British Journal of Ophthalmology, 1982, 66, 398–404

Stimulus and response AC/A ratios in intermittent exotropia of the divergence-excess type

JEFFREY COOPER, KENNETH J. CIUFFREDA, AND PHILIP B. KRUGER

From the Amblyopia Laboratory, State College of Optometry, State University of New York, 100 East 24th Street, New York, NY 10010, USA

SUMMARY Objective infrared recording devices were used to measure simultaneously and continuously both accommodation and accommodative vergence to near stimuli in 4 subjects with intermittent exotropia of the divergence-excess type (2 simulated and 2 true). In addition standard clinically determined stimulus accommodative convergence to accommodation (AC/A) ratios were measured. Results showed the mean group response AC/A ratio to be 5·9/1 (range 4·5–8·0/1) with no differences between true and simulated divergence-excess. Similar AC/A ratios were found after 45 minutes of monocular occlusion. Our results clearly demonstrate relatively normal response AC/A ratios in these subjects. Thus, contrary to what is believed by many clinicians, the reduced ocular deviation at near compared with distance vision cannot be attributed primarily to an abnormally high AC/A ratio. We believe that fusional convergence after-effects and/or proximal convergence effects contribute to inflate the clinically determined stimulus AC/A ratios.

The divergence excess type of intermittent exotrope, with the deviation being greater at distance than at near vision,[1] is commonly believed to have a very high accommodative convergence to accommodation (AC/A) ratio.[2–5] This notion has been based largely on distance/near measurements of the horizontal oculomotor exodeviation (i.e., tropia or phoria). The average deviation is 29 Δ at far and 9 Δ at near,[6] resulting in an average stimulus AC/A ratio of approximately 14·0/1. However, reported values of the AC/A ratios in these patients show much variation. Ogle *et al.*[7] found the mean stimulus AC/A ratio to be 3·0/1 using the fixation disparity method.[8] Moore *et al.*,[9] grouping all exotropes (divergence excess, convergence insufficiency, and basic exotropes) together (45% were the divergence excess type), found the mean stimulus gradient AC/A ratio to be 4·7/1. Calculations from Brown[5] suggest presence of a stimulus AC/A ratio of 13·0/1 or greater based on the +3·00 dioptre gradient technique. Von Noorden[10] found a wide range of stimulus AC/A ratios (3·3 to 9·0) using the ±3·00 dioptre gradient technique in a large sample of patients. In 2 cases presented by Burian and Smith[11] gradient stimulus AC/A ratios were 6·0/1 and 8·0/1, while they were 14·0/1 by the distance/near phoria method.

There are 2 basic problems with the above mentioned studies. First, only stimulus AC/A ratios were determined. One is never certain of the actual accommodative response but rather assumes it to be equal to the stimulus demand. This assumption results in an 8% underestimation of the AC/A ratio in normal persons,[12] but the correction factor is unknown in patients with binocular vision abnormalities. Secondly, the distance/near type measures of the oculomotor deviation are probably contaminated, that is, inflated by proximal convergence effects,[13 14] which may be more dramatic in intermittent exotropes than in normal persons.[7] Thus, we sought to determine and compare stimulus and response AC/A ratios under several test conditions in 4 patients with the divergence excess type of intermittent exotropia. The AC/A ratio in these patients has important clinical implications, especially with respect to determination of appropriate surgical procedures to attain binocular alignment,[15] as well as in understanding the contribution of the components of vergence in Maddox's sense.[16]

Correspondence to Dr Kenneth J. Ciuffreda.

Patients and methods

All subjects had intermittent exotropia at distance (6 m) and an exotropia/phoria at near at least 10 prism dioptres less than at distance when fixating a 20/25 Snellen letter, and thus were classified as divergence excess.[17] Visual acuity was at least 20/20 in each eye. Stereopsis was at least 40 seconds of arc as determined on the Randot test.[18] Only adult subjects (aged 20-26 years, 3 females and 1 male) were tested, as rigorous test conditions essential to obtain objective measurements necessitated the use of a bite bar, head rest, and chin rest to stabilise the head for extended periods of time. Owing to the rigour of our test conditions objective recordings from 2 other subjects were discarded because of baseline shifts and gain changes in the records resulting from the inability to remain very still in the apparatus. Corrective lenses were worn during all testing.

