in the cervical sympathetic as purely inhibitory, and terms this nerve the "cervical splanchnic."⁶

¹ On the Peculiar Proclivity of the Oculo-motor Nerve to Paralysis. See Dalchow: Die Aetiologie und ätiologische Diagnostik der Oculomotorius-paralyse mit Berücksichtigung der pathologischen Anatomie. *Zeitschr. f. klin. Medicin.*, 1893, Bd. xxii.

² Ueber die Bewegungen der Iris, 1855.

³ *Jour. of Phys.*, vii, 1, p. 38, On the Structure, Distribution, and Function of Visceral Nerves.

⁴ Ueber der Einfluss des Sympathicus a. d. Auge, *Pflüger's Arch.*, Bd. lii. 5 *Op. cit.*

⁶ See also Critical Digest, by Dr. Anderson, *Brain*, vol. xi, p. 528; and Review by Hale White, *Brain*, vol. ix, p. 271.

The original investigations of Hensen and Voelckers⁷ in dogs, indicating that the contraction of the ciliary muscle for accommodation is effected through the most anterior group of cells in the oculo-motor nucleus; and the contraction of the sphincter iridis by those immediately posterior to this group have been with certain modifications confirmed by successive observers; and several important additions to our knowledge of the complex structure of this nucleus have been made by Edinger,⁸ Westphal, Von Gudden, Bruce, and Van Gehuchten. The constitution as given by Van Gehuchten, following Perlia, exhibits fifteen nuclei, of which one is central and seven others arranged laterally on either side. The original article by Van Gehuchten well repays perusal.⁹

PUPILLARY ANOMALIES.

The excessive sensibility of the retino-iridal apparatus to reaction under stimulation renders the size of the pupil and behaviour of the iris one of the most valuable indications of centric nervous disturbance; and when such reflex adjustments are at fault it becomes essential in the first place to decide how far such is due to centric or eccentric agency. Inequality of the pupils may in itself be a sign of but small moment, as being due to eccentric cause, as in adhesions of the iris to the lens capsule, or from the accidental or intentional instillation of myotics or mydriatics; or, on the other hand, it may be a sign of great import, as indicating not alone a centric cortical disturbance, but as having a localising value in indicating the hemisphere at fault.

After eliminating possible fallacies due to eccentric agencies, we have first to decide which of the pupils—the larger or the smaller—indicates the eye affected; and this is by no means so easy a matter in all cases as it at first might appear. This is mainly due to the fact that pupillary inequality is by no means necessarily associated with defective reaction. If, however, with unequal pupils, we note that either the larger or the smaller present a sluggish, impaired, or totally abolished reaction to the normal stimuli, we naturally infer that this is the affected organ; it is therefore always necessary to examine systematically the reaction to light where pupillary inequality presents itself. A few remarks on the functional activity of the iris will render our subject clearer.

(a) *Rhythmic Oscillation.*—If the eyes of a healthy individual are directed in diffused daylight towards a distant point, with accommodation thus at rest, we observe a constant slight oscillation of the iris betwixt dilatation and contraction corresponding to the respiratory rhythm and undoubtedly dependent thereupon. Each inspiration is accompanied by a pupillary dilatation—each expiration by a corresponding contraction, immediately effected by the varying fulness of the iridal blood vessels dependent on the changes in blood pressure in the organism at large. This phenomenon is not of much clinical significance, but must be recognised for purposes of elimination when studying the more important pupillary movements.

(b) *Muscular Movements.*—If now under similar conditions the subject be requested to poise himself on his toes, to flourish his arms, or otherwise bring a complex musculature into activity, a notable dilatation of the pupil ensues, due also in part to variations in blood pressure, but also in part probably to the direct effect of motor stimuli from the cortex calling such musculatures into activity. This likewise is of minor clinical import, but must be borne in mind for similar reasons with the dilatation of respiratory rhythm—obviously so in attaching significance to pupillary dilatation in genuine and feigned epilepsy.

(c) *Sensory Stimulation.*—In the next place, if we gently stroke the face, stimulate by tickling, by the prick of a pin, or by the electric brush, any cutaneous surface of the body supplied by any sensory nerve from the fifth to the last spinal, we shall find a well-marked dilatation of the pupil ensues, varying with the intensity of the stimulus applied. Moreover, if we shout in our patient's ear or otherwise stimulate the acoustic nerve, even under the influence of chloroform, a similar iridal reaction takes place. Hence every

⁷ *Graefes Archiv*, Bd. xxiv, 1878.

