

Visual Acuity, Geometric Optical Effects of Spectacles, and Aniseikonia

Visual Acuity

Basic Physiologic Concepts

Before discussing the clinical assessment of visual acuity in Chapter 11, certain theoretical considerations are in order. Visual acuity is a highly complex function that consists of (1) the minimum visible, (2) the minimum separable (hyperacuity), and (3) the minimum resolvable (ordinary visual acuity).

The *minimum visible* is concerned with detection of the presence or absence of a visual stimulus. Its threshold value is only 1 second of arc; however, Westheimer³⁹ pointed out that the minimum visible is a brightness rather than a spatial-visual threshold function.

The *minimum separable* concerns the judgment of the location of a visual target, usually relative to another element of the same target. The best-known example is Vernier acuity, that is, the ability to detect minimal differences in the horizontal alignment of two vertical lines. The threshold of the minimum separable in normal observers is only 2 to 10 seconds of arc.⁴⁰ In view of this extraordinary sensitivity, the term *hyperacuity*, frequently used as a synonym for the minimum separable, seems appropriate.

More familiar to the clinician and therefore occasionally referred to as “ordinary visual acuity” is the *minimum resolvable*, that is, the ability to determine the presence of or to distinguish between more than one identifying feature in a visible target.³⁹ The threshold of the minimum resolvable is between 30 seconds and 1 minute of arc.

When one tests visual acuity with Snellen letters or number charts, picture charts, Landolt rings, or the illiterate E, the patient is asked to name the test objects or to specify a critical part of them. All these tests are designed to present an object or a series of objects with a critical dimension of 1 minute of arc when viewed from a standard distance (generally 6 m or 20 ft). Patients unable to recognize these objects at the standard distance are tested with increasingly larger objects that have a critical dimension of 1 minute of arc at greater distances (e.g., from 12 m, 15 m, or 60 m). An eye with substandard vision is then said to have a visual acuity of 6/12, 6/15, or 6/60. Many people have better than standard vision in at least one eye, for example, a vision of 6/4.5 or 6/3, which indicates that they are able to recognize objects which from the standard distance have critical dimensions smaller than 1 minute of arc. It is better therefore not to speak of 6/6 vision

as normal but rather as *standard vision*. Also, recognition improves when the subject is allowed to use both eyes. Binocular visual acuity, as determined in clinical tests, is better than monocular visual acuity. The reasons for this are complex and not well understood.

The recognition task is highly involved and undoubtedly is composed of resolution and localization, without which there could be no recognition. Recognition and naming are higher visual functions that bring into effect other variables, such as the ease with which certain numbers or letters are recognized. The picture charts used for the visual screening of preliterate or illiterate patients have to be designed by keeping cultural and geographic differences in mind. Once, while visiting a visual screening station in a remote part of Central Africa one of us (GKvN) observed children and adults staring in frustration at preliterate test charts used by the health workers and depicting objects familiar to every child in the United States, such as birthday cakes, Christmas trees, and houses, none of which were known by those to be screened. Clearly, the objects to be depicted have to be familiar to be identified. On the other hand, identification of a familiar object becomes possible even if only parts thereof are seen because higher integration functions defined as gestalt are involved in the recognition process. Ideally, stimuli should be used which are void of any meaning, such as Landolt Cs or illiterate Es.

The basis for the resolving power of the eye is generally assumed to be the structure of the retinal mosaic. It is not necessary to consider whether the dimensions of the retinal mosaic or the optical aberrations of the eye are the limiting factor of visual acuity and how higher visual acuities, such as those obtained by the Vernier method, come about. The reader is only reminded that the cones are thinnest and most densely packed in the rod-free area of the fovea, which has a diameter of about 1.7° , and that the cones in that area, each of which has a separate nerve fiber, have an average width of 2.5μ . These facts are recalled because the thought has been expressed that some of the properties of the fovea of amblyopic eyes might be accounted for by the encroachment of rods into the cone-free area.

Variables Affecting Visual Acuity

Numerous variables affect visual acuity, but only some of them will be discussed here. For a more complete discussion, see that of Westheimer.⁴⁰

RETINAL ECCENTRICITY. That visual acuity depends on the location of the retinal stimulus and decreases sharply with an increase in distance of the image of an object from the center of the fovea is well-known. Some quantitative information on this subject must be given since it has an important bearing on the clinical and theoretical aspects of amblyopia (eccentric fixation) (see Chapter 14).

