CHAPTER 5

The Near Vision Complex

When an observer transfers binocular fixation from an object at one fixation distance to one at another fixation distance, changes in the refractive power of the eyes and in the relative position of the visual axes are required to maintain sharply defined retinal images and to preserve binocular fixation. Thus when an observer binocularly fixates an object situated in his or her median (sagittal) plane at a certain distance (say, 6 m) and when the object then approaches the observer's head, the eyes must increase their refractive power more and more to produce a continuously well-focused image of the approaching object on the retinas. At the same time, the angle formed between the visual axes must grow increasingly larger to allow the object's images to remain on the two foveae. The opposite process takes place when an object recedes from near fixation. Refraction and the position of the visual axes also change if the change in fixation distance is discontinuous, as in a jumplike change from one fixation distance to another.

The process by which the refractive power of the eyes is altered to ensure a clear retinal image is known as *accommodation*. The change in the relative position of the visual axes is called *convergence* when the angle formed by the visual axes increases and *divergence* when this angle decreases. In addition, with fixation at near vision, *pupillary constriction* occurs.

The association of accommodation, convergence, and pupillary constriction during fixation at near may be termed the *near vision complex*. This triad of events is not a true reflex but rather a synkinesis, that is, an association of different functions elicited by nearness of the observed object. Each of these functions can be dissociated from one another.

Accommodation

A detailed discussion of the process of accommodation and the mechanisms that have been proposed is outside the scope of this book. Fundamentals will be discussed only to help the understanding and investigation of neuromuscular anomalies of the eyes.

Mechanism of Accommodation

There is no disagreement that a change in the shape of the lens—an increase or decrease in curvature and thickness of its central parts that produces an increase or decrease in the dioptic power of the eye—is the basic mechanism underlying accommodation. Nor is there any disagreement that this change in the shape of the lens is caused by a contraction of the ciliary muscle. Whether this contraction produces loosening of the zonular fibers[23] or tightening with forward pull on the choroid and increased intravitreal pressure, affecting the periphery of the lens,[53] or some other more complex effects, is not essential to this discussion. It is sufficient to know only that the change in shape of the lens, resulting from a contraction of the ciliary muscle, represents the peripheral mechanism of accommodation set in motion by a central mechanism of accommodation, which in turn responds to a stimulus to accommodation.

The stimulus to accommodation is the blurred retinal image. The afferent pathway is represented by the visual pathway up to area 17 and continues to area 19. The ciliary muscle is innervated by cranial nerve III, with the majority of the fibers being relayed through the ciliary ganglion from the midline nucleus (Edinger-Westphal) of the oculomotor complex. The projection of area 19 to the midbrain oculomotor complex is by way of the internal corticotectal tract, but no details of the supranuclear connections are known. Jampel[27] succeeded in increasing refraction in both eyes of the macaque accompanied by convergence and in some cases pupillary constriction by unilateral faradic stimulation of area 19.

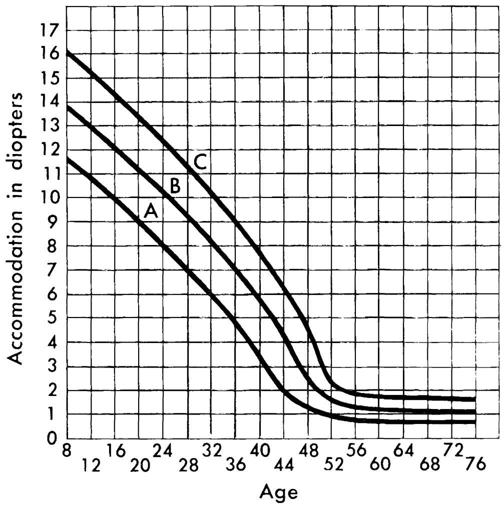

FIGURE 5–1. Duane's standard curve of accommodation in diopters in relation to age. A, lowest values; B, average values; C, highest values. (From Duane A: Studies in monocular and binocolar accommodation with their clinical applications. Am J Ophthalmol 5:865, 1922.)

Units of Measurement of Accommodation and Definition of the Prism Diopter

Accommodation is measured in *diopters* (D), that is, in terms of the reciprocal of the fixation distance. Thus if the fixation distance is 1 m, the accommodation is said to be 1D; if 1/2 m, 2D; if 1/3 m, 3D; and so forth. There is a near fixation distance inside which the eyes cannot effectively accommodate. This limiting distance is termed the *near point of accommodation* (NPA). It is assumed that at optical infinity no accommodation is exerted by emmetropic or corrected ametropic eyes. This distance is termed the *far point of accommodation* (FPA). The range from infinity to the near point of accommodation is the *range of accommodation*. The distance of the near point—and therefore the range of accommodation—is a function of advancing age. The standard curve describing this relationship was established by Duane in 1912 and revised in 1922 on the basis of 4200 cases[12] (Fig. 5–1). During the second, third, and fourth decades, the loss of accommodation is gradual (on average, 2.0D in the second decade and 2.9D in the fourth decade). It is very rapid in the fifth decade and after that asymptotically reaches a minimum. In measuring the NPA in older children and teenagers we have found that the average values determined by Duane are about 1.5D higher than the average values in our clinic population.[37]

Sympathetic Innervation

In all probability there is also a sympathetic innervation of the ciliary muscle so that instillation of sympathicomimetic drugs in humans or stimulation of the cervical sympathetic ganglion in animals may increase hypermetropia. One cannot be sure whether this effect is the result of vasoconstriction with reduction in the mass of the ciliary body and increased tension of the zonular fibers and flattening of the lens. There also may be a direct effect on the ciliary muscle. In any event, the effect is small compared with the positive accommodation resulting from parasympathetic impulses.

Convergence

As stated in Chapter 4, convergence is a disjunctive (vergence) movement that produces an increase in the angle formed by the visual axes, ordinarily through simultaneous, synchronous adduction of each eye. The result is a convergent position of the visual axes. The term *convergence* is applied equally to the movement of the eyes, to the convergent position they assume through this movement, and by implication to the innervational pattern required to maintain the convergent position. A terminological differentiation would be desirable for clarity's sake, but one does not exist at present. In general the meaning of the word "convergence" is readily understood from the context in which it is used, and it will be used in this volume without the cumbersome qualifying

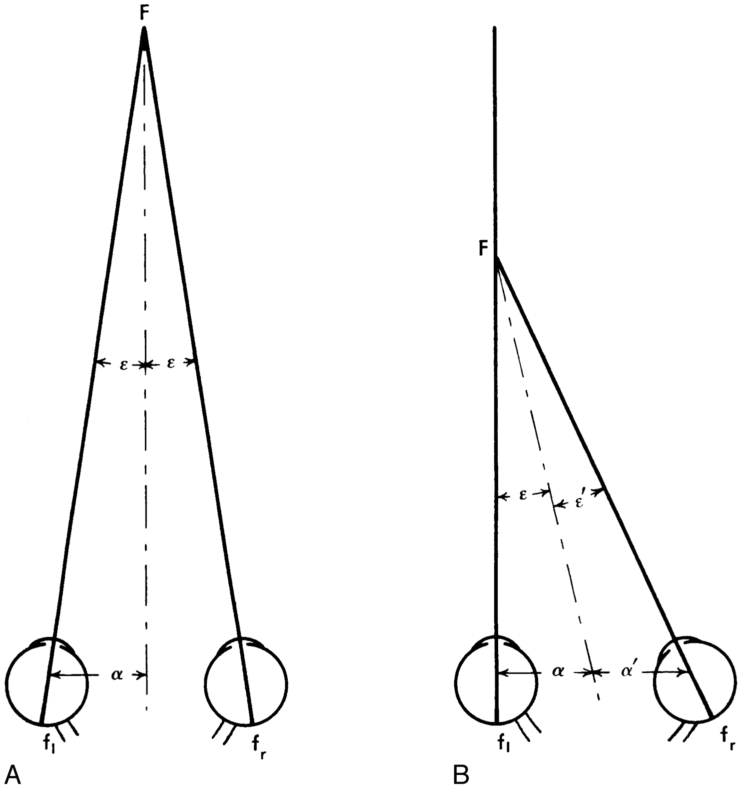

FIGURE 5–2. *A*, Symmetrical convergence of the visual lines; a equals one half the interpupillary distance. *B*, Asymmetrical convergence of the visual lines.

