PRESBYOPIA*

BY

R. A. WEALE

Department of Physiological Optics, Institute of Ophthalmology, University of London

(1) THEORY OF ACCOMMODATION

IT is only reasonable to expect that theories of accommodation should attempt to account for presbyopia (Fincham, 1937). It will be recalled that the modification by Fincham (1925) of the theory of von Helmholtz (1855) explains a large number of the incidental facts (Duke-Elder, 1938), and may be briefly stated as follows:

When the ciliary muscle constricts, the tension on the zonule is released. The suspensory ligaments being attached to the equatorial region and to the anterior face of the capsule (Schulze, 1867), the elastic properties of the capsule are enabled so to act on the (plastic) lens substance as to translate an axipetal movement of the equatorial region into a centrifugal displacement of the axial portion of the lens. In virtue of the fact that the thickness of the capsule is non-uniform, being maximal in an annulus surrounding the anterior pole of the lens at a distance of 2 mm. (Fincham, 1925) or 3 mm. (Salzmann, 1912), the anterior pole bulges forward during accommodation more than the posterior pole is displaced backward: the elastic properties of the capsule are said to mould the plastic lens substance. As the lens substance "sets" in its unaccommodated, flat form, the capsule can mould it to a progressively smaller extent and the amplitude of accommodation decreases till it reaches a zero value during the sixth or seventh decade.

Fincham believes that the lens substance is plastic, *i.e.* that its shape can be modified by an external force and—by definition of the term "plasticity"—that this shape will be maintained even if the external moulding force is withdrawn. This implies that the lens substance offers to deformation no opposing force other than viscous or frictional effects. Two of his observations are, however, not entirely consistent with this view. He noticed in the first place that "the lens-substance could be easily distorted by light pressure, *but always returned to the same form* which was assumed to be its natural form when not under compression by the capsule". This suggests that there must be restoring forces at work in the lens matrix which are capable of maintaining the "natural form". Secondly, Fincham noticed that the lens substance removed from the capsules of young monkeys "loses its typical

* Received for publication December 29, 1961.

conoidal form of anterior surface, becomes thinner, and increases its equatorial diameter. . . . That is to say, it changes from the accommodated form, which it had assumed on being released from its suspensions, to a shape which closely resembles the unaccommodated state". If the lens substance were truly plastic, *e.g.* like putty, it would, of course, remain in the form to which the capsule moulded it before it was peeled off. These observations suggest that the lens substance houses restoring forces which tend to maintain it in the unaccommodated form as would, indeed, be expected from a tissue characterized by as regular a structure as the fibres bestow on the lens matrix.

The above reasoning supports Fincham in his criticism of the view as originally stated by von Helmholtz, namely that the natural form of the lens substance is more nearly spherical (as it would be if it were more nearly fluid like the vitreous). But it changes the emphasis given to the interaction of capsule with substance. If, as would appear from Fincham's work, the lens substance contains restoring forces, then part of the forces antagonizing capsular elasticity must reside within the lens (but see below). Secondly, non-cataractous old lenses cannot be said to be really hard. No one has as yet measured lenticular hardness, *e.g.* by determining applanation or indentation pressures: moreover an infantile lens appears to be—subjectively—softer than a much older lens. But progressive senile reduction in capsular elasticity could produce the same falling off in accommodation as would sclerosis of the lens substance. Where von Helmholtz was perhaps less incorrect than later views have allowed one to believe was in his view that the lens substance had some elastic properties: contrary to von Helmholtz's view, these do not allow the lens to assume its accommodated form as shown by Fincham, but, as von Helmholtz implied, they exist. It would appear to follow that, in the young human eye, the elastic forces of the capsule are relatively greater than those of the lens substance, which antagonize them, but in the presbyopic the reverse is true. This view embodies all the facts explained by Fincham's modification of von Helmholtz's theory, and also explains why the old lens tends to remain in its unaccommodated state.

It would, however, be erroneous to assume that a change in the relative capsular and matrix elasticities of the human crystalline lens is the only factor bearing on the amplitude of accommodation and its variation with age. It is profitable to speculate on the effects produced by a variety of age changes on this function, and, conversely, to inquire into what results may perhaps stem from presbyopia in relation to ocular—as distinct from visual—function as a whole.

(2) THE SHAPE OF THE CRYSTALLINE LENS

The first point to consider is the growth of the human crystalline lens with age. Priestley Smith (1883) found that lenticular mass and volume increased

approximately linearly with age after puberty, the same being true of the equatorial diameter. Raeder (1922) and Johansen (1947) extended the measurements to the sagittal diameter. Priestley Smith's data were subjected to severe criticism by Krause (1934) and by Brown and Evans (1937), largely because the growth of other mammalian lenses obeys a different law. Nonetheless, the survey of Scammon and Hesdorffer (1937) and the measurements of Johansen (1947) and of Salmony (1961) fully support Priestley Smith's conclusion regarding the continued growth of the human lens almost throughout life (Fig. 1). As the infantile lens is more nearly spherical than is the adult lens (Wolff, 1961), the equatorial diameter must grow postnatum at a greater rate than does the sagittal: after puberty there occurs a diminution in the difference between the two rates of growth.