⁸ See Diagrams in *Quain's Anatomy*, 10th Edition, vol. iii, pt. 1, p. 99.
⁹ D: l'Origine du Nerf Oculo-Moteur Commun. Par G. Van Gehuchten. *La Cécité*, Tome viii, Fasc 12, 1892.

acoustic, tactile, painful, and electric stimulus entails a dilated pupil. One obvious implication from such observations is that the pupil-dilating centre must be in connection with widely distributed and diverse sensory mechanisms. With respect to this reaction, the "reflex dilatation" of Erb, it is important to note that sensation need not actually follow the stimulation, for in stimulation of the anæsthetic skin of hemianæsthetic subjects, and also in sleep and coma, the same reaction may be obtained. Moeli states that the reaction is not so marked in women over 50 years of age, nor at all elicited except by powerful stimuli in men over 50.¹⁰ I have indicated elsewhere¹¹ the failure of this reaction in cases of general paralysis and epilepsy to the extent of about 36 per cent. in the former and 24 per cent. in the latter, but, taking the results of later examinations into account, I find the percentage of loss of reflex pupillary dilatation in all cases of general paralysis is for male subjects nearer 43 per cent. Loss of the light reflex is not necessarily associated with failure of reflex dilatation on stimulating the skin, as we shall see later on. Gowers believes that when the pupils are not small the "skin reflex" persists, and when they are small it is lost.¹²

(d) *Emotional or Psychical States.*—If our subject be a specially sensitive individual the mere attempt to use the needle or electric brush will probably be preceded by a notable dilatation due to his timidity; in fact, any strong emotion—fear, rage, or joy—will induce extremes of dilatation, just as intensity of pain will induce the same. Hence we may say that voluminous psychical changes (emotional) are likewise associated with dilatation of the pupil. Epileptics notably present this feature, and the phenomenon is of importance clinically, since it may mislead us when examining the reactions in cases of insanity, and especially epilepsy, in which the subjects show much emotional instability.

There can be little doubt that this dilatation accompanying emotional disturbance is due to the agency of the sympathetic—in fact, to stimulation of the vaso-constrictor fibres of the iris; yet it should be noted that it is present when by disease of the cilio-spinal tract no response can be obtained to the usual cutaneous stimulation. In like manner when the pupils fail to dilate on shading the "psychical" reaction may still be obtained. Thus in one illustrative case both pupils measured 4.5 mm.; the right was very sluggish to light, the left normal; dilatation on shading and cutaneous stimulation was absent in both, but present in excess during emotional states. Thus in moderately dilated pupils shading and cutaneous stimulation produced none or next to no change, whereas the dilatation was excessive upon emotional disturbance. Ordinary sensory dilatation (on cutaneous stimulation) does ensue even when the cervical sympathetic is divided, as shown by Vulpian, and this clearly indicates that the cilio-spinal and sympathetic tract is not the only one by which stimuli inciting to dilatation may be conducted. It is therefore probable that the psychical dilatation referred to is induced through a higher centric tract.

(e) *Morbid Oscillations.*—We must not mistake the respiratory oscillations for one which is of much greater import—the so-called "hippus." Here dilatation and contraction, but of much larger range, occur rhythmically, but not in unison with the respiratory movements. It is found in cases of serious cortical implication, such as general paralysis, and probably depends upon circulatory disturbances in the cortical blood vessels. Thus, in a female aged 32, suffering from general paralysis, both pupils being equal, somewhat dilated but neither fixed to light, with accommodation relaxed, a wide rhythmic dilatation, and return to former size of the right pupil, occurs without any corresponding movement of the left.

(f) *Consensual Movements.*—With accommodation at rest upon alternately shading and exposing one eye, both pupils are observed to dilate and then contract—the reaction in the eye not shaded being spoken of as the consensual reflex; the reaction in the eye alternately shaded and exposed being the direct light reflex. Since however, the contraction and dilatation are induced through two distinct motor channels—that

of the third nerve and sympathetic respectively, it is convenient to distinguish these movements as:

- Indirect light reflex—(A) Consensual dilatation.
(B) Consensual contraction.
Direct light reflex—(A) Direct reflex dilatation.
(B) Direct reflex contraction.