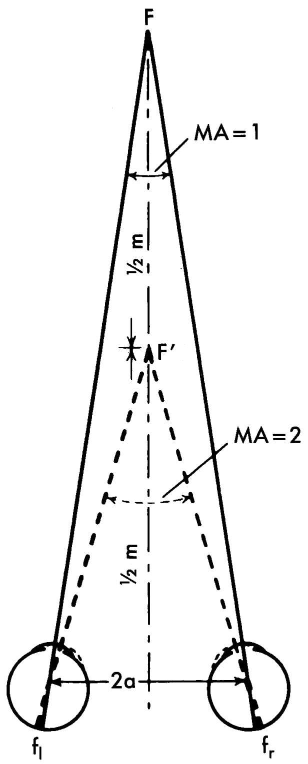

FIGURE 5–3. Definition of meter angle (MA). The visual lines converge 1 MA at 1 m fixation distance, F, and 2 MA at 1/2 m fixation distance, F′.

words "movement" or "position," except for the sake of clarity.

If the fixated object is situated in the median (sagittal) plane of the head, equal angles are formed between each visual axis and a line erected perpendicular at the midpoint of a line connecting the centers of rotation of the eyes (*symmetrical convergence*, Fig. 5–2*A*.) If the fixation point lies to the right or left of the median plane, the angles differ (*asymmetrical convergence*, Fig. 5–2*B*.)

The nearest point on which the eyes can converge is the *near point of convergence* (NPC). As a rule, it is much closer to the eyes than the NPA and in general does not change with age. In clinical practice an NPC of 10 cm is considered adequate. The proper clinical determination of the NPC and its value in assessing neuromuscular anomalies of the eyes are discussed in Chapter 12.

Units of Measurement of Convergence

The unit for the measurement of convergence is the *meter angle* (MA), introduced by Nagel.[35] This unit is numerically the reciprocal of the fixation distance in meters; that is, it is formed in analogy to the diopter (Fig. 5–3).

The MA is defined as the amount of convergence required for each eye to fixate an object located at 1 m from the eyes in the median plane. Thus if the fixation distance is 1 m, an emmetropic subject must converge 1 MA and accommodate 1D; if the fixation distance is 1/2 m, the MA equals 2 and the accommodation required is 2D; if the fixation distance is 2 m, the MA equals 1/2 and the required accommodation is 1/2D; and so on. The MA is a convenient unit since it relates convergence numerically to accommodation, but the values are precise only if the reference points for the two functions are identical. This usually is not the case, since accommodation is ordinarily specified from the spectacle plane and convergence from the center of rotation of the eyes.

Also, we are accustomed to thinking of convergence in terms of the angle formed between the two visual axes rather than the amount of vergence of each eye, which is the most convenient and common usage, particularly for clinical purposes. The amount of vergence of both eyes may be designated as the large MA to distinguish it from the small MA of Nagel.[35]

The large MA is a relative unit and is the same in every person. The absolute or actual amount of convergence is greater the larger the interocular separation. Consequently, for identical MAs the absolute amount of convergence varies for different individuals.

If the interocular separation, 2a, and the fixation distance, d, are known, the angle (half the angle of symmetrical convergence) can be found by the formula $\tan \epsilon = 2a/d$ (see Fig. 5–3).

The *prism diopter* is the unit of measurement of an ophthalmic prism, and in clinical usage convergence is described in terms of prism diopters. A prism is defined as having the power of 1 prism diopter ($^{\Delta}$) when it displaces the visual axis, referred to the center of rotation, by 1 cm at a distance of 1 m (Fig. 5–4), and 1^{Δ} is equivalent to 0.57° of arc. Since prisms or their equivalents are used to measure vergences and the relative position of the eyes, the term prism diopter has acquired a broader meaning and also is used clinically to specify vergences and ocular deviations.

The angle of 2ϵ of symmetrical convergence is found by multiplying 1/d (the MA of convergence) in meters by the interocular distance in centimeters. Thus if a person converges at 1 m and has an interocular separation of 6.5 cm, the convergence this person must exert is 6.5^{Δ}. If an object is fixated at a distance of 1/3 m, there must be a convergence of 19.5^{Δ}. This is known as a person's *convergence requirement* for the given fixation distance.

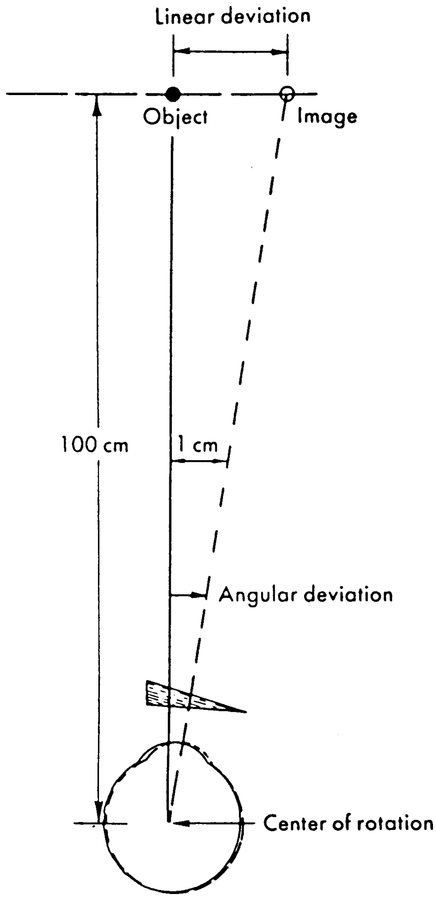

FIGURE 5–4. Diagram demonstrating definition of prism diopter.

Strictly speaking, one should specify the interocular separation as the distance between the centers of rotation. For clinical purposes the term *interpupillary distance* is sufficient. To the distance d, measured habitually from the spectacle plane, approximately 0.027 m should be added to account for the distance from the spectacle plane to the center of rotation. In common clinical usage, this may be safely disregarded.

Components of Convergence

VOLUNTARY CONVERGENCE. Convergence is the only vergence movement that also may be voluntary, which means that convergence may be exerted without an external stimulus to converge. However, in the ordinary use of the eyes, convergence is a reflex, as are all other vergence movements.

Jampolsky[28] denied the existence of voluntary convergence and believed it to be a "trick" when someone converges the eyes seemingly at will. In his opinion such a person is making an accommodative effort even without a fixation object and converges accordingly. Jampolsky based this view on his observation that with alleged voluntary convergence a pupillary constriction is always present.

