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THE PUPIL IN EXTRA-OCULAR DISEASE.[1]

BY

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New York City.

So intimate is the relationship which the pupil bears to the nervous system, so much more complete than is the case with any other structure, and so characteristically are many nervous diseases mirrored by its deviations from the normal, that it may well be considered the barometer of the nervous system.

In order to thoroughly understand the significance of pathological alterations in the pupils in their relation to extra-ocular conditions, we must know the mechanism governing them in the normal state.

Two antagonistic sets of nerves control the normal tonus of the iris; the sympathetic, acting under appropriate stimulation as a dilator and the oculomotor nerve as a sphincter, either by direct action in accomodation, or indirectly through stimulation of the centripetal fibres of the reflex arc.

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[1]Read before the Harvard Medical Society of New York, Feb. 22, 1908.

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The nucleus of the oculomotor nerve is in the floor of the aqueduct of Sylvius quite near the median line. In this situation it is in close relation to the nucleus of the sixth of the opposite side, from which it derives its fibres to the internal rectus. It enters the orbit through the sphenoidal fissure, after dividing into a superior and an inferior branch. The first of these supplies the rectus superior and levator palpebrae superioris, while the inferior branch divides into three;—one for the internal rectus, one for the inferior rectus, and the third for the inferior oblique and ciliary ganglion, this latter being for us the most important since it supplies the motor fibres to the iris and to the ciliary muscle. The sympathetic fibres have their immediate origin in the ciliary ganglion, the remote nucleus being in the cervical cord.

The ciliary ganglion which is situated in the rear of the orbit, receives, as already indicated, sensory filaments from the nasal, motor from the third and sympathetic fibres. These functions are re-distributed through the ciliary nerves, the sensory (and trophic), going to the eyeball as a whole and the motor and sympathetic to the ciliary muscle and iris.

The reflex center of the iris is situated somewhat farther back than is that of the third nerve proper, its fibres joining the latter only after it has left its own nucleus.

By bearing in mind these details, we can locate with fair precision many lesions in various parts of the tract by observing concomitant muscular involvement.

When a lesion exists in the nucleus, owing to its close relationship with other nuclei, we are apt to get paralysis of functions other than those controlled by the third.

A normal pupil with paralysis of all the extra-ocular muscles supplied by that nerve, except the internal rectus, would indicate, other things being equal, a nuclear lesion purely, while the same condition including paralysis of the internal rectus and pupillary dilatation, point to one of extra-nuclear origin, since, as was noted above, the fibres of the internal rectus are derived from the sixth nucleus of the opposite side.

There are several reasons why this theoretical ease of localization in the centrifugal path is rarely possible. In the first place, lesions are not often so limited as to involve the territories at issue without producing widespread destruction of other centres. In the second place, should the process, whatever its nature, be irritative rather than destructive, we might be misled by an increased myosis where a mydriasis would have been looked for; thirdly, certain toxines may have selective qualities with respect to certain fibres, leading us to

suspect a lesion at a point where none exists. These sources of error should all be carefully excluded.

It can, moreover, be also readily understood that so far as the significance of pupillary changes is concerned, the centripetal path of the reflex arc has as important a bearing as has the centrifugal tract.

This path is to a large extent in common with the optic tract. It follows, and its fibres are mingled with those of the visual nerves, its constituent elements participating in the decussation, but they can be definitely traced no further than the external genticulate bodies; beyond that they are lost.

It has been supposed that their function was served by Mynert's fibres which extend back from that region, and a hypothesis on this basis seems to answer most questions as to the physiological function and identity of these fibres, though I believe actual proof is still wanting.

But as was the case with the centrifugal tract, so here, the localization of a lesion depends largely upon associated phenomena, the essential difference being that whereas in the former these phenomena concerned chiefly the extra-ocular muscles, in the latter they have to do principally with sensory disturbances, i. e. visual defects.

A few instances to be specific will be more illuminating.

