Nursing of Diseases of the Eye.

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GLAUCOMA.

Glaucoma, in one form or another, is one of the commonest diseases seen in the ophthalmic ward. It may be defined as the condition resulting from increased intra-ocular tension. The cardinal feature, therefore, of all forms of glaucoma is hardness of the globe. In fact, in all cases, except where increased density of the coats is present, increased resistance to external pressure is synonymous with glaucoma.

The method of estimating the intra-ocular pressure, or, as it is often called, the tension, is as follows:—The patient should be seated, and should look downwards. The examiner, standing in front, places one forefinger on the upper lid above the globe, wedging it slightly between the globe and the orbital margin, and with the other makes slight pressure on an adjacent region so as to dimple the sclerotic. The amount of pressure required varies with the intra-ocular tension and must be referred to the other eye, if that be healthy, or to a mental standard if the fellow eye be unavailable. It follows from this that different observers will have different names for the same degree of tension, as their mental standards will differ. Intra-ocular tension is usually denoted by the letter T. If in the observer's judgment the force required to dimple the sclerotic is not greater than usual, he will note Tn. If a distinctly greater resistance is present, he will note T + 1 or T + 2; T + 3 is reserved for cases where the sclerotic cannot be dimpled. (A similar scale, T − 1, T − 2, T − 3, indicates decrease of intra-ocular tension below the normal standard.)

It will be remembered that in the lectures dealing with the physiology of the eye, the course of the intra-ocular fluid and their bearing on the tension of the globe was briefly alluded to. The inflow of fluids at the ciliary processes and outflow through the spaces of Fontana were then mentioned. The diagrammatic section then given showed the position of the channels of excretion between the base of the iris and the posterior surface of the cornea (*vide* Vol. 28, p. 65).

Unfortunately, in the section drawn none of the actual openings were presented. It must be borne in mind that in reality the spaces of Fontana, shown as oval tubes in the figure, open widely into the anterior chamber. These openings would be closed by the base of the iris as by a valve if this membrane were to fall forward against Descemet's membrane, and all excretion would be prevented. Any variation of the intra-ocular pressure must clearly depend either on diminished outflow of the ocular fluids, or increased inflow. It is generally held that, of the two, the former is by far the more important factor in the production of disease.

Much work has been done by various observers, and much remains yet to do, as to the method of production of the first stage of primary glaucoma, but it has been shown beyond all possible doubt that in the later stages the spaces of Fontana, at the angle between the iris and cornea, through which the aqueous humour escapes into the canal of Schlemm, are stopped by the iris base, which there lies in contact with, and eventually becomes adherent to, the posterior surface of the cornea.

The experiments of Priestley Smith have shown further that if the pressure in the vitreous rises beyond that of the aqueous humour, the iris-base is driven forward and the excretion from the anterior chamber much diminished, the intra-ocular tension rising with this diminution.

The cause of the first disproportion between the pressure in the two chambers is, however, by no means always clear, and it is at least possible that this may sometimes be hyper-secretion. There can be no doubt, however, that when once the base of the iris has taken up this faulty position, and lies in apposition with the cornea, obliterating the normal iritic angle, the consequent diminution of excretion is the chief cause in maintaining the high tension.

Glaucoma as a clinical whole is sub-divided under two heads.

In one class of cases the exciting cause is easy to discover, and the disease is spoken of as secondary to such cause. The other and more important group is called primary glaucoma, because no obvious lesion precedes it.

In secondary glaucoma we have obviously to deal with diminished excretion. If, from severe iritis, the margin of pupil in its whole circumference is fixed to the lens so as to prevent the passage of the aqueous humour from the posterior into the anterior chamber, the retention of fluid in the eye causes an increase of intra-ocular tension.

Again, in some instances the lens is dislocated through the pupil and lodges in the anterior chamber; here it will act as a ball valve blocking the excretory passages and producing glaucoma.

Lastly, the nature of the aqueous fluid may be altered; it may be more albuminous, and escape with greater difficulty. This may cause increase of intra-ocular tension. But by whatever condition brought about, whether primary or secondary, the symptoms of glaucoma are alike, though varying in degree with the acuteness of the attack.

The appearance of the eye in an acute case of primary glaucoma is one of intense ciliary engorgement. The circumcorneal zone is deeply congested. The cornea is lustreless, steamy, and finely rough. The pupil is dilated and fixed, the anterior chamber

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