Dealing with the consensual first, it must be assumed that the channel for excitation of the third nerve nucleus will comprise not alone the fibres crossing at the optic chiasma, but the direct fibres of the optic nerve passing from the excited retina down the tract of its own side. (Knoll, Bechterew, Erb, Baumgarten.) This may safely be inferred, since in lesions of one tract, resulting in homonymous hemianopsia, the consensual reaction can still be obtained in both eyes. Thus in a lesion of the left optic tract excitation of the right eye still induces contraction of the left pupil, which would not occur if the current passed along the commissural crossing only, and not, as is the case, by the medium of the right tract, whence it reaches the motor oculi of the left eye through commissural fibres connecting the nuclei of both sides. By this mechanism it is readily seen that lesion of the optic tract of one side does not abolish the consensual or the direct light reflex. Again, the consensual dilatation in normal states is equal to the direct reflex dilatation, both shaded and uncovered eye having an equally dilated pupil; for just as much as the shaded eye lessens the contraction of the exposed eye, so much does the exposure of the latter diminish the resultant dilatation of its fellow.¹³

I have occasionally heard a question raised as to the actual inequality of the pupils, on the ground that one eye was shaded and the other illumined; whereas, in healthy states, these conditions would not result in inequality.

The conjoint operation of consensual and direct excitation of both eyes admits of a far greater range of movement than is obtained by either separately. Thus the moderate dilatation of pupil obtained by shading one eye is greatly augmented upon shading both, and the same statement applies to the contraction upon exposure. It is important to remember this, since, in testing the integrity of the sympathetic dilating fibres, the consensual dilatation should not be alone relied upon, but the more notable effect upon shading and uncovering both eyes simultaneously.

If a paralysing lesion affects the sympathetic fibres of one side, three important signs will indicate the fact:

- (a) Upon shading the eye of the sound side no consensual dilatation of its fellow is induced.
(b) Upon shading the eye on the affected side no direct reflex dilatation is induced, although its fellow dilates consensually.
(c) Upon shading both eyes a marked contrast occurs. On the affected side the pupils remain motionless, on the sound side a wide dilatation ensues.

In short, unilateral sympathetic lesions are indicated by a failure of the affected eye to dilate consensually or directly, and by a marked contrast in the two upon shading both pupils. The optic nerve comprises, according to Gudden, two distinct varieties of nerve fibre, which are recognised microscopically. The finer of the two subserve vision, the coarser conduct impressions back to the central nuclei, subserving the reflex movements of the pupils.¹⁴ These latter are more resistant to pathological changes than the visual series, which are often atrophied when the pupillary fibres remain intact.

In serious lesions, however, in front of the chiasma, either retinal or affecting one optic nerve and entailing monocular blindness, we find:

- (1) Failure to contract or dilate on direct light stimulation.
(2) Failure to excite its fellow consensually.
(3) Its own consensual reaction follows excitation of the sound eye.
(4) No marked contrast occurs on shading both eyes as in sympathetic lesions (*v. supra*).

(g) *Direct Light Reflex.*—From what has already been stated it will be understood that we cannot absolutely exclude the operation of consensual influence when testing the eye for

¹³ As to time element involved in consensual movements, see Donders, *Accommodation and Refraction of the Eye*, *Syd. Soc.*, p. 573.

¹⁴ See also Heddeus in *Abstract. Ophth. Rev.*, June, 1887. Gudden even states that the pupillary reactions remain unaffected by the ablation of the nates.

¹⁰ *Archiv. f. Psych.*, Bd. xiii.

¹¹ *West Riding Asylum Medical Reports*, vol. vi, p. 140.

¹² *Diseases of the Nervous System*, vol. ii, p. 175.

direct light reflex—the latter always includes a consensual element—for where the eye not under examination be shaded or exposed, its excitation must affect and modify the movement of its fellow. This, however, as an invariable rule applies only to healthy states; for when the sympathetic cilio-spinal fibres or the nucleus of the third nerve are implicated unilaterally the result is necessarily greatly altered.