FIG. 1.—Growth of the human lens.
Ordinate: Volume in cu. mm (Scammon and Hesdorffer, 1937) and Mass in mg. (Salmony, 1961; Scammon and Hesdorffer, 1937; Johansen, 1947).

The available evidence points to the view that the normal eye-ball is fully grown at the age of about 16 years (Scammon and Wilmer, 1950). There is no question of its paralleling the growth of the lens. It follows, therefore, that the lenticular equator approaches the ocular wall throughout life, so

much so that in an old eye the circumlental space, visible, *e.g.* in aniridia, disappears altogether. In its advance towards the coat of the eye, however, the equator of the crystalline lens is not alone. It pushes before it the proximal attachment of the zonule. One can reason (Fig. 2) that, even if the properties of the lens and zonule preserved their pristine state, the tension on the zonule would be gradually decreased simply because the lens grows.

FIG. 2.—Schematic comparison of the anterior parts of the eye in the second and seventh decades respectively. The shaded parts represent the ciliary muscle (after Stieve, 1949, *cf.* Fig. 4).

As we have seen, the shape of the accommodated lens is governed by the properties of the capsule. Hence an eye, equipped with the above hypothetical, physically invariant, but nonetheless growing, lens, would tend to become myopic but for the fact that the radii of curvature of the lens surfaces increase. This factor (Smith, 1883; Alexander, 1921) would make the power of the lens less positive. It would, however, only tend to preserve the youthful *power* of the lens. It could not prevent the loss of accommodation entailed by lenticular growth. The loss comes about in the following manner.

Elastic elements in the ciliary body (Fincham, 1937), in addition to the structure of the choroid (Rohen, 1953) and the afore-mentioned restoring forces in the lens matrix, antagonize the elasticity of the capsule: when accommodation is relaxed the resultant push on the lenticular equator and pull on the zonule increase the tension on the suspensory ligaments and flatten the lens. It follows that, if the growth of the lens has led to an approximation of the lenticular and ciliary ligamental attachments respectively, some slack will have to be taken up as the ciliary

muscle relaxes its contraction. Consequently, less ciliary power becomes available for flattening the lens, *i.e.* the amplitude of accommodation is reduced. (It has been shown *ad nauseam* that the ciliary muscle is a functional entity and that its meridional, radial, and circular portions represent different perspectives of the same structure: Bowman (1849), Meyer (1865), Schulze (1867), Fuchs (1928), Fincham (1937). There is, therefore, little point in looking within the ciliary muscle for the antagonist to the ciliary muscle.)

Another point meriting attention is that, according to the measurements of Raeder (1922), the rates of senile increase in the thickness of the lens and of the corresponding decrease in the depth of the anterior chamber (Fig. 3) are as follows:

Depth of anterior chamber -0.013 mm. per annum
Lenticular thickness $+0.017$ mm. per annum

FIG. 3.—Variation with age of the depth of the anterior chamber and of the lenticular sagittal diameter.

Now there is no reason to assume that the lens grows because new fibres are deposited merely in its anterior part; if this were so the embryonic nucleus would not be embedded in the centre of the lens but in the posterior part. If, however, the growth is symmetrical about the equatorial plane, then the depth of the anterior chamber should decrease at half the rate at which the lens thickness increases. This is not found to be the case: the depth of the

anterior chamber decreases at a rate greater than this, a fact which suggests that the lens moves bodily forward. An optical result of this movement is that the power of the eye becomes more positive, *i.e.* it counteracts a senile tendency to hyperopia by about 1.5 D in 40 years. A possible anatomical cause of this movement will be considered in the next section. The progressive axial thickening of the lens as revealed in Raeder's work and that of others tends to make the lens more positive by an amount corresponding to a small fraction of a dioptré.

