FAILING SIGHT AFTER MIDDLE AGE.

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If the allotted span of life be taken as seventy years, then middle-age is by definition thirty-five, but the type of patient with whom this article deals is generally ten years, or so, older than this.

The commonest single cause for failing sight after the age of forty-five is diminution in accommodative power (*i.e.*, presbyopia), owing to loss of elasticity in the lens. The effect of this on reading and close work is so well known as to need no comment but it may also affect distant vision in cases of hypermetropia and astigmatism. A young hypermetrope of, say, 2 dioptres, will have no difficulty in neutralising this by accommodation and seeing 6/6. At the age of forty-five, however, when his total power of accommodation is in the region of 3.5 dioptres, the feat becomes much more difficult and he finds that he needs glasses for distance as well as for reading. At first, these may be required only when the patient is tired, but, as time goes on, he comes to need them more and more. A fair proportion resent this dependence on glasses for distant vision and date their blurred sight from the time when they "took to glasses". In a sense this may be true, but it is not entirely so. In the first place, before having the glasses, the patient's distant sight may have been not quite clear, but, as the condition came on gradually, he did not notice it, whereas the sharp contrast between having the glasses on or off is at once noticed. In the second place, when the hypermetropia is corrected by the lenses, the eyes are being used under physiological conditions and once they have, so to speak, "tasted with joys of freedom", they will not willingly return to the cramped conditions of continuous accommodation. This is where the exponents of "perfect sight without glasses" have their innings, but it is not usually a long one because accommodation is becoming steadily less each year and the struggle against glasses becomes correspondingly harder.

In addition to weakening of accommodation two other changes occur in the lens with advancing years. After the age of sixty, or so, the lens becomes more homogeneous and, therefore, less efficient for refracting light. In consequence of this, rays of light which previously came to a focus on the retina, now come to a focus behind it so that the eye is rendered hypermetropic and needs the assistance of glasses for distance, as well as for reading. On the other hand, if the lens is beginning to develop cataract of the type due to excessive sclerosis, its refractive index is increased and the eye becomes myopic. In this case, glasses will improve distant vision but the patient may find, to his joy, that he can read without them (so-called "second sight"). This change occurs fairly often in connection with diabetes, so it is as well to test for glycosuria. If this condition is present and is successfully treated, the myopia may disappear. There is a case on record of a man aged about fifty who was diabetic and required reading glasses when dieting properly. If he failed to observe his dietetic restrictions he developed a sufficient amount of myopia to enable him to read without glasses.

A simple method of telling whether a patient's defective vision is due to an error of refraction is to try the effect when he looks through a pin-hole in a piece of cardboard. If this causes marked improvement of vision, say from 6/60 to 6/12, one can be reasonably sure that an uncorrected error of refraction is, at least, one factor. The test type card should be well illuminated because the pin-hole cuts down the amount of light which enters the eye.

The second common cause of failing sight after middle-age is the development of cataract. Care should be taken in the diagnosis of this condition and no opinion should be expressed until after ophthalmoscopic examination. The lens may appear grey when examined by oblique illumination and yet a good pupil reflex be obtained with the ophthalmoscope. The precise diagnosis of the type of cataract present and the prognosis with regard to vision and operation are matters for the ophthalmologist, but some general observations will not be out of place. First, it is inadvisable to use the word "cataract" to a patient, if only a few opacities are found in the lens and if useful vision is still present. Secondly, quite a number of cataracts show no increase in density over a period of years and may, in fact, never come to maturity. Thirdly, if the fundus can be seen reasonably clearly with the ophthalmoscope, even though there are some lens opacities present, the patient's vision ought not to be seriously affected; if it is, some other cause must be looked for, *e.g.*, an error of refraction, or fundus changes. Fourthly, it is not necessary for the patient to wait until his cataract is mature for it to be removed. Circumstances alter cases, but in the general sense, operation is indicated if the vision in the better eye, with correction of error of refraction, is 6/18 or worse, and if reading in a good light and with adequate glasses has become really difficult or impossible. Dilatation of the pupil is sometimes of great help in patients whose cataract is limited to the central portion of the lens, but care must be exercised in the use of a mydriatic in case glaucoma should develop. For this reason it is advisable first to take the intra-ocular tension and, if it is normal, to instil a drop of homatropin and cocaine ($\overline{aa}$ 1%) since the effect of this can be neutralised with eserine, whereas that of atropine cannot. If no sign of tension occurs after dilatation of the pupil is complete and if there is a definite improvement in vision, gutt. atropine sulph. ½% may be ordered and be instilled. One drop alt. die is usually sufficient to keep the pupil dilated.

