support Loewenstein and Foster's idea that "the basic change is senile but the process may be aggravated by proteolytic enzymes (lysins) in the aqueous, the product of glaucomatous metabolism." Long-standing glaucoma may lead to iris atrophy and shrinkage of anterior stromal fibres with consequent development of ectropion uveae in many cases. In the case under discussion, however, the pupil has been kept in a state of miosis for fifteen months, and theoretically the taut radial fibres of the iris might rupture as they gradually weakened, instead of producing ectropion uveae. Fixation of the pupil margin by posterior synechiae (as in the first case described by these authors) might conceivably have a similar effect.

The fact that the lower half of the iris is mostly involved in the reported cases of iridoschisis may, as Vogt suggested, be due to gravity, but another possibility is that convection currents in the anterior chamber play a part.

## A NOTE ON THE EFFECT OF SLEEP ON GLAUCOMA*

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A DISCUSSION arose in out-patients concerning the effect of sleep on the symptoms associated with a rise in tension in cases under observation for suspected glaucoma. Such varied opinions were put forward and so many different authorities were quoted in support of them, that it was considered worth while to submit the subject to criticism.

A patient who has once had an attack of raised tension, discomfort or even pain in the eye accompanied by blurring of vision during the daytime, or by the appearance of haloes round lights at night, can always recognise a second one as being similar. An attack will often start in the evening when the patient is tired and has settled down to read or sew. She will often volunteer the statement that if she goes to sleep the attack is relieved and she awakes refreshed in the morning.

This clinical observation has been known for many years. Fuchs recorded it in his famous Handbook[1] thus: "When the attacks come on in the evening they always cease with sleep; during the day also, an attack can be interrupted by going to sleep." The point is ignored in most of the modern text-books and the apparent conflict between it and the diurnal variations in the intra-ocular pressure are not discussed.

It is well established that in a normal eye the tension is lowest

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between 1 p.m. and 5 p.m., and that it rises slightly (3 mm., Schiötz) during the night to its highest about 5 a.m.[2]. In the early prodromal stage of glaucoma the difference between the lowest and highest readings increases, and the increase occurs much more rapidly. Furthermore, peaks of tension are not confined to the early morning, but may appear at any time during the 24 hrs.[3]. Ultimately the low pressure periods become shorter and shorter until the tension remains up the whole time.

The rise in tension during the night is not fully understood, but is perhaps mainly due to immobility of the lids and eyeball, with consequent lack of massage of the globe by the muscles.

One source of confusion is the well-known fact that in a pre-glaucomatous eye the tension tends to rise in the dark, due of course to the dilatation of the pupil with blocking of the angle. The Seidel dark-room test is based on this observation. During sleep, however, the pupil contracts, and dilatation from darkness does not occur. It has been suggested that miosis explains the beneficial effect of sleep, and could be looked on as the reverse of the "dark room" effect. In a normal eye dilatation of the pupil in the dark does not raise the intra-ocular pressure[4], because the drainage is so good that filling of the angle makes no difference. Conversely the miosis of sleep does not prevent the normal slight physiological rise. In a pre-glaucomatous eye, however, conditions of drainage are so precariously balanced that the dilatation in the dark causes a rise, whilst the miosis of sleep may, by opening the angle, cause a fall in the tension.

There are other factors to be considered. The anxiety and worry to which this type of patient is so prone is relieved during sleep. In this connection it is interesting to note that a nightmare can precipitate an attack of raised tension. Again, very few people submit to discomfort without taking aspirin or some other analgesic, and this may directly influence the intra-ocular pressure.

The early hours after waking, when a number of adverse factors come into play, are a common time for attacks of cloudy vision and pain. The physiological rise has just passed its peak, and the pupil may be dilated because the patient has been lying in the dark with his eyes open. Prolonged decubitus with no muscular massage to the globe, and the return of psychogenic stimuli may also help to provoke a rise in tension.

I wish to thank Mr. Eugene Wolff for his helpful observations. It was in his clinic at Moorfields Westminster and Central Eye Hospital that the subject was discussed.

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