The most frequently quoted work is that of Wertheim,³⁸ whose number for the decrease in visual acuity in the horizontal meridian (Fig. 7-1) is the one generally reproduced. The number shows that visual acuity is reduced to 6/12 at 2.5° and to 6/30 at 10° on the nasal side of the fovea. On the temporal side visual acuity decreases somewhat more rapidly. Of special importance is Wertheim's observation that it decreases more sharply below and, especially, above the fovea, so that lines connecting points of equal visual acuity are elliptic, paralleling the outer margins of the visual field. Later studies have given more detailed information about reduction in visual acuity in the region from the center to 10° from the fovea²⁶ (Figs. 7-2 and 7-3). Peripheral visual acuity declines with advancing age.¹⁴

A decrease in visual acuity from the center to the periphery must be related in some way to the retinal mosaic. Ludvigh's data²⁶ (see Fig. 7-3) indicate that the visual acuity curve does not parallel linear or areal density of cones from center to periphery. Weymouth⁴² made the intriguing and reasonable suggestion that the resolving power of a retinal area depends not on the number of cones present but on the number of perceptual units in that area. It is generally believed that the number of receptors connected by a single fiber to the brain defines the extent of a sensory unit.³¹ The ganglion cells are the cells of origin of the optic nerve fibers. Weymouth, who proposed that the density of ganglion cells rather than that of the cones should be related to the minimal angle of resolution, showed that this minimal angle of resolution, the reciprocal of visual acuity, was linearly related to the distance from the fovea. He also found a linear relationship between the density of the ganglion cells and their distance from the fovea.

LUMINANCE AND STATE OF ADAPTATION. The effect of luminance on visual acuity has been well summarized by Riggs,³³ who stated that visual acuity is poor at scotopic levels where parafoveal or peripheral rod receptors predominate. As the