It is difficult to prove that voluntary convergence can be independent of accommodation and pupillary constriction is not absolute proof for the presence of an "accommodative effort." Moreover, voluntary accommodation with one eye occluded does not necessarily trigger the change of vergence usually associated with accommodation.[32] Many people are capable of maintaining the convergent position of their eyes even when the fixation object has been removed. Since this obviates the need for accommodation, the maintenance of convergence is thought to be achieved by voluntary convergence. Whether or not this is correct, to evaluate a patient's ability to maintain convergence is helpful in forming a judgment about the quality of overall convergence function (Chapter 12). In a clinical setting, voluntary convergence is occasionally used by children to gain the attention of or cause anxiety in their parents. Indeed, we have on more than one occasion examined children with a history of intermittent crossing of the eyes who had discovered the trick of voluntarily convergence.

Reflex convergence may be divided into a number of components[30]: tonic convergence, accommodative convergence, fusional convergence, and proximal convergence.

TONIC CONVERGENCE. The anatomical position of rest of the eyes (see Chapter 8) is generally believed to be one of divergence. To bring the eyes from this position into the physiologic position of rest, the only position that one can define operationally in awake and conscious subjects is tonic convergence. Tonic convergence presumably is brought about by the tonus of the extraocular muscles. Extraocular muscles are never without electrical activity when the eyes are at rest in the intact, awake human. The sources of tonus of the extraocular muscles are discussed also on page 11.

In the absence of quantitative data on the absolute position of rest, it is impossible to make quantitative statements about tonic convergence. However, it would appear that tonic convergence in the visually adult person remains rather stable throughout life, as judged by the stability of the "fusion-free" position.[40]

The apparent increase of tonic convergence after sustained periods of fixation appears to be the result of prolonged decay of fusional vergence rather than a change in the level of tonic innervation of the extraocular muscles.[44]

Jampolsky[28] questioned the existence of tonic convergence or tonic divergence—that is, the existence of any convergence or divergence mechanism that is not set into motion by retinal stimuli (see also Chapter 22). For example, he pointed out that in death and before rigor mortis the eyes commonly are only slightly divergent or even straight. This is not directly comparable to the situation in which a live person fixates at a distance. Others have made positive observations in favor of the existence of tonic convergence. Cohen and Alpern[9] among others, in studying the effect of ethanol on the accommodative convergence–accommodation (AC/A) ratio, have shown that ethanol increases convergence at distance; that is, it produces an increase in tonic convergence. Jampel[27] produced convergence movements without pupillary responses by stimulating certain areas of the central nervous system. Jampolsky[28] was not convinced that tonic convergence can be produced by these experiments because of the possibility that the electric current may have stimulated a broader area than that intended. Be that as it may, the evidence appears to favor the existence of tonic convergence. Moreover, we believe that a *nonaccommodative convergence excess type of esotropia* (see Chapter 16), that is, an esotropia greater at near than at distance fixation, in a patient with a normal or abnormally low AC/A ratio can only be explained on the basis of excessive tonic convergence.[39] The relentless recurrence of certain types of comitant esotropia despite repeated maximal surgery has also been blamed on excessive tonic convergence.[38] From a clinical point of view the mechanism of tonic convergence is a useful and necessary concept even though nothing is known about the source and trigger mechanism of such innervation.

ACCOMMODATIVE CONVERGENCE AND THE AC/A RATIO. When a person exerts a certain amount of accommodation, a determined amount of convergence is elicited. Convergence so elicited is called accommodative convergence. The reverse would seem to hold true also; for example, forced convergence with ophthalmic prisms may cause changes in accommodation.[14] However, these changes in accommodation have no clinical importance.

The synkinesis between accommodation and convergence has important physiologic effects on binocular vision in near fixation, and an understanding of this role is essential in the study of comitant strabismus.

It is reasonable to assume that the basic convergence requirement is fulfilled through accommodative convergence. Tonic and fusional convergence have their own functions, and proximal convergence is a supplementary one. Therefore a normal, emmetropic person should be expected to exert 1 MA of convergence for each diopter of accommodation (or its equivalent in prism diopters), but this is not the case. Each individual responds to a unit stimulus of accommodation with a specific amount of convergence that may be greater or smaller than is called for by the convergence requirement. The convergence response of an individual to a unit stimulus of accommodation may be expressed by his or her AC/A ratio. This ratio, which has the dimensions [Δ/D], is a measure of the responsiveness of a person's convergence function to a unit of stimulus of accommodation. The concept of a ratio between accommodation and convergence was first clearly defined by Fry[18] who later with Haines[20] introduced the abbreviation, AC/A ratio.

METHODS FOR DETERMINATION OF THE AC/A RATIO

Heterophoria Method. This method consists of measuring the deviation in distance fixation (optical infinity) with full correction of a refractive error, if one is present, on the assumption that no accommodation is exerted under these circumstances. The deviation then is measured at near-vision distance (33 cm or 3D) on the assumption that the convergence exerted is caused wholly by the accommodation-convergence synkinesis. The AC/A ratio is obtained from the equation

$$\mathrm{AC/A} = \mathrm{PD} + \frac{\Delta_n - \Delta_o}{\mathrm{D}}$$

where PD is the interpupillary distance (in centimeters), Δ_n and Δ_o the deviations near and at distance, and D the fixation distance at near in diopters. This equation is explained by the fact that the convergence requirement equals the interpupillary distance multiplied by the fixation distance in diopters and that the change in deviation from distance to near fixation equals $\Delta_n - \Delta_o$. Note also should be made of the fact that the sign conventionally is as follows: esodeviations are considered to be positive (+) and exodeviations to be negative (−).

To give an example:

$$\mathrm{PD} = 6.0\ \mathrm{cm},\ \Delta_n = 8^{\Delta}\mathrm{X\ at\ 3D},\ \Delta_o = 2^{\Delta}\mathrm{X}.$$

$$\mathrm{AC/A} = 6.0 + \frac{-8-(-2)}{3} = 4^{\Delta}/\mathrm{D}$$

The AC/A ratio equals 4^{Δ} of accommodative convergence for each diopter of accommodation.

A simple comparison of the deviation in distance and near fixation is commonly used in clinical practice to estimate the AC/A ratio. If the two measurements are equal, the AC/A ratio is said to be normal.[41] If the near measurements in an esotropic patient are greater by 10^{Δ} or more, the AC/A ratio is said to be abnormally high.

Caution is necessary when making such a determination. The difference between the deviation in distance and near fixation is of great practical importance in assessing the degree of comitant strabismus in patients with esotropia or exotropia. The use of the term *normal* or *excessive AC/A ratio* should be avoided, however, unless the ratio has been actually determined by using equation (1) or by some other method. If a patient has an esodeviation of 30^{Δ} for distance and for near fixation (e.g., at 33 cm) and a PD of 5.7 cm, his or her AC/A ratio would be

$$5.7 + \frac{30-30}{3} = 5.7^{\Delta}/\mathrm{D}$$

The AC/A ratio is equal to the interpupillary distance. Theoretically, the expected "ideal" AC/A ratio is when the convergence requirement is fulfilled by accommodative convergence. If the term *normal* is understood in a statistical sense, however, one would say that the AC/A ratio of this patient is close to the upper limit of the distribution of a population with a normal sensorimotor system. In such a population the mean of the AC/A ratio is somewhat over half the interpupillary distance. If a patient has an esotropia of 30^{Δ} in distance fixation and 36^{Δ} when fixating at 33 cm (which is not considered to be a clinically significant difference) and again an interpupillary distance of 5.7 cm, the AC/A ratio would be

$$5.7 + \frac{36-30}{3} = 7.7^{\Delta}/\mathrm{D}$$

This figure is outside the normal range and by all counts a high AC/A ratio. *These considerations must be kept in mind by those who determine the AC/A ratio by comparing distance and near deviations*.