A moment's consideration will indicate that in its consensual influence each eye possesses an element of antagonism to its own over-action under direct light excitation. Thus excitation of the left eye being followed by contraction of the right lessens the consensual influence of that eye, and hence tends to restrict its over-contraction. If the right pupil through lesion of the motor oculi nucleus had not contracted, then its larger range of retinal excitability would tend still further to intensify the contraction of the left pupil. Again, if the left eye be shaded the dilatation of the right pupil tends consensually to oppose excessive dilatation of the left; if the right pupil had remained immobile from lesion of the cilio-spinal fibres, a more extensive dilatation of the left pupil might be anticipated. If I am correct in so concluding, then clinically we should expect all cases of unilateral implication of the third nerve nucleus or of the sympathetic would entail beyond the defective or abolished reaction of the affected eye an exaggerated reaction of the sound organ.

The more satisfactory method of examining the pupillary reactions is as follows: Place the patient before a window in diffused daylight, with the eyes directed at a distant object; or, to ensure a standard illumination, he may be seated at one end of the darkened ophthalmoscopic room, the further wall of which is illumined by the reflecting lamp. The pupils are closely observed, and note made as to: (a) Any irregularity of contour; (b) inequality in size; (c) activity of ordinary rhythmic movements; (d) presence of "hippus"; (e) non-existence of adhesions to lens. The dimensions of the pupils should now be carefully measured by pupilometer. Covering the left pupil with one hand we observe the degree and activity of dilatation in the right eye, and after a few seconds we remove our hand and observe in like manner the range and activity of contraction of the right pupil. Similarly we test the consensual reaction in the left eye. The left eye is again shaded, and its dilatation noted; the organ is uncovered and its contraction likewise observed, being careful to note that no convergence occurs indicating an effort of accommodation. The consensual and direct reflex are now carefully noted by some prearranged system of symbols. Both eyes should next be simultaneously covered and exposed in rapid succession, and the activity and range of movement noted.

If we find the direct reflex impaired in vigour and sluggish, it will be well to test the same by concentrated light through a bull's-eye condenser, which will often induce distinct contraction when the pupil under less excitation appeared fixed to light. In certain cases of general paralysis the initial contraction thus induced may be succeeded by a secondary dilatation—the pupil expanding widely in spite of the concentrated illumination. This, as I have elsewhere indicated, is often seen (13.6 per cent. of all cases of general paralysis) as one of the earliest signs of a failing motor oculi nucleus—the centric expansions of the third nerve being readily exhausted.¹⁵

(h) *Accommodative Movements of Pupil.*—The subject is placed in a moderate light with the eyes fixed on a distant point, and with the accommodation thus relaxed the size of the pupil is noted. He is then requested to direct the eyes at the tip of the finger, held a few inches in front of the bridge of his nose, and the range and vigour of contraction noted, or any unilateral or bilateral defect estimated. This contraction of the pupil is well known to be much more intimately associated with the convergence of the eyeballs than with the act of accommodation itself, for where accommodation occurs without convergence we get no contraction of pupil; but where accommodation does not accompany convergence we still have the pupillary contraction.¹⁶

Having thus passed in rapid review the motor phenomena presented by the iris, and requiring careful observation in

cases of suspected cerebro-spinal disease, let us now consider the significance of unequal pupils, and the relationship of inequality to morbid pupillary reaction as illustrated by a series of cases of general paralysis.

(1) *Inequality with Normal Reaction.*—Cases presenting this condition of simple inequality fall naturally into two categories: (a) Inequality of transient duration due to temporary cortical disturbances, such as epileptiform discharges, apopleptic seizures, the onset of certain paralytic lesions. (b) Inequality of more persistent character due to grave disease of the brain; to the pressure effects of fluids, growths, etc., or to the cortical implications of inflammatory and degenerative affections, such as those of general paralysis.