Glaucoma is a condition which should always be borne in mind when examining an elderly patient who complains of failing sight. The acute variety is sufficiently obvious in its manifestations to need no description here, but the chronic is easily missed, particularly as central vision may not be affected until a late stage. The most outstanding feature is loss of visual field, which loss appears first, as a rule, in the upper or lower nasal quadrant. It can be readily detected by the method of confrontation. In this, the examiner faces the patient at a distance of about two feet. If the field of the right eye is being tested, the patient looks at the examiner's left eye and he is thus able to test his field against his own, the other eye in each being, of course, covered. A suitable test object can be improvised from an ordinary pen by inserting a square of paper about 5 mm. in diameter between the points of the nib. Ophthalmoscopic examination may be of help in showing cupping of the disc but the differentiation of this from physiological cupping may require considerable experience. If arterial pulsation should be seen on the disc it is a very valuable positive sign of glaucoma unless aortic regurgitation is present. Venous pulsation has no pathological significance. Intra-ocular pressure as measured by the fingers is often difficult to estimate. If it is definitely raised the diagnosis is assured, but it is quite common for a case of chronic glaucoma to appear to have a normal pressure at the time of examination, and yet the disease to be in a fairly advanced state. In cases of doubt, the blind spot can be mapped out, using the same test object against a black background, *e.g.*, a piece of cloth hung on the wall with a white mark in the centre of it to which the patient directs her gaze. A sickle shaped extension from the upper, lower, or from both borders of the blind spot is characteristic of glaucoma.

Macular changes are a fairly common cause of failing sight in old people. The macula, or rather the foveal portion of the retina, is highly specialised and any interference with the central cones causes a marked diminution of visual acuity. This interference may come about in several different ways. Quite commonly, it begins with hæmorrhages which undergo changes, resulting in the formation of a mass of fibrous tissue which destroys the cones (central senile exudative retinitis). In other cases, the change is a purely degenerative one, the highly specialised central cones being the first structures to be affected by senility. Possibly there is a toxic element also and good results have sometimes followed the elimination of focal sepsis. In a third group of cases, the retinal degeneration is due to sclerosis of the underlying choroidal capillaries.

As a rule, it is necessary to dilate the pupil to see the macula in old people, and for this purpose it is wiser to use cocaine than homatropine. Two drops of 5% cocaine instilled, with an interval of five minutes, will produce adequate mydriasis in half-an-hour, or so. The patient should keep his eyes closed after the drops have been instilled, or the corneæ may become hazy. Ophthalmoscopically, there may be very little change in the early stages, all that is seen being perhaps a few specks of pigment and some lightening of colour in a small area of the fundus. This may be quite enough, however, to reduce vision to less than 6/60 and to render the eye useless for reading.

As a contrast to this condition, it is not uncommon to find apparently gross retinal changes at the macula in old people, with retention of normal, or nearly normal visual acuity. This is due to the development of colloid excrescences in the innermost layer of the choroid which is composed of elastic tissue. These localised thickenings press against the overlying pigment layer of the retina and cause some disturbance of it, with resulting ophthalmoscopic changes, but little or no visual impairment. The following case is an example of how deceptive this condition may be: A.B., aged about 65, came to hospital one morning and was found to see only 6/60 in each eye. He was examined in the dark-room and a note was made "gross macular changes right and left eyes". Later on in the morning, without knowing that he had been seen by someone else, I worked out A.B.'s refraction and found that with a + 3 D. sphere each eye read 6/6. I was entering this in his notes when I came across the statement recorded above, and a second visit to the dark-room was sufficient to establish the fact that the gross macular changes were, in reality, no more than colloid bodies.