An additional caveat regarding the heterophoria method concerns those patients who have an abnormal near-distance relationship of their esotropia that is unrelated to a high AC/A ratio (nonaccommodative excess). Von Noorden and Avilla[36] showed that the AC/A ratio as determined with the gradient method is actually normal or may be subnormal in such patients and that reliance on the heterophoria method will miss the correct diagnosis of this entity.

Gradient Method. Another method of determining the AC/A ratio which is preferred by us over other methods is the so-called gradient method. Here the change in the stimulus to accommodation is produced by means of *ophthalmic lenses,* not by a change in viewing distance. For a given fixation distance, minus lenses placed before the eyes increase the requirement for accommodation and plus lenses relax accommodation. It is assumed that −1D lenses produce an equivalent of 1D of accommodation, whereas +1D lenses relax accommodation by 1D, and that the accommodative response to the lenses (and therefore the accommodative convergence produced) is linear within a certain range. For a given fixation distance the AC/A ratio inferred from the effect of ophthalmic lenses may be readily ascertained from the simple formula

$$AC/A = \frac{\Delta_1 - \Delta_0}{D}$$

where Δ_0 is the original deviation, Δ_1 the deviation with the lens, and D the power of the lens. If the original deviation for a given fixation distance was an exodeviation of 2^{Δ}, and if −2D lenses induced an esodeviation of 8^{Δ}, the AC/A ratio would be

$$\frac{8-(-2)}{2} = 5^{\Delta}/D$$

The AC/A ratio computed by the heterophoria method is usually larger than the one obtained by the gradient method, mainly because of the effect of proximal convergence. It is held, therefore, that only the gradient method gives a true estimate of the AC/A ratio,[40, p 121ff; 48] but it is necessary that more than two points be determined.

First, one cannot be sure of the limits within which the AC/A ratio is linear. Alpern and coworkers[3] showed linearity over the intermediate stimulus levels (+1 to 5D) in prepresbyopic subjects but nonlinearity at the lower and higher stimulus levels.

Second, the deviations of the ocular axes are subject to considerable random variation in magnitude. By determining a greater number of points and drawing the best-fitting line through them, one can lessen the effect of the random variations on the computed AC/A ratio. Table 5–1 shows the way in which Sloan and coworkers recorded their data. In Figure 5–5 the data from this table are plotted in a graph. The slope of the best-fitting line, s = 3.9, is a measure of the AC/A ratio.

TABLE 5–1. Illustrative Data on Accommodation-Convergence Relationship

Dioptric Power of Added Spheres	Diopters of Accommodation Required for Clear Image at 33 cm	Lateral Phoria at 33 cm in Prism Diopters	Equivalent Convergence in Prism Diopters (PD = 6.4 cm)	Supplementary Observations
+4.0	−1.0	18X	1.5	Target details blurred
+3.0	0	18X	1.5	Target details clear
+2.0	1.0	17X	2.5	
+1.0	2.0	12X	7.5	
0	3.0	9X	10.5	Passes bar-reading test
−1.0	4.0	5X	14.5	
−2.0	5.0	1X	18.5	
−3.0	6.0	2E	21.5	Passes bar-reading test
−4.0	7.0	7E	26.5	
−5.0	8.0	11E	30.5	
−6.0	9.0	14E	34.5	
−6.5	9.5	21E	41.5	Target details clear with effort
−7.5	10.5	29E	49.5	Target details blurred

*Normal subject, age 26, interpupillary distance 6.4 cm, exophoria of 0.5Δ at 20 ft.
X, exophoria; E, esophoria.
Modified from Sloan L, Sears ML, Jablonski MD: Convergence-accommodation relationships. Arch Ophthalmol 63:283, 1960.

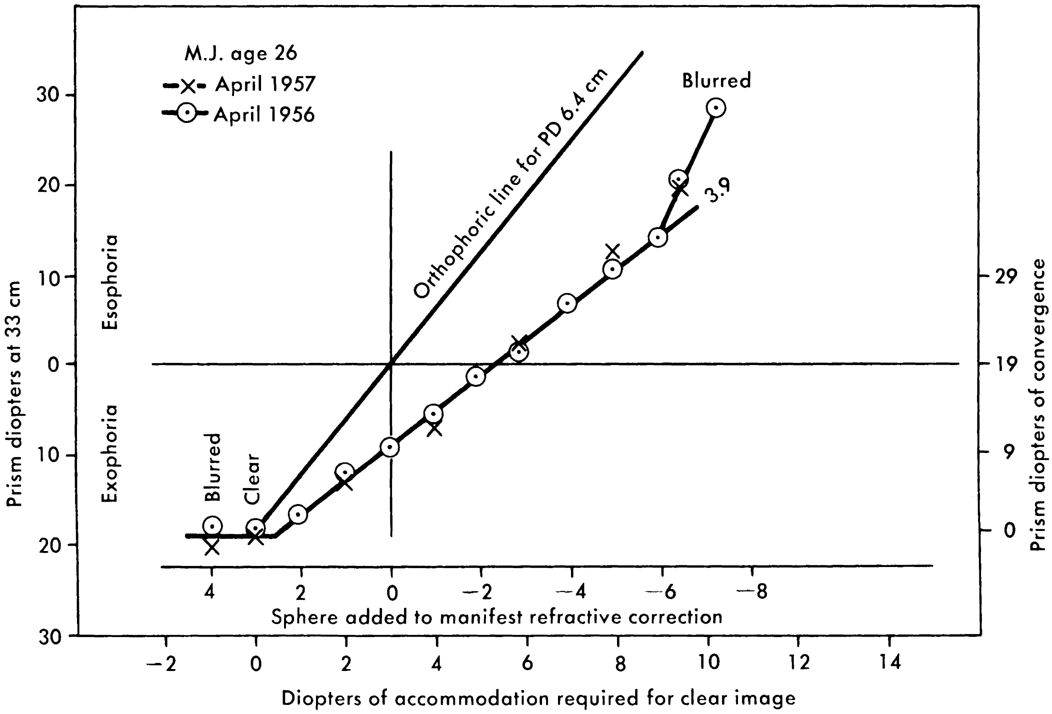

FIGURE 5–5. Graph showing the accommodative convergence–accommodation (ratio) data given in Table 5–1. PD, prism diopter. (From Sloan L, Sears ML, Jablonski MD: Convergence-accommodation relationships. Arch Ophthalmol 63:283, 1960.)

For clinical purposes it suffices to measure the deviation with the eyes in primary position at a fixation distance of 33 cm and the patient fully corrected and then to repeat these measurements after the addition of +3.00D and −3.00D lenses.

Fixation Disparity Method. The fixation disparity method has been used extensively by Ogle and coworkers[40] to obtain the AC/A ratio. The AC/A ratio is indirectly derived from this technique. Ogle and coworkers determined in one set of data the changes in fixation disparity induced by forced convergence using prisms and in a second set of data the changes induced by altering the accommodative stimulus with lenses. From these two sets of data they determined the stimuli for convergence and accommodation that gave the same fixation disparity. These results agreed with those obtained by means of direct determination of the AC/A ratio. The authors pointed out that the value of their method is the fact that it is binocular test.[40] However, because of its complexity, the test has found little application in clinical work.