A lesion of one optic tract peripheral to the chiasm, or destruction of the retina, by destroying the reflex fibres, will give us in addition to blindness in the affected eye total unilateral loss of the direct light reflex, as well as of the consensual reflex in the fellow pupil, but leaves us with preservation of the indirect reflex in the pupil of the affected side, because of the still existing integrity of the motor tract.

Such lesions peripheral to the chiasm are more frequently the result of fracture of the base of the skull than of tumor growth.

Going back a step farther, a patient presenting a bi-temporal hemianopsia with loss of both direct and indirect pupillary reflexes when light is thrown upon the nasal hemispheres of the retinas, but with the preservation of these reactions when light is thrown upon the temporal halves, must needs present a sagittal lesion of the chiasm with total or at least almost complete interruption of the decussation.

That such is the only possible condition is clear when we remember that the fibres from the nasal halves of the retinas decussate (both light-reflex and visual), while those of the temporal halves take the direct paths.

This lesion is quite frequently found in the presence of acromegaly, resulting from an over-growth of the hypophysis.

The next point at which we should expect a characteristic symptom-complex as a result of destruction of the tract would be between the chiasm and the point of deflection of the light reflex and visual fibres, say, on the left side. This would cause left homonymous hemianopsia with total loss of both direct and indirect pupillary light reflex with illumination of the right side of either retina but with the preservation of both varieties of reflex with illumination of the left side of either retina.

Lesions far enough back to cause the hemianopsias are more frequently the result of new-growths, and less often follow fractures of the base.

A hemianopsia with normal pupillary reactions would indicate a lesion behind the point of deflection above alluded to,—in all probability, cortical.

Finally, the destruction of any portion of the reflex tract between the external genticulate bodies and the reflex center would give us reflex pupillary rigidity, but without limitation of the visual field, in other words, the tabetic pupil, though in this particular disease, as indicated later, the exact point at which the interruption occurs is still a matter of active dispute.

So definite, then, are the anatomical relations between the pupillary reflex and the visual fibres within the limits just given, that lesions affecting these structures can be located with great precision.

In contradistinction to such definite symptoms, those which are reflected in the pupil, betray little more than evidence of intracranial pressure.

Neoplasms, embolism, hemorrhage, abscess, and more or less diffuse types of meningitis are scarcely to be diagnosed without rather a comprehensive study of the other symptoms pointing to diffuse or focal intra-cranial pressure, such as nausea, cephalalgia, choked disc, convulsions or convulsive movements, local paralysis, and in abscess and meningitis, fever, etc.

In traumatic lesions there is ordinarily an inequality of the pupils, the larger one usually being on the side of the lesion, but this rule is by no means constant. Here, local paralyses generally of the extremities are our most certain guide in their localization.

In the meningeal infections, especially of the vertex, beside fever, relatively low pulse, and a tendency to paralysis of the extremities, the pupils are usually contracted at first, later dilated and frequently unequal. The same is true when the base is affected except that other cranial nerves

are more universally involved than in the vertex infections.

In diseases of the spinal cord other than specific, instances of which are to be considered later, there are, generally speaking, no characteristic changes either in the pupils themselves or in their reactions.

In such affections as spinal transverse myelitis, caisson disease, and spinal meningitis, we do not expect pupillary symptoms either as diagnostic or confirmatory factors, and even in those instances in which there is widespread destruction, we usually find pupillary changes chiefly as a result of some lesion of the sympathetic which arises to a large extent in the floor of the fourth ventricle and leaves the cerebrospinal axis through the anterior roots of the upper dorsal nerves. Involvement of the sympathetic, therefore, indicates a high lesion, usually not lower than the second dorsal segment.

We must not forget that while stimulation or irritation of the sympathetic dilates the pupil, its destruction causes a myosis resulting from the now no longer opposed action of the oculomotor nerve.

High injuries to the cord from the action of fractures, accompanied either by hemorrhage or laceration, are fairly prolific of sympathetic phenomena. For the same reason, spinal syringomyelia, when not

lower than the second dorsal segment, and neoplasms of the cord, may produce through their influence upon the sympathetic, distinctive pupillary changes, usually unilateral, less frequently bilateral, though even in the latter event, generally with pupillary inequality, and in most instances, (unless the effect is irritative rather than destructive), with myosis.