Occasionally, patients are seen who complain of defective vision, which is not improved by correction of the error of refraction and in whom ophthalmoscopic examination is apparently negative. The majority of these are cases of tobacco blindness, though syphilis, diabetes and rapidly growing pituitary tumours may sometimes produce the same effect. They are diagnosed by the presence of a "cæco-central scotoma for colours", *i.e.*, there is an area embracing the fixation point and the blind spot, in which the patient is colour blind. This is easily established by the confrontation method. The patient cannot tell the colour of a small disc of paper when looking straight at it though if it is held a little to one side, in the nasal portion of his field of vision, he is able to name the colour. The condition of tobacco blindness is usually due to smoking shag tobacco or cigars, and there is generally some alcoholism which has produced arterio-sclerosis. In the early cases, the patient may be blind only to green and not to red and it is worthy of note that the presence of glycosuria increases the sensitiveness to the toxic effect of tobacco. Prognosis in the majority of cases is good, and if the

patient can be persuaded to relinquish tobacco entirely, improvement of vision will begin in about 6 weeks and may progress to complete restoration of normal sight.

Vascular lesions of the retina are not uncommon in old age, owing to increased blood pressure and fragility of the vessels. The association of retinal hæmorrhages with subsequent deterioration of macular vision has already been mentioned but the commonest lesion is thrombosis of the central retinal vein or one of its branches. In the latter case, the thrombosis usually occurs at the point where a branch of the central vein is crossed by a sclerosed branch of the artery, while if the main vein is thrombosed, the lesion originates at the lamina cribosa. This is a serious condition which may lead to the development of secondary glaucoma and loss of the eye. In other cases, the hæmorrhages slowly absorb, the circulation is re-established through anastamoses and a certain amount of useful peripheral vision is retained. Fortunately, the lesion is seldom bilateral. Ophthalmoscopically the veins are dilated and tortuous, the outline of the disc is blurred and there are numerous retinal hæmorrhages. Other vascular lesions may occur in the retina but these are not sufficiently common to need description in this paper.

Summing up, the common causes of failing sight after middle-age are:—

1. Errors of refraction.
2. Lens changes.
3. Glaucoma.
4. Macular changes in the retina—inflammatory or degenerative.
5. Tobacco—alcohol blindness.
6. Vascular changes.

This list is not by any means exhaustive, but it contains the conditions which most commonly bring about failing sight after middle-age.

The following routine examination will possibly be of service in cases of this type, and it can be carried out in any ordinary consulting room without any ophthalmic appliances except an electric ophthalmoscope, and a set of test types for distance and reading.

1. Examine the pupil reaction and palpate the globes through the upper lids with the patient looking downwards, to exclude rise of tension due to glaucoma.

2. Take the vision of each eye separately on the test type, which should be well illuminated. (If the patient has distance glasses he should be wearing them.) Repeat with the patient looking through a pin-hole. Definite improvement of vision (say 6/60 to 6/18) shows that an error of refraction is partly, or wholly, the cause of the defective vision.

3. Examine the fields by the method of confrontation and test for cæco-central colour scotomata.

4. Examine the eyes with an electric ophthalmoscope. Start with the + 12 lens in the sight hole and look for opacities in the pupil (cataract); then work the lenses down and look for floating opacities in the vitreous. When the fundus comes into view examine the disc for cupping and the arteries for pulsation, variability in calibre and indentation of the veins at the crossings. Examine the fundus generally for the presence of hæmorrhages and white areas. If nothing is found and the intra-ocular pressure is normal, dilate the pupils with cocaine and examine the macular regions of the retinæ. Look particularly carefully for hæmorrhages (which may be quite minute), streaks and masses of pigment, white areas and areas of rarefaction. In cases where only one eye is involved, it is wise to dilate both pupils so as to be able to compare the two maculæ.