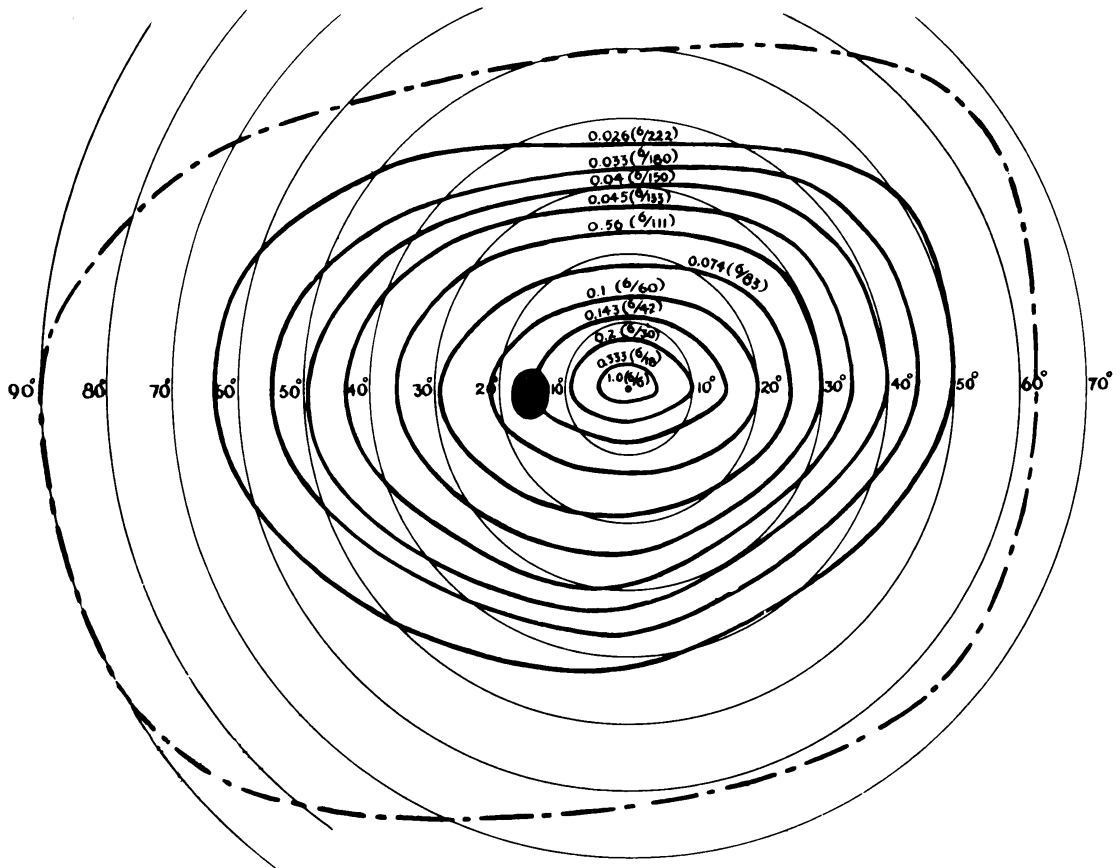


FIGURE 7-1. Visual acuity of the retinal periphery. Continuous black lines indicate points of equal visual acuity. Note that the gradient of visual acuity is steepest in the upper half of the retina. The decline in acuity with eccentricity is least on the temporal side. The broken line indicates the peripheral limits of the visual field. (Data from Wertheim T: *Über die indirekte Sehschärfe*. *Z Psychol Physiol Sinnesor* 7:172, 1894; modified from Hofmann FB: *Die Lehre vom Raumsinn*. In Axenfeld T, Elschnig A, eds: *Graefe-Saemisch Handbuch der gesamten Augenheilkunde*, ed 3, vol 3. Berlin, Springer-Verlag, 1925.)

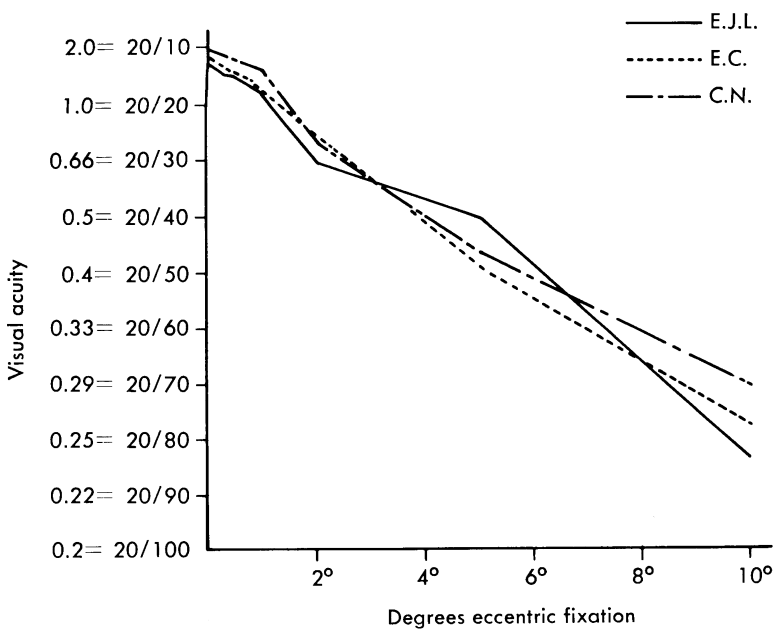


FIGURE 7-2. Decrease in visual acuity for three subjects from the fovea to 10° eccentricity. (From Ludvigh E: *Extrafoveal visual acuity as measured with Snellen test letters*. *Arch Ophthalmol* 25:469, 1941.)