It may be objected that a consideration of the geometry of the situation is inadequate to establish the reduction in the amplitude of accommodation on the basis of lenticular growth. For accommodation depends not only on lenticular curvature but also on the difference between the lenticular refractive index and that of the neighbouring media respectively. It may be possible, for example, that the refractive index increases at a greater rate than the radii of curvature of the lens, in which case the power would increase with age. In this connexion it may be noted that Woinow (1874) observed small, but Freytag (1908) and Huggert (1948) negligible, increases in the refractive indices of the various lenticular layers. Reference to Fig. 2, however, shows that, although the radii of curvature increase, the lens *in situ* is somewhat less flat in the old than it is in the young eye as might be expected from the progressive loss of zonular tension (p. 663). If the ratio *R* of the sagittal to the equatorial diameter is taken to be a measure of its flatness, we find:

Age (yrs)	<i>R</i>
10-20	0.39
60-70	0.48

It is easily shown (*e.g.* Houstoun, 1930) that the smallness of the difference between the lenticular refractive index and that of the surrounding media allows one to neglect the increase in power due to sagittal thickening of the lens. Similarly, we have seen that senile changes in the refractive index would appear to be of little consequence. However, this cannot be said of the shape of the nucleus: this structure represents a lens in miniature within the lens and the fact that the "normal" eye remains by and large emmetropic provides striking evidence for the view that the nucleus, like the lens as a whole, becomes less flat as we age. In so far as the nucleus shrinks relatively to the lens as a whole (Gallati, 1923), its power may well be thought to increase with age although no measurements on this point appear to exist and may well be unobtainable.

We see, therefore, that the state of refraction of the lens is subject to influences pulling in opposite directions: the increase in the radii of curvature of the lens tends to produce hypermetropia, whereas the increase in its thickness and forward movement in the eye, together with the reduction in zonular tension and in the relative size of the nucleus, act together in the opposite sense, namely to make the eye myopic.

(3) THE CILIARY BODY

The above considerations show that growth of the lens in itself would lead to a reduction in the amplitude of accommodation even if no change occurred in the distribution of elasticity in the capsule-matrix complex. In fact, the assumption is not true. It has unfortunately not proved possible to trace detailed quantitative data on the hardness of the lens substance and on the mechanical properties of the lens capsule as a function of age (*cf.* Nordmann, 1954), but the data of Johansen (1947) show that when a lens of thickness t_o is progressively compressed and the capsule fractures at a thickness t_f , then the ratio t_f/t_o is approximately independent of age. There is, however, little doubt that the shape of the lens is modified with increasing difficulty as we age. It is noteworthy in this connexion that Stieve (1949) and Rohen (1953) have confirmed and extended the findings originally made by Fuchs (1928) on the senile alterations in the transverse shape of the ciliary complex. Fig. 4 shows estimates of the shape of the ciliary body as a function of age, which are not subject to the same degree of uncertainty (Abrams, 1961) as analogous sections (Kerschbaumer, 1888) of the ciliary processes.

FIG. 4.—Variation with age of the size and shape of the human ciliary muscle (after Stieve, 1949).

This senile alteration of the ciliary muscle reveals several striking features. In the first place the muscle attains its maximum size (in traverse section) in

the fourth decade. Secondly, it changes its shape quite appreciably: the juvenile obtuse angle of the equatorial region becomes acute in the seventh decade. Stieve describes numerous morphological alterations, the increase in connective tissue being among the more important findings. Atrophy of muscular fibres does not seem to occur before the seventh decade, although the proliferation of connective tissue may, of course, interfere with their mobility in relation to their environment.

The reason for the hypertrophy of the ciliary muscle is in dispute. Because Fuchs (1928) had come across 80-year-old eyes which had strong ciliary muscles, he disbelieved that the hypertrophy was a precursor of work atrophy. There is, however, no information whether or not the muscles he studied were obtained from eyes with "sclerosed" lenses or perhaps from eyes with long-standing aphakia. Old people with almost normal accommodation are met with sometimes, albeit infrequently, their reading glasses being of the order of as little as +1 D. The age at which the ciliary muscle grows to its maximum size coincides with the time at which lenticular "sclerosis" is fairly advanced. Two qualitative results can be deduced from the picture presented by Stieve (Fig. 4). In the first place, there occurs an effective reduction between the opposite points of attachment of the zonule. The reduction in the amplitude of accommodation with advancing years is thus a snow-ball effect, attributable to at least three causes, namely the growth of the lens, the growth of the ciliary muscle and the changes in elasticity mentioned in Section 1. Secondly, there is an obvious advance of the ciliary attachments of the zonule toward the anterior part of the eye. This provides an anatomical explanation of the relatively great rate of shrinkage of the depth of the anterior chamber of the eye.

(4) LENTICULAR GROWTH AND SENILE OCULAR PATHOLOGY

It would be foolish to suggest that, in a labile biological system, any effect can be traced to but a single cause. It is, however, interesting to inquire to what other effects the growth of the lens may contribute. One thinks, of course, immediately of lenticular metabolism. The growth of the lines of communication between the interior of the lens substance and the source of supply of most of its metabolites—namely the aqueous—must lead to internal atrophy unless the diffusion gradient is balanced by an alteration in the concentration of the relevant ions in the aqueous. There is no evidence that this occurs. Although growth cannot be the only cause of the metabolic difficulties of an ageing lens (the large cattle lenses do not exhibit youthful senile cataract), it does impede both the utilization of metabolites and the disposal of waste-products.