The part played by the sympathetic fibres in their course between the spinal exit and the ciliary ganglion is essentially extra-neurological, since in this situation they are not ordinarily subject to diseases peculiar to the nervous system.

Passing through the upper thoracic ganglion, the upper cervical ganglion, then along the internal carotid artery, over the gasserian ganglion to the ophthalmic division of the fifth nerve, then along its nasal branch to the ciliary ganglion and nerves, the pupil reflects those lesions to which these nerves are subjected by the structures with which they are in relation.

In the thorax they are frequently involved in a tuberculous pulmonary process and in aneurism of the aorta, while in the neck they will often show pressure effects of goitre or other tumor formation, such as that resulting from Hodgkin's disease, or glandular infiltration from malignant disease of the throat.

In diseases characterized by over or under development, such as hypertrophy, or its reverse, in cretinism and myxedema, in fact in all nutritional diseases affecting the nervous system, there are not as a rule pupillary changes.

Diseases characterized by the intoxications having a selective quality with respect to the central nervous system, reflect nothing peculiar to themselves in the pupils.

In alcoholism the pupils may be somewhat sluggish, in epilepsy dilated, as in other forms of acute auto-intoxication, but in diseases such as paralysis agitans, and in poisoning from metals, they are, generally speaking, unaltered.

The same is true of most of the other cerebro-spinal diseases, in which supervening defections, when they occur, are of irregular importance, and when they do indicate a focal lesion, it is ordinarily but incidental to the general process.

Of equal interest and of as great importance are certain characteristic pupillary changes in the presence of specific diseases, chief of which is locomotor ataxia.

So well known are its appearances in this disease that its classical qualities of reflex rigidity and accommodative contraction with or without myosis need but to be mentioned. Yet the average physician is apt to depend too much upon the presence

or absence of this condition in making a diagnosis, and while it justly constitutes one of our strongest confirmatory symptoms, it is by no means constant. Though statistics vary greatly as to its frequency, depending largely upon the stages of the disease at which the observations were made, as well as upon the personal equation of the compilers, still 60-70% may be accepted as an average proportion of the cases of tabes in which it is found.

Many subjects, while not exhibiting a total absence of the light reflex, will show but slight contraction, and even when a fair amplitude of constriction exists, a sluggish response should arouse an immediate suspicion of this para-syphilitic disease.

It appears not to be generally appreciated that the reflex rigidity exists not alone with respect to light but that it is true of all stimuli that can be applied whether through the sympathetic or otherwise.

The tabetic pupil may also be unilateral (infrequently) it may differ in diameter from its fellow, and it may be irregular but while these peculiarities may all be looked for, and expected, they are not common.

Again, while in this condition myosis is the rule, the pupil is not infrequently normal in diameter, and at times abnor-

mally dilated; and this dilatation has a certain prognostic importance, in that it is more often associated with or followed by optic nerve atrophy than is the myotic pupil. Still, it is sometimes present as a late development of the disease without the atrophy, but with paralysis of accommodation, and finally, it may exist in the absence of all these complications.

The exact pathological basis of the Argyll-Robertson and myotic pupils has not yet been established with the precision which one might expect, for investigation of the regions theoretically indicated shows no constant lesion capable of producing the characteristic phenomena. While there are many theories, some of which are partly confirmed by experimental evidence, that which assumes the involvement of a special reflex center in the posterior columns of the upper cervical cord and distal end of the fourth ventricle appears to have the firmest support. Clinically speaking, however, this is not of great importance, since the matter of cerebral localization is not a factor in the diagnosis, though it would be of intense scientific interest.

That the lesion, whatever its nature, is more or less diffuse, is shown by the fact that reflex pupillary rigidity and myosis may be present each without the co-existence of the other.