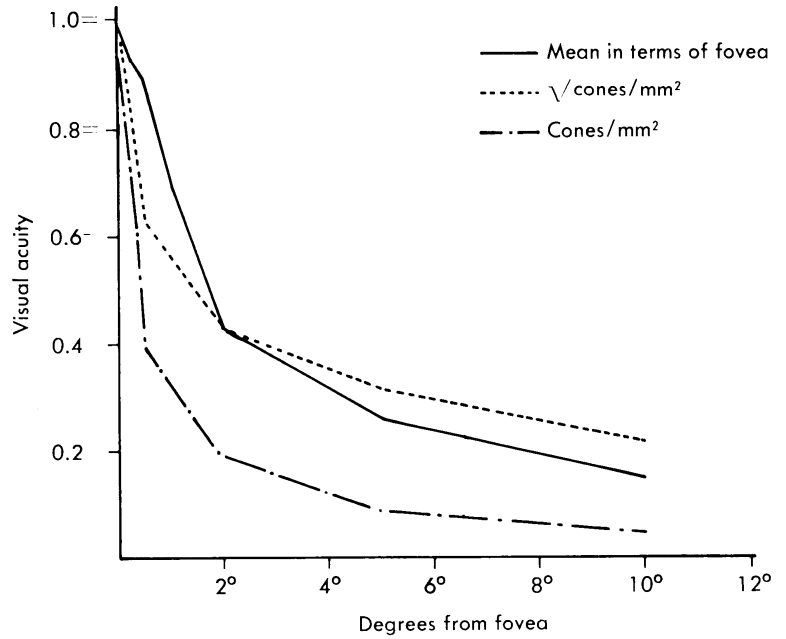


FIGURE 7-3. Data indicating that the decrease in visual acuity from the fovea to 10° eccentrically (*solid line*) does not parallel the linear (*broken line*) or areal (*dotted line*) density of the cones. (From Ludvig E: Extrafoveal visual acuity as measured with Snellen test letters. Arch Ophthalmol 25:469, 1941.)

level of intensity is raised, thresholds of the cone receptors are exceeded and acuity rises steeply. With a further increase in intensity, this maximum acuity is maintained over a wide range of increas-

ing intensities. This is, in fact, a description of the classic curve of König for the relation between visual acuity and intensity, which uses objects on a white background (Fig. 7-4).

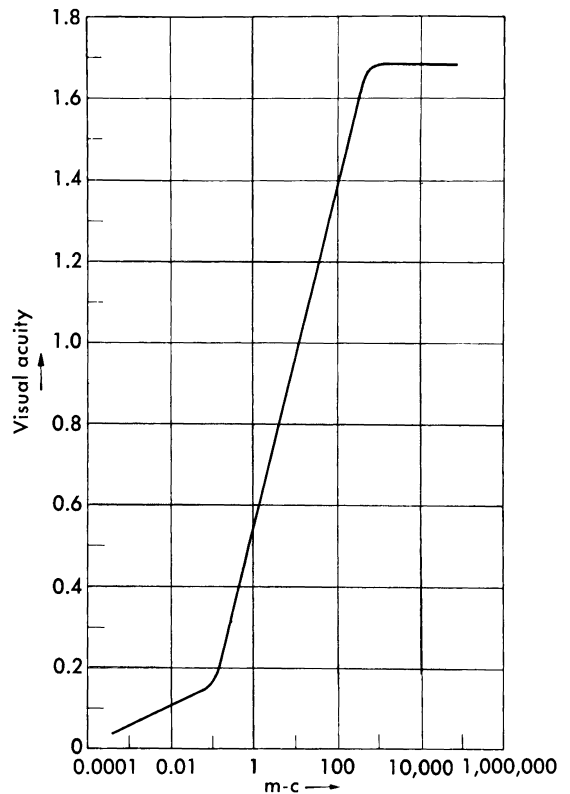


FIGURE 7-4. Curve of König showing relationship of visual acuity and luminance. The ordinate is in meter candles ($m \cdot c$).

The pupil size is also involved in the effect of luminance on visual acuity. A large pupil allows more light to enter the eye but increases the effect of the optical aberrations. These aberrations are minimized with a small pupil, but a very narrow pupil reduces visual acuity by markedly reducing retinal illuminance and by increasing the effect of light diffraction. Generally speaking, visual acuity is optimal with an intermediate-size pupil of about 2 mm, but the optimal size varies with conditions of luminance, size of test object, and other factors.

CONTRAST. In the discussion of contrast effects, two concepts must be differentiated. The *objective or photometric contrast* refers to differences in luminance of adjacent fields or objects. The *subjective or physiologic contrast* refers to such subjective phenomena as the change in apparent brightness of objects of a given luminance, which depends on the luminance of the surround. In general, the visual acuity decreases with reduction in objective contrast, this effect being more pronounced the smaller the test object.

The effect of subjective contrast is of greatest importance for vision. The borderline between a bright and dark surface produces a blurred, unfocused retinal image caused by the optical aberrations of the eye and the veiling effect of stray light. These effects are offset by subjective contrast. The image of a white field appears whiter, the image of a dark field darker, and the borderline sharp when the two border on each other. Tschermak-Seysenegg³⁷ stated that without the subjective contrast phenomenon one would be unable to read. Ludvigh²⁷ showed that with low degrees of contrast, visual acuity varies markedly; but at high contrast levels, relatively great changes in photometric contrasts have little effect on visual acuity.