The partial forward movement of the lens, however slowly it may occur, may well facilitate the entry of some fluid into the vitreous chamber of the eye, a finding which is so common as to be thought virtually normal (Koepepe,

1918). The concomitant forward movement of the ciliary system can lead to only one result, namely a senile narrowing of the angle of the anterior chamber and all this may imply (Fig. 2).

These considerations are admittedly speculative. It may be, however, that a study of lenticular mitosis coupled with an examination of the positions which the lens and ciliary complex occupy at various ages may offer fields of research on cataract and glaucoma, supplementary to those pursued at present.

Summary

The implications of the von Helmholtz-Fincham theory of accommodation are examined with special reference to the causes of presbyopia. Senile alterations in the anterior part of the eye-ball are shown to contribute to changes in both the amplitude of accommodation and the power of the eye. The possible relations of these changes to other senile phenomena are briefly discussed.

REFERENCES

- ABRAMS, J. D. (1961). Private communication.
 ALEXANDER, G. F. (1921). *Amer. J. Ophthalm.*, Ser. 3, 4, 676.
 BOWMAN, W. (1849). "Lectures on the Parts concerned in the Operations on the Eye". Longman, Brown, Green, and Longmans, London.
 BROWN, E. V. L., and EVANS, E. I. (1937). *Trans. Amer. ophthalm. Soc.*, 1935, 33, 220.
 DUKE-ELDER, S. (1938). "Text-book of Ophthalmology", 2nd imp., vol. 1. Kimpton, London.
 FINCHAM, E. F. (1925). *Trans. opt. Soc. (Lond.)*, 26, 239.
 — (1937). "The Mechanism of Accommodation", B.J.O. Monogr. Suppl. No. 8. Pulman, London.
 FREYTAG, G. (1908). "Die Brechungsindices der Linse und der flüssigen Augenmedia des Menschen und höherer Tiere in verschiedenen Lebensaltern in vergleichenden Untersuchungen". Bergmann, Wiesbaden.
 FUCHS, E. (1928). *v. Graefes Arch. Ophthalm.*, 120, 733.
 GALLATI, J. (1923). *Z. Augenheilk.*, 51, 133-144.
 HELMHOLTZ, H. VON (1855). *v. Graefes Arch. Ophthalm.*, 1, pt 2, p. 74.
 HOUSTOUN, R. A. (1930). "A Treatise on Light", 6th ed. Longmans, Green, London.
 HUGGERT, A. (1948). *Acta ophthalm. (Kbh.)*, 27, Suppl. 30.
 JOHANSEN, E. V. (1947). "Undersøgelser over det indbyrdes Størrelsesforhold mellem Cornea og Lens crystallina hos Mennesket". Munksgaard, Copenhagen.
 KERSCHBAUMER, R. (1888). *v. Graefes Arch. Ophthalm.*, 34, pt 4, p. 16.
 KOEPPE, L. (1918). *Ibid.*, 96, 232.
 KRAUSE, A. C. (1934). "The Biochemistry of the Eye", Monograph No. 2, Wilmer Ophthalmological Institute, Baltimore. Johns Hopkins Press, Baltimore.
 MEYER, G. (1865). *Arch. path. Anat. Physiol. klin. Med.*, 34, 380.
 NORDMANN, J. (1954). "Biologie du cristallin". Masson, Paris.
 RAEDER, J. G. (1922). *v. Graefes Arch. Ophthalm.*, 110, 73.
 ROHEN, J. (1953). "Die funktionelle Gestalt des Auges und seiner Hilfsorgane". Steiner, Wiesbaden.
 SALMONY, D. (1961). Private communication.
 SALZMANN, M. (1912). "The Anatomy and Histology of the Human Eyeball in the Normal State," trans. E. V. L. Brown. University of Chicago Press, Chicago.
 SCAMMON, R. E., and HESDORFFER, D. B. (1937). *Arch. Ophthalm. (Chicago)*, 17, 104.
 — and WILMER, H. A. (1950). *Ibid.*, 43, 620.
 SCHULZE, F. E. (1867). *Arch. mikrosk. Anat.*, 3, 477.
 STEVE, R. (1949). *Anat. Anz.*, 97, 69.
 SMITH, PRIESTLEY (1883). *Trans. ophthalm. Soc. U.K.*, 3, 79.
 WINOW, M. (1874). *Klin. Mbl. Augenheilk.*, 12, 407.
 WOLFF, E. (1961). "Anatomy of the Eye and Orbit", rev. R. J. Last. Lewis, London.