I endeavoured to indicate in an article published in 1876 that the more transient forms of inequality were not only due to cortical discharges, but had a localising significance of some value;¹⁷ but I did not at that time appreciate a still more important fact which has since been impressed on my mind—namely, that these transient conditions were often the precursors and earlier stage of the more persistent inequality, and that these latter more established conditions must be attributable not to bulbar and spinal but to cortical implications where the pupil reflex was not involved. It is now clear to my mind that we have every reason for regarding the early pupillary inequality as an indication in these cases of general paralysis of cortical lesion, whilst the defective iridal reaction is due to implication of cilio-spinal and bulbar territories. But here again we must distinguish betwixt early and late inequality, the latter being due to the advancing lesions of spinal and bulbar arrangements. In the article above referred to dealing with discharging and destroying lesions of the cerebrum in epilepsy, chronic brain atrophy, and general paralysis, it was shown that out of four-and-twenty such instances, there was but one exception to the rule that the larger pupil observed prior to the seizure was always on the side of the lesion. During the attack, or immediately following thereupon, these conditions are often, though temporarily, reversed. In all such instances the reactions to retinal and sympathetic stimuli were preserved.

From an important series of observations Mr. Jonathan Hutchinson, jun., deduces the following facts:—¹⁸

1. In cases of concussion ... (a) Pupils respond slowly to light, and there is no marked myosis or mydriasis.
(b) In a small proportion a temporary unilateral or bilateral mydriasis ensues.
2. Inflammation following severe injuries to brain... Myosis usually present.
3. Compression from meningeal hæmorrhage ... Mydriasis on side of lesion in at least half the cases; double mydriasis next in frequency, and myosis very rare.

If, as suggested, the early inequality of pupils presented in general paralysis indicates the cortical implication, it becomes of interest to inquire in what proportion of cases do we find this pupillary inequality preceding any notable defect in the reflex adjustments of the medulla? It is unquestionable that in a certain proportion of cases such inequality does precede any implication of the ocular reflexes;¹⁹ thus out of 147 cases examined, 71 patients showed the direct light reflex intact, or at the worst merely sluggish; and of the latter as many as 33 exhibited a notable inequality of pupils. Again, it was noted that of the 73 instances where consensual and direct light reflex in both eyes were absolutely abolished, 34 presented an inequality of pupils. Thus the proportion in both cases is such as to indicate no relationship betwixt pupillary inequality and the efficiency of the iridal reactions respectively, since the unequal pupil is found as frequently before as subsequent to the affection of these reactions, as indicated in the following scheme:

Light reflexes intact ...	71	Pupils unequal ...	33
Light reflexes abolished ...	73	Pupils unequal ...	34
	144		67

Taking all cases alike into consideration, we find 67

¹⁷ *West Riding Asylum Reports*, vol. vi. See also an admirable summary, *The Pupil in Health and Disease*, in Swanzy's *Handbook of Diseases of the Eye*, third edition.

¹⁸ *Ophth. Rev.*, vol. vi, pp. 97, 120, 153.

¹⁹ The most diverse opinions have been expressed as to the relative frequency of pupil inequality in health. Dr. Reche concludes that it may be ignored. See *Deut. med. Woch.*, 1893, No. 13.

¹⁵ *Text-book of Mental Diseases*, p. 274. W. Bevan Lewis.

¹⁶ De Ruiter and Cramer; see also Donders, *loc. cit.*, p. 574.

instances of unequal pupils in a total of 144 subjects examined, and of these the left pupil was the larger in 35 and the right pupil in 32 cases.

(2) *Defective Reaction*.—It was apparent, from an analysis of 100 of the above cases, that in the large proportion of 31 the consensual and direct light reflex were absent from the first observation in both eyes, but that a later complete loss of these reactions occurred in subsequent stages of the disease in a further series (11), whilst in a small minority (4) unilateral failure of these reactions prevailed. A still further series (24) revealed a very sluggish or doubtful reaction, whilst in the remaining 30 normal response was elicited both by consensual and direct light stimuli. For the convenience of study it is well to apply to the varied reactions presented by the iris to stimulation under conditions of disease a definite phraseology, and the one adopted here is as follows:

Consensual Failure:
Partial reflex iridoplegia ²⁰ :	1. Loss of light reflex.
2. Loss of reflex dilatation, "skin reflex," shading.
Complete reflex iridoplegia:
Associative iridoplegia:	3. Both conjoined.
4. Loss of contraction upon convergence only.
Complete iridoplegia:
Cycloplegic iridoplegia ²¹ :	5. All the above conjoined.
6. Loss of accommodation superadded.