The mechanisms underlying subjective contrast have been and continue to be a matter of controversy. Helmholtz,²¹ and others after him thought of contrast as dependent on a judgment of relative brightness, thus making it the result of highest nervous system activity. Hering²² considered contrast to be a physiologic change in sensation in the sense that the sensation of brightness depends on the interplay between the illuminances of a given retinal area and its surround. The electrophysiologic findings of Hartline²⁰ on the lateral inhibition in the *Limulus* eye and of Kuffler²⁵ on retinal receptor fields, as well as the psychophysical studies by Harms and Aulhorn,¹⁹ established that sensitivity to each side of adjacent fields of different retinal illuminance is reduced, pointing

to the physiologic mechanisms that may underlie subjective contrast.

EYE MOVEMENTS. The eyes are never completely motionless, even with a strenuous effort at steady fixation (see Chapter 4). These miniature fixation movements may have the effect of blurring or “smearing” the retinal image, just as the motion of an object or of a camera may produce a blurred photographic picture. They also may have the opposite effect of enhancing the neuronal activity on which visual acuity depends by allowing retinal receptors to scan the contours of an object.³³

Riggs and coworkers³⁴ showed that there is no evidence to prove that eye movements serve to improve visual acuity. Ratliff³² determined the instantaneous value of visual acuity by presenting a grating test object for 75 ms and simultaneously recording the eye movements for an interval beginning before the test exposure and ending after it. The involuntary drifts of the visual axis were clearly a hindrance, and the rapid tremors were detrimental to visual acuity. No evidence was found that scanning the retinal image contributed to visual resolution, as has been postulated by some investigators.^{3, 43}

Whether miniature eye movements have evolved to counteract image fading (Troxler effect)²³ or whether they are simply the expression of random noise in the eye movement control system is not clear.¹³

CONTOUR INTERACTION. Visual acuity can be reduced by the spatial arrangement of additional contours in the field of vision in amblyopic patients (see Chapter 14). Flom and coworkers¹⁸ investigated this phenomenon in normal subjects and pointed out that it is related to the size of the receptive field associated with the retinal region used to fixate the target. Contour interaction is not limited to ordinary visual acuity but also interferes with Vernier acuity⁴¹ and stereoacuity.¹² It is highly exaggerated in amblyopia where it causes the crowding phenomenon (see Chapter 14). Thus the spacing of optotypes on acuity charts must not be left to chance, in which case visual acuity will differ depending on which chart has been used. Rather, the spacing between letters and lines should be related to the letter size.

Geometric-Optical Effects of Spectacles

Whenever eyeglasses are worn, a series of far-reaching visual changes are introduced. These ef-

fects are discussed extensively in treatises dealing with the geometric optics of spectacles (e.g., see Erggelet¹⁶ and Ogle³⁰).

To begin with, spectacles have a profound effect on the neuromuscular mechanism of the eyes through their influence on accommodation. The region within which the spectacles wearer must accommodate, as well as the range of accommodation, is affected. For example, a young uncorrected hypermetrope of 4D will have to accommodate by that amount to see clearly at infinity and correspondingly more for near fixation. If a full correction is worn, the person does not have to accommodate for infinity. On the other hand, an uncorrected myope of 4D can do close work at 25 cm without using accommodation. If the correction is worn, close work requires accommodation.

The association between accommodation and convergence is discussed fully in Chapter 5. It is clear from what is known about this association that the excessive convergence that the uncorrected hypermetrope must overcome is automatically relaxed when the refractive error is corrected. In contrast, the refractive correction that the myope wears stimulates convergence. These factors form the basis of treatment of certain forms of strabismus by spectacles.