Haploscopic Method. Another laboratory method which must be mentioned makes use of haploscopic arrangements (p. 72). In fact, the haploscope was originally invented for study of the accommodation-convergence relationship and is ideally suited for the purpose.

Normal Range of the AC/A Ratio. Quantitative studies on persons with normal sensorimotor systems have shown that in the vast majority of people the AC/A ratio does not fulfill the convergence requirement. The normal range of the AC/A ratio is between 3 and 5. Values above 5 are considered to denote excessive accommodative convergence and values under 3, an insufficiency.[46] Figure 5–6*A* shows the frequency distribution in 256 subjects studied by Ogle and coworkers[40] with the fixation disparity method. Franceschetti and Burian,[16] using a gradient method (Fig. 5–6*B*), found a somewhat different distribution in 355 subjects of a random population.

THE ACCOMMODATION-CONVERGENCE RELATIONSHIP: ACQUIRED OR INNATE? From the clinical standpoint the most important questions are whether the AC/A ratio is a stable function, constant throughout life, and whether it can be manipulated by glasses, drugs, surgery, or orthoptic treatment. The answers depend to some extent on whether one believes the AC/A ratio to

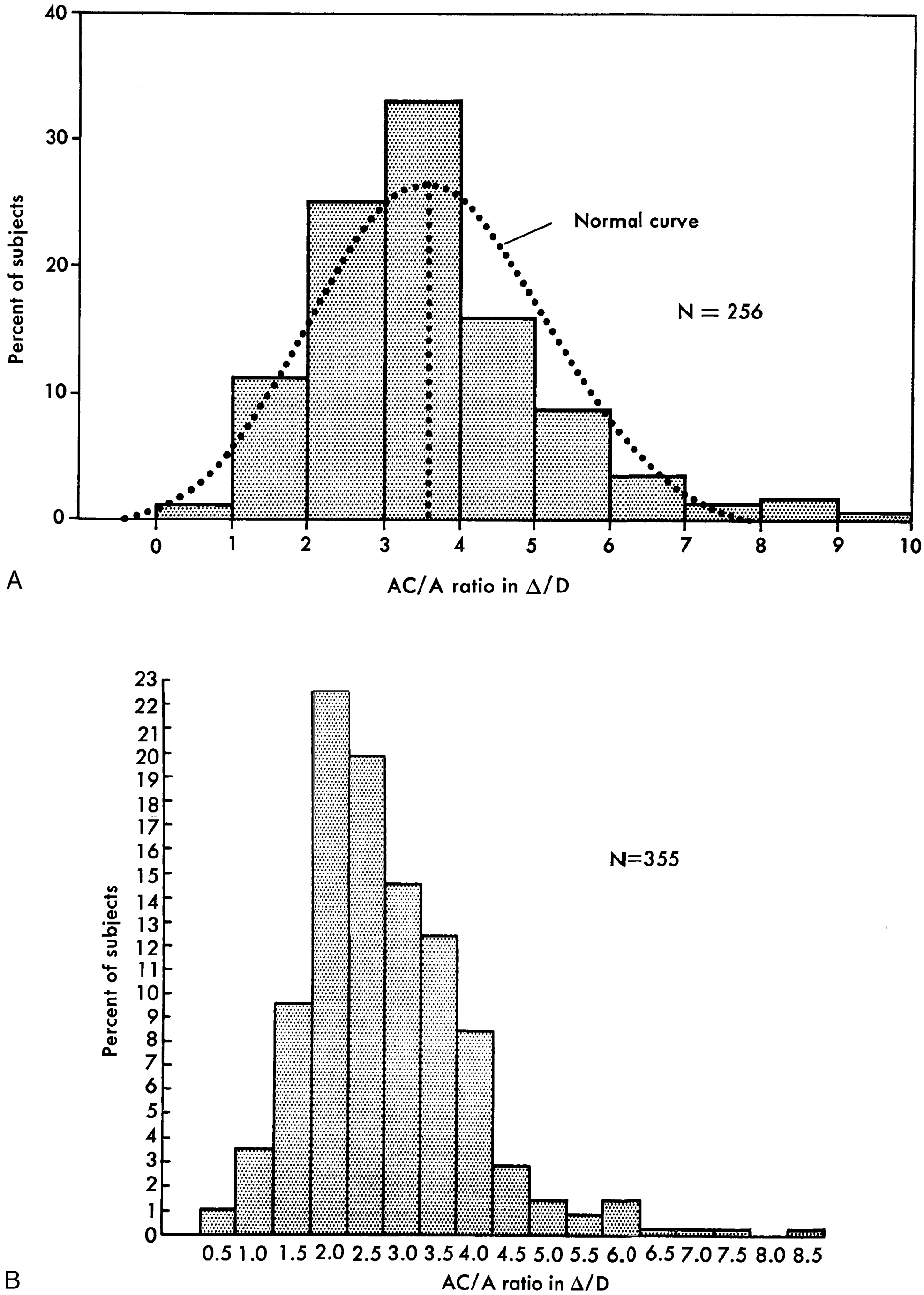

FIGURE 5–6. Distribution of accommodative convergence–accommodation (AC/A) ratios in a normal population (Δ/D). (*A* from Ogle KN, Martens TG, Dyer JA: Oculomotor Imbalance in Binocular Vision and Fixation Disparity. Philadelphia, Lea & Febiger, 1967; *B* from Franceschetti AT, Burian HM: Gradient accommodative convergence–accommodative ratio in families with and without esotropia. Am J Ophthalmol 70:558, 1970.)

be an acquired (learned) or an intrinsic association.

The view of Helmholtz,[24] that the association between accommodation and convergence develops early in life as a result of constantly repeated, simultaneous use of related degrees of the two functions—in other words, that it is a *learned association*—has been accepted and elaborated on by many workers. An acquired association implies a certain degree of independence in the relationship of the two functions; that is, one function can change to some degree without a change in the other. This elastic relationship is expressed in the classic teaching of Donders[11] and many others of a "relative" accommodation and "relative" convergence (p. 97). In the earlier orthoptic literature the treatment for accommodative esotropia therefore was spoken of as aimed at the "dissociation" of the two functions. Judge and Miles[29] showed that by increasing the interpupillary distance with periscope glasses the coupling between convergence and accommodation can be challenged and undergo adaptive modification. The clinical significance of this finding must be explored. Hainline and coworkers[22] showed that infants under 1 year of age had appropriate convergence for targets at various distances. However, accommodation lagged behind convergence in development.

Any change in the stimulus to accommodation that can be shown to lead to a change in convergence or that accommodation can be changed by forced convergence would favor an innate and stable relationship between the two types of convergence. If it could be shown that any change in the stimulus to accommodate causes a change in convergence and, conversely, that accommodation is altered by prismatically induced convergence, this would favor an innate and stable relationship between these two functions. Such a situation was found by Ames and Gliddon[4] and by Morgan.[33] Furthermore, if the association is learned, one would not expect it to exist in patients who have had strabismus throughout most or all of their lives. Hofstetter[25] found, however, that strabismic patients have the same pattern of the accommodation-convergence relationship as that in random samples of nonstrabismic populations. Hofstetter[26] also, by analysis of variance of this relationship in 30 pairs of identical twins, noted that there was a greater difference between families than between members of the same family. This would point to some genetic factors. Franceschetti and Burian,[16] in comparing the AC/A ratio of a random population with that of members of families with an esotropic propositus, also found a significant difference in their average AC/A ratio and percentile distribution, which indicates that the AC/A ratio is a factor in the inheritance of esotropia.