In the examination of the patients studied—147 in all—it was very evident that they fell naturally into five self-constituted groups, characterised by the mode of onset, grouping of symptoms, and subsequent course and termination of their disease. It is not thought necessary here to reproduce the tabulated series, since the object desired is to indicate to the reader the chief features presented by each group. Each group was examined (1) for visual and oculo-motor symptoms; (2) for bulbar and cerebral symptoms; (3) for spinal symptoms; (4) for the prevailing mental symptom.

FIRST GROUP.

The first group to which I would draw attention is remarkably well defined both in its clinical and pathological aspects; the symptoms characterising this division are notably constant, and appear to stand in direct relationship with the ascertained morbid changes of the central nervous system presented on death.

Taking the oculo-motor symptoms first, we find a moderately large pupil steadily becoming more and more dilated, usually involving both pupils, but very rarely of unilateral implication. On examining the iridal movements, failure of consensual and direct light reflex will be recognised without implication of the movements associated with accommodation; in other words, reflex iridoplegia or the "Argyll-Robertson symptom." The signs clearly indicate a paralytic implication of the retino-iridal arc, not in its sensory, but in its motor segment or nucleus (third nerve). It has been assumed that the failure to respond to light (whilst contraction upon convergence is still preserved) necessarily implies lesion of Meynert's fibres, which connect the optic termini in the quadrigeminal bodies with the nucleus of the third nerve, and were the defect bilateral then a bilateral implication of this tract might explain the phenomena. Often, however, we find the defect unilateral, and upon the assumption that an intranuclear connection exists betwixt the two third nerve centres this explanation obviously must be incorrect. This intranuclear connection undoubtedly exists, and is the tract whereby consensual movements are still preserved in unilateral lesions of the optic tract (*vide supra*).

In the series of cases now under examination eleven presented unilateral failure to light without impairment of the accommodative contraction, and clearly therefore we are driven to the conclusion that the motor segment or nucleus must be the affected region. Since this unilateral failure often precedes the failure on both sides for some time, there is no reason to assume that Meynert's tract is involved in the latter case any more than in the former.

The gradually advancing mydriasis is fully accounted for by the unopposed action of the sympathetic, and the dimensions of the pupil form a gauge of the sensory and psychical stimuli affecting the iridal-dilating mechanism. Shading the eyes no longer produces dilatation, since the third nerve

nucleus being involved the exclusion of retinal excitation can no longer inhibit as heretofore. To indicate, however, that the cilio-spinal tract is free cutaneous stimulation may be applied when the pupils further dilate. Occasionally such response is not obtained on cutaneous stimulation, and we then discover, on exploration, that common and other forms of sensibility are considerably impaired.

We find certain exceptional cases occur in this group where the mydriasis cannot with justice be wholly assigned to a lesion of the third nerve nucleus. In these cases the pupils are of moderate size or somewhat larger than usual, but their reaction to light and to shading and to all other forms of sensory stimulation may be either extremely sluggish or altogether abolished, a large size fixed pupil being the feature.

It would appear that this can only result from a paralytic implication of both the sympathetic and third nerve supply, a genuine iridoplegia or paralysis of circular fibres and dilating mechanism resulting. On the other hand, a distinctly mydriatic pupil failing to react to light may also fail to dilate upon shading, although to cutaneous stimulation, to co-ordinate movements of the limbs, and to psychical stimuli the normal response is obtained. In these latter cases the failure to dilate on shading as before indicated is obviously due to interruption of the retino-iridal arc, the third nerve nucleus being paralysed, the shading of the eye no more than its brighter illumination produces any result.

It is different in the case of the small or moderate size pupil due to paralysis of the sympathetic; here the pupil, whilst reacting to light might *a priori* be imagined to fail in dilatation when shaded. This, however, is not the case—the movement may be sluggish, but distinct slight dilatation is perceived, not however due, I take it, to sympathetic influence, but to the differential betwixt a greater and lesser light stimulus affecting the intact retino-iridal arc. These features, therefore, especially indicate the importance of cutaneous stimulation as a test of the integrity of the vaso-motor iridal innervation.²² As an instance of this paralytic mydriasis with sluggish or immobile pupils the following case may be cited: Where with moderately large pupils, in which the right fails to react to light but contracts on convergence, no dilatation occurs upon shading, but normal action of the sympathetic is at once elicited upon sensory stimulation and co-ordinate movement, the visual acuity being normal.