Furthermore, spectacles lenses, which change the direction in which object points appear in indirect vision, cause changes in perspective and the perception of space. These changes are particularly evident to the spectacles wearer with a high refractive correction, for instance, after cataract extraction. They are minimized by the use of contact lenses.¹¹

Spectacles change the size of the retinal image in emmetropic eyes and the blurred image in uncorrected ametropic eyes. For geometric-optical reasons, these changes in size occur only if the patient has a refractive ametropia. If the ametropia is axial in origin, a correcting lens placed into the anterior focal plane of the eye produces an image equal in size to that of the emmetropic eye. This is known as Knapp's rule.^{29, p. 264} This rule has been interpreted to the effect that contact lens correction of an anisometropia caused by an axial ametropia may actually induce aniseikonia, whereas correction with a spectacles lens will not. This does not necessarily hold true in all clinical situations. While the geometric-optical basis of Knapp's rule is correct, it has been shown that aniseikonia may occur after spectacles correction in spite of the axial nature of anisomy-

opia.^{4, 9, 24, 35, 36} The reason for this is a reduction of retinal receptor density caused by stretching of the posterior pole in high myopia that causes perceptual micropsia despite equal size of the retinal images (*basic aniseikonia*).

The effect of spectacles lenses on measuring the angle of strabismus is discussed in Chapter 12.

Aniseikonia

There is one effect produced by spectacles to which the wearer does not always readily adapt and which may cause great difficulties. Whenever refractive ametropias in the two eyes of a person are different (i.e., when there is an anisometropia), the corrected retinal images of the two eyes, and consequently the two visual images, differ in size. This condition has been termed *aniseikonia*, literally meaning unequal imagery.

Aniseikonia resulting from a corrected refractive anisometropia may be termed *refractive aniseikonia*. However, this condition may also exist in patients with an equal ametropia in the two eyes or who may have no ametropia at all. This type of image size difference may be termed *basic aniseikonia*. In this case, the aniseikonia is presumably a result of a difference in the distribution of the retinal elements, or rather their spatial values, in the two eyes. Examples of basic aniseikonia were mentioned previously and are also provided by patients with epiretinal membranes and vitreomacular compression that may cause aniseikonia from separation or compression of photoreceptors.^{6, 15}

The incongruities of the retinal images may be of different types. The image size may differ or may be the same in all meridians (*overall size difference*), or one of the two images may be larger only in the horizontal or vertical meridian (*meridional size difference*). The images may differ in oblique meridians (*oblique meridional size difference*), or they may be *asymmetrically* different in the two eyes (e.g., larger on the temporal side in one eye than on the nasal side). Finally, there may be *irregular differences*, as in a patient with a healed retinal detachment, and two or more of the listed image size differences may be simultaneously present.

In this book we consider aniseikonia only to the extent that it has a bearing on fusion and spatial orientation. Easy and comfortable fusion of the two retinal images demands that they be as

equal as possible in brightness, form, and size. When aniseikonia is present, the last requirement is not fulfilled. Thus, *aniseikonia may be an obstacle to fusion*. The mechanism of this obstacle is the rivalry set up between foveal and peripheral fusion (see Chapter 4). If the centers of the images are fused, the peripheral margins are not; if the peripheral margins are fused, the centers are disparately imaged. If the aniseikonia is very small, the difficulty is negligible. If the aniseikonia is very large, say, size differences of 5% or more, the patient as a rule will suppress part of the image of one eye, thus eliminating symptoms. This must be done at the expense of normal binocular vision. Not all patients are able to suppress equally well. This is especially true if the aniseikonia is of relatively moderate amount, say, between 0.75% and 2.5%; then the compulsion to fuse often prevails, with resulting subjective symptoms. On the other hand, suppression may be strong enough to cause a deviation of the visual lines, a strabismus. Bielschowsky⁸ described a most perplexing case of horror fusionis (see Chapter 13) which responded to a complex correction with iseikonic lenses.

Aniseikonia also has an effect on spatial localization. If a patient has an image size difference in the horizontal meridian, the image of one eye is larger in that meridian; in other words, there is a horizontal disparity of the retinal images. Fusion of horizontally disparate images produces a stereoscopic effect (see Chapter 2). One should therefore expect a stereoscopic effect, a spatial distortion, when aniseikonia is present in the horizontal meridian. Indeed, such a spatial distortion can always be found and is readily explained on a geometric basis.