The effect of age is of interest as well in this connection. From the evidence in the literature the AC/A ratio appears stable up to the presbyopic age and even beyond, although Alpern[2, p. 130] found a slight decline with age. Fry[19] described for his own eyes an increase in the AC/A ratio during the 20 years from his 30th to 50th year. An increase in early presbyopia would not be too surprising and might well be attributed to an increase in impulse to accommodation, somewhat similar to that required with cycloplegia. This view is in agreement with that of Breinin and Chin,[7] who demonstrated in a longitudinal study that the stimulus AC/A ratio remained essentially unchanged from age 16 through 52 but increased significantly beginning with the prepresbyopic age through early presbyopia (see also Fincham[13]).

A stable, genetically determined relationship between accommodation and convergence, based on fixed central nervous system arrangements, presupposes that excitations in these regions of the central nervous system by a stimulus to accommodation elicits simultaneous impulses to the extraocular muscles. Martens and Ogle,[31] among others, found that within the range of response to ophthalmic lenses—that is, within the limits in which neither diplopia nor excessive blurring was induced by these lenses—the responses were indeed linear in 90% of 250 subjects examined. When a nonlinearity with plus lenses was found, it was attributed to a failure to relax accommodation rather than to a nonlinearity of the AC/A ratio itself.

STIMULUS AND RESPONSE AC/A RATIO. To obtain an understanding of the relationship between accommodation and convergence, one must keep in mind the elements involved in the process. These elements are (1) the change in stimulus to accommodation, (2) the peripheral and central nervous system mechanisms that elicit and transmit the impulses and provide the motor impulses to the inner and outer muscles of the eyes, and (3) the effector organs that provide the responses (the change in refraction of the eye and the change in position of the globe). These factors must be briefly analyzed.

So far in this discussion of AC/A ratio determination, the degree of convergence achieved has been related to the stimulus to accommodation (the dioptric power of the lenses used or the change in viewing distance). This relationship has been termed the *stimulus AC/A ratio* by Alpern and coworkers.[3] In laboratory studies one can arrange a haploscopic device so that the stimulus to accommodation, the response to the stimulus (the change in refraction of the eyes), and the change in position of the eyes can be determined simultaneously. With such an arrangement, one can relate the change in convergence to the stimulus to accommodation as well as to the accommodative response. The AC/A ratio related to the accommodative response has been termed the *response AC/A ratio*. This ratio differs from but parallels the stimulus AC/A ratio reported by Alpern and coworkers[3] and by Ripps and coworkers.[43] Alpern and coworkers[3] stated that the response AC/A ratio could be predicted with reasonable accuracy by multiplying the stimulus AC/A ratio by a factor of 1.08. In other words, the response AC/A ratio exceeds the stimulus AC/A ratio by about 8%. Presumably, this applies only to nonpresbyopic adults.

From the clinical standpoint, to determine the response AC/A ratio is impractical and unnecessary. The clinician must be concerned with the stimulus AC/A ratio. Various investigators have shown that the convergence response is generally linear and the stimulus is in the range within which the observers can respond. However, it is evident that a given stimulus to accommodation need not always elicit the required amount of change in refraction of the eye (the accommodative response), for example, in presbyopic patients. Although the impulses to accommodation sent out by the central nervous system may be adequate, or even excessive, and may result in adequate contraction of the ciliary muscle, the accommodative response will not be linear with the stimulus because of hardening of the crystalline lens. In cycloplegia the accommodative response may be too low or altogether absent because of a lack of response of the ciliary muscle. Nevertheless, in presbyopia there is a convergence response linear to the stimulus[40, p. 151] and in partial cycloplegia even an excessive convergence response.

The reason one should use the stimulus AC/A ratio for clinical purposes is, then, that the accommodative convergence response does not depend on the accommodation response as such but rather on what is known in clinical terminology as the greater or lesser ''effort of accommodation,'' which means the greater or lesser impulse to accommodation elicited by the stimulus. This is not an appropriate term. It cannot be defined or quantified operationally. The physiologist does not like it. Nevertheless, it has a certain usefulness to the clinician. When anomalies are present that require a greater stimulation to accommodation, the associated convergence will be greater accordingly. Examples include uncorrected hypermetropia and inadequate ciliary muscle response such as cycloplegia. Conversely, if a lesser stimulus is necessary to achieve sharp retinal imagery, as in uncorrected myopia or with a spasm of the ciliary muscle, less innervation will be sent out to the ciliary muscle, and, accordingly, to the extraocular muscles.

CHANGES IN THE AC/A RATIO WITH GLASSES, DRUGS, SURGERY, AND ORTHOPTICS. Both accommodation and convergence have a central and a peripheral mechanism. The definition given for the AC/A ratio as a measure of responsiveness of the convergence mechanism to a unit of accommodation refers to the central mechanism. Theoretically, this is undoubtedly permissible, since the wide range of AC/A ratios clearly cannot be attributed to differences in the peripheral mechanisms of either accommodation or convergence. Methods of measuring the ''accommodative effort'' or the convergence responsiveness are not available.[6] One can only determine the change in vergence induced by a unit stimulus to accommodation (or by the refractive change produced by such a stimulus, as in the response AC/A ratio). This, then, is the *operational definition of the AC/A ratio.*

Such an operational definition is most needed since it is possible to bring about changes in vergence through various manipulations of the peripheral mechanisms of accommodation and convergence. However, when evaluating neuromuscular anomalies of the eyes, one should keep in mind that a central mechanism—the so-called accommodative effort—in the last analysis controls the AC/A ratio.

This is quite clear in the case of *spectacles lenses*. No one has claimed that spectacles lenses change the AC/A ratio. If, for instance, a patient has an esodeviation of 15^{Δ} for distance and a refractive error of $+3D$ in both eyes (OU) and if the deviation is caused solely by accommodative convergence, correction of the refractive error will reduce the deviation at distance to zero. What happens is that if the patient's eyes are not corrected by glasses, hypermetropia will be overcome by accommodation. There is an AC/A ratio of $5^{\Delta}/D$ and therefore the patient develops an esodeviation of 15^{Δ} in distance fixation. With correction, the need for accommodation at distance is zero and consequently there is no deviation. The AC/A ratio has not changed; only the need to accommodate has been removed. On the other hand, von Noorden and Avilla[36] reported a gradual decrease of esotropia at near fixation without change of the angle at distance in a group of children wearing bifocals. This observation must be interpreted to the effect that the AC/A ratio has changed.

The situation is seemingly more complex when it comes to the effect of topically applied *miotics*. For example, if one uses a parasympathicomimetic drug, such as echothiophate iodide (Phospholine Iodide), clinical experience shows that the deviation has decreased insofar as it is caused by accommodative convergence. Ripps and coworkers[43] demonstrated that since this drug is a cholinesterase inhibitor, it enhances the effect of acetylcholine on the ciliary muscle. There is a facilitation of impulse transmission at the neuromuscular junction, which means that this drug lessens the impulse required to obtain a unit contraction of the ciliary muscle; therefore the AC/A ratio should be reduced as defined operationally, and in fact this is the case. The AC/A ratio obtained by the gradient method is considerably smaller when the eyes are under the influence of diisopropyl fluorophosphate (DFP)[48] or echothiophate iodide[43] than when the eyes are in their natural state. This effect can be verified in every patient in whom these drugs are effective, as in the following example.

CASE 5–1. A female patient, born 9–19–62, had a left esotropia from birth.