The two other conditions in which we find the Argyll-Robertson pupil are paresis and syphilis of the central nervous system, but in neither of these diseases, particularly the latter, is the symptom so common as it is in tabes. The matter of differential diagnosis is, however, rarely obscure, even in the early stages, since each of the maladies is almost sure to present other evidences, constitutional or local that are more distinctive than the Argyll-Robertson pupil, which after all is common to the group as a whole.

While in locomotor ataxia it was noted that the most distinctive feature was the myotic pupil, other defections being of secondary and minor importance, we find that in paresis one of the first and earliest and most constant phenomena is inequality of the pupils with a sluggish reaction to light, both direct and consensual. The tendency of the sluggishness is to be more marked in one eye than in the other. The less constant, hence minor deviations from the normal in paresis are myosis (less constant than in tabes) mydriasis (more frequent than in tabes),pupillary irregularity, and of course, the typical Argyll-Robertson pupil, supervening in about 30% of the cases, usually comparatively late in the disease, hence of less importance, from the diagnostic aspect than in locomotor ataxia.

In so many of these cases does paralysis of accommodation occur as a late development that one is tempted almost to regard in this disease the Argyll-Robertson pupil as the intermediate stage of a process resulting in complete pupillary paralysis.

In syphilis of the central nervous system reflex rigidity is still less common than it is in paresis; too infrequent to be regarded as a cardinal symptom. As an evidence of syphilis, it is much more often found in conjunction with paralysis of the external ocular muscles either on one side or both than it is in the two other para-syphilitic diseases. Instances of its disappearance under antisyphilitic treatment are recorded, an event which rarely, if indeed, never happens in the true para-syphilitic diseases.

Sporadically this pupil is found in association with other organic disturbances, but under all circumstances it indicates definite organic disease of the central nervous system.

The reverse of the Argyll-Robertson pupil is one of the rarest changes with which we meet, and occurs chiefly in those conditions in which, owing to unilateral blindness, accommodation has been lost. It may be congenital, and it may in isolated instances exist as an evidence of central disease, but so rarely as to have but little diagnostic importance.

Occasionally we find it as an evidence of post-diphtheritic paralysis, lasting at times for several weeks, but always eventually disappearing.

Total loss of pupillary innervation is less common than is the Argyll-Robertson pupil. While rare in tabes, it is often found in general paralysis and in syphilis, and when identified with the latter, it is very prone to co-exist with partial or complete ophthalmoplegia externa.

In functional diseases including those insanities not based upon a demonstrable pathology, the pupil is of but little clinical significance except in a negative sense.

In hysterical conditions when pupillary abnormalities exist, there is usually mydriasis, sometimes unilateral, generally bilateral, and as a rule with preservation of the light reflex. A permanent loss of this reflex in hysteria of course never occurs.

While in hysterical convulsions the pupils usually react to light, giving us a diagnostic point in the differentiation from epilepsy, it occasionally happens that here too they are dilated and immobile, though such reflex rigidity is admittedly rare.

While permanent and obtrusive differences in the sizes of the pupils is a very positive sign of central disease, (intraocular conditions being eliminated), slight and fugitive differences are ordinarily of but little import, and indeed, it might be

said that absolute equality under uniform illumination exists only in a minority of cases.

A reflex frequently seen and often puzzling unless understood is hippus, a pupillary oscillation of variable amplitude elicited when the patient is made to observe a distant object. The pupils are seen alternately to contract and to expand though there be no effort at accommodation. This phenomenon is of no material importance beyond suggesting more or less nervous instability, to the degree that one might expect in the average case of neurasthenia.

Still another reflex of psychological rather than of clinical interest is the so-called attention-reflex, a reaction excited by placing in a darkened room with the patient, on a level with the eye, but not in the direct line of vision, a lighted candle, then requesting him to fix his attention upon it. Under these conditions the pupil contracts, the contraction being in direct proportion to the degree of illumination. If, however, the attention is relaxed, though the relation between the source of light and the line of vision remains unchanged, the pupil dilates.

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