Exalted Spinal Reflexes.—The paralytic mydriasis is associated sooner or later with unduly exalted deep reflexes. The knee-jerk is frequently much exaggerated, but ankle clonus is an infrequent accompaniment, nor do other spinal symptoms betray themselves until much later on in the history of the disease. The exalted knee-jerk is not a sequel to convulsive seizures in this group, as it is in other forms alluded to later on, but appears due to a constant suspension of cerebral control, possibly associated with the slow degenerative changes of the nature of a fine descending sclerosis in the lateral columns of the cord; anyhow, it appears to indicate the withdrawal of cerebral inhibition. Certain subjects comprised in this group reveal patches of insular sclerosis throughout the lateral columns of the cord, accounting for the secondary degeneration and resulting knee phenomenon. Gowers has called attention to this fact in the ordinary forms of spastic paraplegia.²³

Speech Troubles.—These are almost invariably a notable feature, and at an early stage of the affection the patient's tongue and lips are badly co-ordinated. The movements of the tongue are performed with hesitancy and difficulty; it is protruded with some considerable effort, or jerked in and out rapidly, and a fine fibrillar tremor may be detected over its surface upon protrusion. The lips also exhibit notable tremulous agitation during speech, the enunciation of labials producing excessive tremor spreading to the lower facial group of muscles generally. Articulation is blurred, thick, and slovenly, like that of a drunkard, varied by a tremulousness and often an explosive utterance verging on unintelligibility. The disturbance is much intensified by coincident emotional states, and may be much diminished when by tact upon the

²⁰ The "Argyll-Robertson" pupil.
²¹ "phthalmo-oplegia interna" of Mr. Hutchinson.

²² It is assumed here that there is no actual failure in sensory conduction, as evidenced by the normal cutaneous sensibility of the region stimulated.

²³ *Diseases of the Nervous System*, vol. i, p. 337.

part of the interlocutor the subject's mental calm remains undisturbed.

Muscular Tremors and Inco-ordination.—In a large proportion of cases comprised in this category tremor is a very notable feature, and is usually limited to the tongue, lips, and muscles of the lower facial group; occasionally, but much less frequently, do we find a fine tremor of the hands when the arms are extended horizontally. The tremor of the oral musculature, especially elicited during voluntary movements as of speech or ingestion of food, or during emotional states, would appear to be due to implication of the cortical mechanism of speech, like the corresponding alcoholic tremor of the hands after a night's debauch. We must distinctly sever it from the further symptom of ataxia, which also in certain of these subjects involves the muscles of the same regions, as indicated by the contortions and grimace presented by them in efforts of protruding the tongue, or of whistling, etc. As I shall indicate later, the ataxia thus elicited upon voluntary stimulation of the speech centre is almost certainly due to the implication of the muscular sense fibres of the muscles of articulation, which probably find their termini in this cortical region. Occasionally the ataxia alone presents itself; at times it is associated with much tremor.

It may be noted here that reference to the tables accompanying this article indicates tremor and twitching of the brow muscles in six cases. I have emphasised this fact in my work on *Mental Diseases*, and have long been accustomed to regard the uneasy furrowing and twitching movements of the muscles of the brow as specially characteristic of chronic alcoholism, and as indicating when seen the alcoholic factor in general paralysis also.²⁴

Tabetic Cases.—Upon examining these exceptional cases we observe as a notable fact that in each case where the ataxic jerking is recorded we have a tabetic sign as an accompaniment. Thus in one there is the Romberg sign of unsteadiness and swaying upon closing the eyes with feet approximated, and in four others the knee-jerk was absent in all. If we now summarise the features characterising this group we find that they embrace:

Paralytic mydriasis with the Argyll-Robertson sign—exalted deep reflexes, or, in a few exceptional cases, abolished reflexes (much tremor of lower oral group of muscles and tongue in the former; ataxic jerkings in the latter exceptional forms) profound speech troubles, and we may add as mental features much optimism and profound dementia. We shall find it convenient to sever into two distinct groups the spastic and tabetic paraplegic subjects now described.

Associative Iridoplegia.—The above by no means complete our category, for we find a certain small proportion of subjects in which the mydriasis, still paralytic in nature, indicates a different distribution of lesion in the oculo-motor region; the pupil is moderately dilated, fails to react to light either direct or consensual, and also fails to contract upon convergence.