> Treatment with glasses was started at 9 months of age. 3–4–65: Recession left medial rectus 4 mm, myectomy left inferior oblique. 9–14–66: Refraction and visual acuity +6.00 sph. + 1.50 ax 30° = 6/6; OS + 6.75 sph. +1.00 ax 175° = 6/8. 10–11–67: s̄ RX more than 70^{Δ} ET for distance and near, c̄ Rx 22^{Δ} ET with 5^{Δ} left hypertropia (LHT); 16^{Δ} ET′ with 8^{Δ}-LHT′. AC/A ratio = 10^{Δ}/D (gradient method). Placed on 0.125% echothiophate iodide in 5% phenylephrine (Neo-Synephrine), one drop to each eye every night. 11–9–67: 14^{Δ} ET with 3^{Δ} LHT; 8^{Δ} ET′ with 6^{Δ} LHT′. AC/A ratio = 4^{Δ}/D (gradient method).

Since the mechanism is a peripheral one, residing in the effectors, whether one wishes to say that the AC/A ratio has changed or to think in terms of a change in response of the effector, which calls for a reduced "accommodative effort," is a matter of definition. There is a certain parallel to the effect of glasses. Glasses change the input—the stimulus to accommodation. Drugs change the state of the effectors.

Tour[52] expressed the thought that parasympathicomimetic drugs affect the pupil. The greater depth of focus of an eye with a narrow pupil would reduce the need to accommodate and, hence, reduce the "accommodative effort." If this were true, these drugs would act, as do spectacles lenses, by reducing the input. That this is not the case was shown by Ripps and coworkers.[43]

The effect on the AC/A ratio of weakening the action of the medial rectus muscles surgically is explained by a change in the relationship between muscular contraction and the resulting rotation of the eyes.[41, 45] Although parasympathicomimetic drugs increase the sensitivity of the ciliary muscle to stimulation, operations on the medial rectus muscles reduce their mechanical effectiveness. In both cases the change is in the effector system, with this difference: when the drug is discontinued, the AC/A ratio generally returns to its original value. With *surgery* the change is lasting.[46] There is no reason to assume that the central linkage of accommodation and convergence has been affected by topically applied miotics or by surgery.

The whole concept presented in this chapter is supported by the effect of cycloplegic agents on the AC/A ratio. The clinician knows that patients with accommodative esotropia (see Chapter 16) may have a larger deviation in incomplete cycloplegia than without cycloplegia, as reported by Maddox.[30] Christoferson and Ogle,[8] in studying the effect of cycloplegia on the AC/A ratio, related the magnitude of this ratio to the NPA. Figure 5–7 shows the data from a patient in whom the AC/A ratio increased from 2.4^{Δ}/D before cycloplegia to 20^{Δ}/D 1 hour after instillation of 2% homatropine hydrobromide drops. Twenty-four hours after the instillation, the AC/A ratio had returned to its initial value.

Interpretation of these findings is as follows. In cycloplegia a change in stimulus to accommodation results in a stronger impulse to the ciliary muscle than without cycloplegia and consequently to the extraocular muscles. A more or less unsuccessful effort is made to clear the retinal image. Remarkably, the relationship between stimulus to accommodation and convergence response remains linear even under those circumstances.

Cycloplegic agents, then, though acting directly on the peripheral mechanisms of accommodation, have an indirect effect on the central nervous

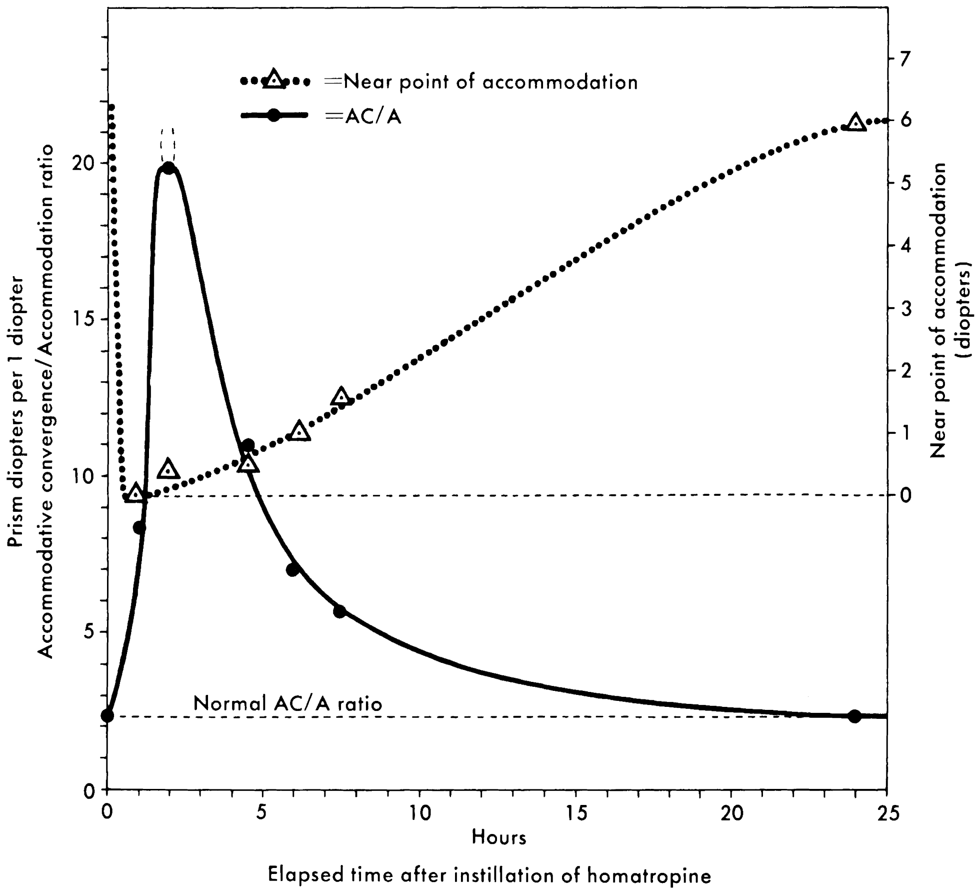

FIGURE 5–7. Effect of instillation of homatropine on the accommodative convergence–accommodation (AC/A) ratio, showing the marked increase in the AC/A ratio and corresponding reduction in the near point of accommodation and their return to normal within 20 to 25 hours. (From Christoferson KW, Ogle KN: The effect of homatropine on the accommodation-convergence association. Arch Ophthalmol 55:779, 1956.)

system control of the accommodation-convergence synkinesis, eliciting a greater "accommodative" effort.

A direct effect of drugs on the central nervous system, which influences the AC/A ratio, though in the opposite sense, is known to exist. Powell,[42] Colson,[10] Adler[1] and others have shown an increase in blood alcohol levels to be associated with an increase in esophoria at distance, an increase in exophoria at 33 cm (a more remote NPC), and some effect on fusional movements. Ethanol appeared not only to increase tonic convergence but also to reduce the AC/A ratio.[9]

Last, a word must be said about the effect of orthoptic exercises on the AC/A ratio. Most authors agree that such exercises do not change the AC/A ratio but Flom[15] reported that in patients with exophoria, orthoptic exercises induced a nominal but only temporary increase in the AC/A ratio.