Assume that the visual lines of a normal observer intersect in symmetrical convergence at point F on a plane on which there are two points, P and N , seen in peripheral vision (Fig. 7-5). If the image of the observer's right eye is now magnified in the horizontal meridian by an appropriate lens, all horizontal distances on the plane are magnified and PFN is increased to $P'F'N'$ for the right eye. This means that the object point P , originally imaged on the retinal point p_r in the right eye, now stimulates the more temporally located point p'_r . The image of the point N is displaced in the right eye from n_r to n'_r . No change in the image size of the left eye has occurred. If the horizontal disparity between the two eyes that has been created is not too large, sensory

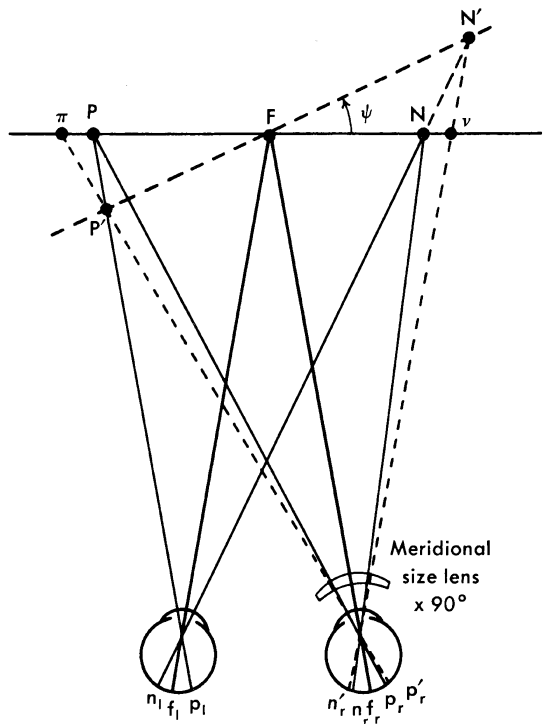


Figure 7-5. Tipping of a frontoparallel plane around a fixation point when the image of the right eye is magnified horizontally by a meridional-size lens placed at axis 90° . (From Burian HM: Influence of prolonged wearing of meridional size lenses on spatial localization. Arch Ophthalmol 30:645, 1943.)

fusion of the horizontally disparate retinal images will occur and must create a stereoscopic effect. Object P will appear to have advanced to point P' and object point N to have receded to N' , since only points situated objectively at P' and N' could fulfill the conditions of stimulating simultaneously the retinal elements p_l and p'_r , and n_l and n'_r . *The impression is created that the plane has rotated around the fixation point F .* In general, objects in the half of the visual field pertaining to the eye with the relatively larger retinal image in the horizontal meridian appear farther away than the fixation point, whereas those in the half of the visual field pertaining to the eye with the relatively smaller retinal image appear to be closer.

Vertical and oblique meridional aniseikonic errors also produce typical distortions of space. All those stereoscopic effects are quantitative, and the empirical data are in good agreement with the theory, so much so that a clinical instrument for the *measurement of aniseikonia* was designed (space eikonometer) based on these stereoscopic effects. For clinical purposes, especially in strabis-