In 6 cases the mydriasis measured as follows in millimetres:

$$\frac{4.5}{5.0} : \frac{6}{3.5} : \frac{3}{3.5} : \frac{-}{4.5} : \frac{-}{5} : \frac{3}{3.5}$$

It is distinctly to be understood that so far as the oculo-motor signs are concerned the Argyll-Robertson sign by no means necessarily terminates in abolished contraction upon convergence. My own experience would indicate that the former sign remains persistent throughout the disease up to its fatal termination; and that where failure of the associated movement (contraction on convergence or accommodation) exists it is met with from the first, and would therefore indicate a topographical significance. In all these cases alike a failure of the usual dilatation upon shading is observed, and this extremely sluggish or fixed state of pupil is only to be explained, according to Mr. Hutchinson, upon two hypotheses:

(a) That neither sympathetic nor retinal incident can produce active dilatation or contraction in the absence of its antagonist.

(b) That the lesion has resulted in paralysing both the fibres of the sympathetic and motor oculi supply.

We have, however, seen reason for doubt as to the implication

of the vasomotor in such cases; as above stated, we should not expect dilatation upon shading, even were the sympathetic intact. Mr. Hutchinson appears to adopt the theory of dual implication (b), and refers the site of lesion to the ciliary ganglion as a crossway for all these fibres; here, he suggests, occurs the degenerative process resulting in this associative iridoplegia. In perfect conformity with this opinion is the fact that such cases (and I can affirm this for general paralysis as for ordinary tabes dorsalis), which fail to respond both to light and convergence, usually run on to an abolition of accommodation also, the ciliary muscle being paralysed. Thus, in two such cases, such cycloplegia presents itself in association with failure of response on convergence and to light stimulus, the pupils being of large size, namely:

$$\frac{6.5}{5.0} : \frac{4.5}{5.0} \text{ mm.}$$

This condition may be described as cycloplegic iridoplegia, or may be termed, after Mr. Hutchinson, ophthalmoplegia interna.

With reference to this point, Mr. Hutchinson remarks: "My suggestion is that when the triad of symptoms is present—inability to use the ciliary muscle, paralysis of the dilating fibres of the iris, and paralysis of the contracting fibres of the iris—without any other form of orbital paralysis, the seat of the disease can be in no other structure than the ganglion itself."²⁵ And further on, in a note, he adds: "My only hesitation on this point is as to whether in some cases the same symptoms may be due to disease near the nucleus of the third nerve. It is not easy, however, in such cases to see why the vasomotor should be affected. I have, however, seen the pupil motionless and accommodation lost in some cases in which the disease was believed to be in this position; always, however, there were other complications."²⁶

It is, of course, possible that such symptoms may result either from this peripheral or from a central lesion; and in the cases quoted by Mr. Hutchinson, it is just as probable that the lenticular ganglion was the site of implication, as it is that in the forms of ophthalmoplegia interna prevailing in general paralysis the lesion is a nuclear one in the extensive segmentations of the third nerve. Nor does it appear to me improbable that so extensively segmented a tract as that presented by the motor oculi nucleus should have very close, though as yet undetermined, connection with the vasomotor tract. Given this latter, and we have a full explanation of cycloplegic iridoplegia in general paralysis (which, moreover, is usually double) from degeneration of the third nerve nuclei in the upper quadrigeminal region. We shall see reasons for accepting both positions in our consideration of these cases which comprise our second group.

(To be continued.)

THE HUNTERIAN LECTURES

ON

INFANTILE SYPHILIS.

Delivered at the Royal College of Surgeons of England.

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[ABSTRACT.]

AFTER some introductory remarks, in which he referred to the conflicting opinions which had been held as to the nature, and even as to the occurrence of inherited syphilis, Hunter having denied it, Dr. Coutts proceeded to discuss the influence of either parent, both as to the transmission of the disease and as to its severity.

THE INFLUENCE OF THE FATHER.

After quoting published statistics as to the apparent immunity in most cases of the mothers of syphilitic infants, he said: "I have collected notes from my out-patient

²⁵ On Paralysis of the Internal Muscles of the Eye (Ophthalmoplegia Interna), *Medico-Chir. Trans.*, vol. lxi., p. 216.

²⁶ *Loc. cit.*

²⁴ *Op. cit.*, p. 326.