"RELATIVE" CONVERGENCE AND "RELATIVE" ACCOMMODATION. The observation that, within limits, one can force convergence by the use of prisms without blurring the fixated object and, conversely, that one can change accommodation by means of lenses without causing diplopia suggested to Donders[11] and his followers that there is an elastic relationship between accommodation and convergence. The limits within which convergence and accommodation could be changed without producing blurring or diplopia were termed the "amplitude of relative convergence" and the "amplitude of relative accommodation." This teaching has prevailed until recently, but it has now been shown that every change in accommodative stimulus produces a change in convergence. The limits within which single vision is possible with changes in accommodative stimulus depend not on an elastic relationship between accommodation and convergence but on the availability of fusional amplitudes that enable one to cope with the change in the position of the eyes. This concept is of basic importance for the understanding of binocular cooperation and of the neuromuscular anomalies of the eyes.

FUSIONAL CONVERGENCE. Accommodative convergence provides for gross adjustment of the position of the eyes, but when acting alone it rarely if ever provides binocular fixation. As stated on page 92, the AC/A ratio is too low in the majority of people, relative to the convergence requirement, leaving a divergence of the visual axes at near fixation. In some people the AC/A ratio is too large, causing excessive convergence of the visual axes and esodeviation in near fixation. The fine adjustment of the visual axes necessary for binocular fixation is obtained by *fusional vergence movements.* Fusional convergence does not differ in its general characteristics from other fusional movements. It is involuntary, and the stimulus for it is disparate retinal imagery.

PROXIMAL CONVERGENCE. A common experience in clinical testing of a patient's deviation is that esodeviations measured on a major amblyoscope are generally larger than those detected by the prism and cover test.[5, 17, 50] This difference is present despite the fact that the major amblyoscope is arranged so that the targets are set at optical infinity. Furthermore, when the esodeviation of a patient who wears full correction is determined at near, one should expect to find the deviation to be equal to that in distance vision when lenses equivalent to the fixation distance at near are placed in front of the eyes. Under these conditions the need for accommodation has been obviated. One may find, however, that even then the deviation is larger at near than at distance. There are two explanations for this situation. The glasses worn by the patient may not have fully corrected the refractive error; or if it was definitely established that correct glasses were worn, the difference may be attributable to proximal convergence.

Proximal convergence is induced by the awareness of nearness of an object. This may be either an object at near vision distance or one that, while situated at near vision distance, has been optically placed at infinity, as in a major amblyoscope.

The actual amount contributed by proximal convergence to fulfill the convergence requirement has been variously estimated. Morgan[33] examined 413 subjects and estimated the mean to be 3.5^{Δ}. An inverse relationship between proximal convergence and observation distance has been noted.[21, 47] Wick and Bedell[54] measured the magnitude and velocity of proximal convergence and found that the peak velocities averaged to be substantially faster than the velocities of comparably sized fusional or accommodative convergence responses. These authors suggest that proximal convergence may, in fact, play a greater part in contributing to the near vergence response than traditionally assumed.

Ogle and coworkers[40, p. 131] determined the AC/A ratio with the heterophoria method (R_d). They established a ratio PC/D, proximal convergence over distance, by the formula $PC/D = R_d - R_L$, where R_L stands for the AC/A ratio determined by the gradient method. They then found the proximal convergence of 28 subjects to have a mean value of 2.25^{Δ}, with a spread of -3.12^{Δ} to 7.25^{Δ}. It is difficult to interpret the meaning of the negative values.

SUMMARY. In the foregoing pages, following the lead of Maddox,[30] we subdivided convergence into a number of subclasses; however, we must emphasize that this is an artificial separation. In reality, there are certain central nervous system arrangements, the details of which are little known, that control impulses to the nuclei of the third cranial nerve and to the medial rectus muscles so that simultaneous adduction of the globes occurs, which we call convergence. These centers are probably located in the midbrain but have numerous connections with various cortical, subcortical, and peripheral retinal areas. As a result, convergence movements (or changes in convergent positions) can be elicited in many different ways: through stimuli arising in the cortex (tonic and proximal convergence); through the ‘‘accommodative effort’’ elicited by retinal stimuli by means of cortical areas 17 and 19 (accommodative convergence); and through convergence elicited by disparate retinal stimuli, again through the primary visual cortical areas and beyond (fusional convergence). One must not assume that the central arrangements for convergence distinguish between the various sources of impulses received. The central arrangements for convergence respond or do not respond to the stimuli reaching them and transmit them to the nuclei of cranial nerve III.

Although for analytic reasons, both physiologic and clinical, it is necessary to separate the various sources of convergence movements; do not forget that convergence is a unitary process.

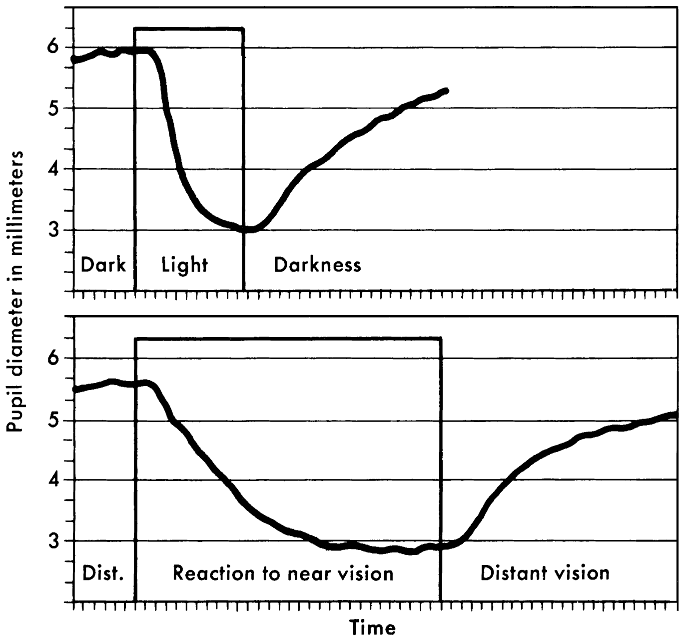

FIGURE 5–8. Graph showing the difference in temporal characteristics of light and near reaction of the pupil.

Pupillary Constriction

When changing fixation from a distant to a near object, in addition to accommodation and convergence, the pupils constrict. This reaction of the pupils differs from that which occurs with a change in retinal illuminance. It is slower (tonic) in nature and is maintained as long as the near fixation distance is maintained. When fixation is shifted to a more distant object, the pupils slowly dilate after a relatively long latency time of about 0.5 second. The time it takes to regain the original pupil size is roughly 10 times longer than the values of other pupil reactions.[55] In contrast to this slow, maintained constriction, a change in retinal illuminance causes speedier constriction, which is not maintained. If the retinal illuminance remains constant, the pupils return to their physiologic width corresponding to the level of illuminance (Fig. 5–8).

Pupillary constriction, although occurring in association with convergence and accommodation, does not depend on either one.[51] Discussion continues whether, for instance, it is possible to have miosis with convergence of the visual axes while eliminating accommodation with plus lenses in front of each eye. Likewise, miosis occurs at near fixation in patients with uncorrected myopia and advanced presbyopia.[51]

On the other hand, recent work has shown that under rigorous alignment conditions accommodation may occur without pupillary constriction.[49] Myers and Stark[34] showed that the addition of a near stimulus reduces the latency of vergence eye movements and of accommodation more than pupillary latency. They concluded from these findings that the dual interaction between vergence and accommodation on the one hand and miosis on the other may be asymmetrical rather than symmetrical as previously assumed.

Constriction of the pupil at near fixation will be equal in both eyes even though vision in one eye may be impaired.

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