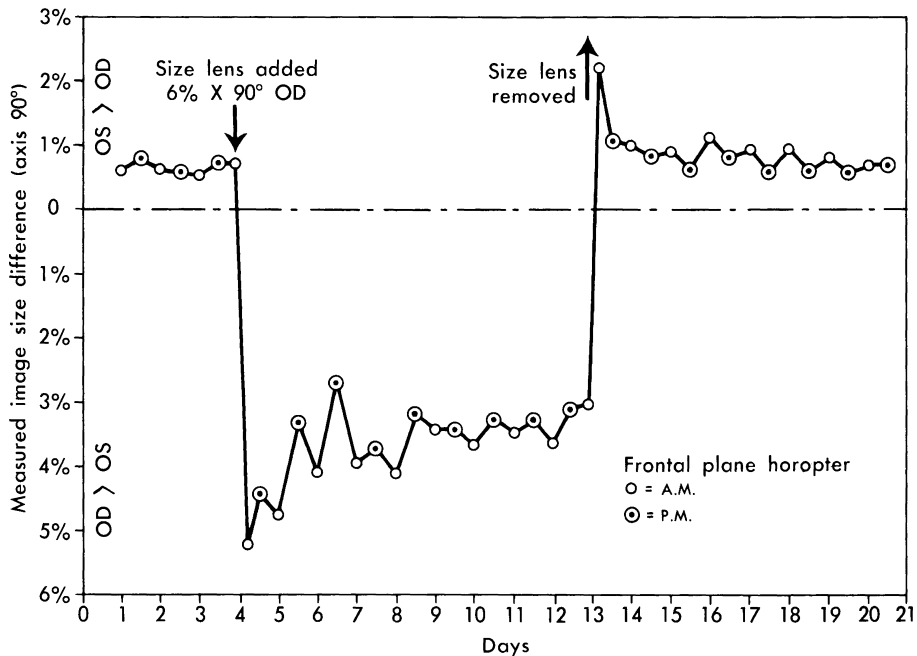


FIGURE 7-6. Data showing that prolonged wearing of a meridional-size lens has only minimal adapting effect on the frontal plane horopter. (From Burian HM: Influence of prolonged wearing of meridional size lenses on spatial localization. *Arch Ophthalmol* 30:645, 1943.)

mic patients, aniseikonia is determined with Aulhorn's phase difference haploscope (see Chapter 4). Awaya and coworkers⁵ developed a simple and useful "new aniseikonia test"* that is based on image separation with red and green spectacles. In comparing this test with the eikonometer, McCormack and coworkers²⁸ found that the Awaya test may underestimate the degree of aniseikonia (see also Yoshida and coworkers⁴⁴). Another new test,† combining the features of the Aulhorn phase difference haploscope (see Chapter 4) with those of the Awaya test was introduced by Esser¹⁷ and also has the advantage of permitting aniseikonia measurements in the presence of manifest strabismus. Unfortunately, neither of these instruments is available in most clinical settings.

Why do patients with corrections for anisometropia generally not complain about spatial distortions? The answer is that many patients will report that during the first day or two of wearing a new correction they experience various changes in the appearance of their surroundings; for example, the floor may appear to have slanted to one side, or it may have seemed to slant up or down in front of

them. These distortions are caused by aniseikonia. However, after a short while the distortions disappear. By what mechanism do they disappear? The first thought is that the aniseikonia may have been compensated for by some physiologic mechanism. This is not the case. Burian¹⁰ demonstrated in an experimental study, using the subjective frontoparallel plane in the horopter apparatus as a criterion, that the amount of aniseikonia measured was changed by only a fraction after the phenomenologic adaptation had taken place, regardless of how long the adaptation had lasted (Fig. 7-6).

What actually happens is that there are two general classes of clues to spatial orientation: the *binocular stereoscopic clues* and the *uniocular experiential clues* (see Chapter 2) Only the stereoscopic clues can be affected by aniseikonia, for they alone depend on the disparity of retinal images. The experiential clues remain unaffected. If stereoscopic perception is changed suddenly in such a way as to convey an incorrect impression of the surroundings, for instance, by a pair of new spectacles, stereoscopic clues will at first dominate and the environment will appear distorted. In time, however, uniocular clues—born from experience and active also in binocular vision—make themselves felt and gradually dominate stereoscopic clues. One learns how to disregard or suppress

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stereoscopic clues, and under the influence of uniocular clues the surroundings resume their normal appearance.

This brief discussion of some of the basic concepts of aniseikonia in a book on the neuromuscular anomalies of the eyes is justified because it enlarges insight into the binocular function. Since aniseikonia is an obstacle to fusion it could be a factor in the etiology of certain types of comitant strabismus (see Chapter 9). The response of normal observers to artificially induced aniseikonia under certain experimental conditions, as well as the responses of naturally aniseikonic patients to the same experimental situations, also gives an inkling of some of the important adaptive mechanisms in patients with neuromuscular anomalies of the eyes (see Chapter 13).

The treatment of aniseikonia has become almost a lost art since the Dartmouth Eye Institute closed its doors in 1947. Most of what is known about aniseikonia today is based on the work of such Dartmouth notables as *Ames*, *Lancaster* (who coined the term), *Linksz*, *Ogle*, *Burian*, and *Boeder*. Although the clinical significance of aniseikonia as a cause of asthenopia was undoubtedly overemphasized in those days, recent interest in aniseikonia has surged since the advent of keratorefractive surgery and the optic calculation of intraocular lens implants. A discussion of the clinical management of aniseikonia exceeds the purpose of this text and the reader is referred to other sources.^{1, 2, 7.}

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