Stimulus AC/A ratios were determined by 4 clinical methods.[19] (1) distance/near cover test: the alternate cover test was performed while the subject fixated and maintained in clear focus a 20/25 Snellen letter at 6 m and a J1 print at 40 cm. Prisms were used to neutralise the angle of deviation. Both subjective and objective angles of deviation were measured. (2) Gradient test at near: the subject fixated and maintained in clear focus a J1 print at 40 cm, while the horizontal deviation was neutralised both objectively and subjectively with prisms. Lenses of the following powers, +2·00, +1·00, plano, −1·00, and −2·00 dioptres were placed before both eyes and were used to calculate the AC/A ratio. (3) gradient test in a synoptophore: the subject viewed a superpositioned target which subtended an angle of 1 degree. Lenses of the following powers, plano, −1·00, −2·00, and −3·00 dioptres were placed before both eyes and were used to calculate the AC/A ratio based on subjective and objective neutralisation of the deviation. (4) Phorometric method: phorias, if possible, were measured in a phoropter with vertical dissociation to induce diplopia at distances of 6 m and 40 cm. The subject fixated and maintained in clear focus a 20/25 Snellen letter at 6 m and a J1 print at 40 cm. The AC/A ratio was calculated by comparing distance and near alignment values as related to the change in fixation distance.

On completion of the initial clinical portion the objective response and stimulus AC/A ratios were measured. An infrared optometer (Fig. 1) was used to monitor dynamic changes of accommodation in the fixating left eye. The optometer had a band width of 5 Hz, a resolution of 0·12 dioptres, and linearity of −6 to +6 dioptres. This dynamic optometer has been fully described elsewhere.[20] The accommodative stimulus was a fine-lined cross etched on the front surface of a clear piece of Plexiglass. The crosses subtended an angle of 1·5 or 3·0 degrees at distance and near respectively. The fine lines forming the crosses subtended an angle of approximately 2-4 min arc. The experimenter used a silent 2-position switch to illuminate either the far (67 cm, 1·5 D) or near (33 cm, 3·0 D) stimulus with temporal randomisation of the step inputs. All testing was done in a dark room, only the cross target being allowed to act as the primary stimulus to accommodation. An infrared scleral-reflection eye movement system[21] was used to monitor dynamic changes of accommodative vergence in the occluded right eye. The eye movement system had a band width of 40 Hz, a resolution of 0·5 degrees, and linearity of ±10 degrees (with DC bias to compensate for the outward deviation of the occluded, verging eye). The eye monitoring system was placed 10 mm from the eye. Calibration and cross-talk checks were performed before and after each test session. The accommodation response, accommodative vergence response, and stimulus were recorded simultaneously on a strip chart recorder (band width 150 Hz), from which the response AC/A ratios were calculated. Following the initial transient component in response to a stimulus change, the steady-state response was estimated by eye for the subsequent 2–5-second part of the record.

All stimulus and response AC/A ratios were repeated after 45 minutes of monocular occlusion to

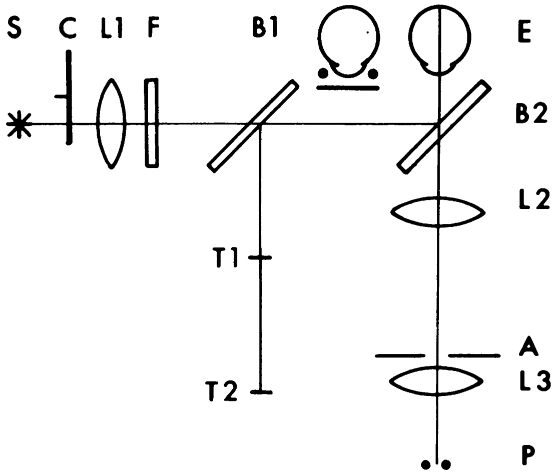

Fig. 1 *Top view of apparatus used to measure accommodation and accommodative vergence consisting of 4 components: (1) Light source (S=halogen lamp, C=light chopper, L1=collimating lens, F=infrared filter, B1 and B2= beam splitters, and E=eyes. (2) Targets (T2=far target, T1=near target, plus B1 and B2. (3) Accommodation monitor (P=photodetectors, A=aperture, L2 and L3=focusing lenses, plus B2. (4) Eye movement monitor (occluder, infrared emitter/detectors in front of right eye).*

Table 1 *Clinical and experimental data of subjects*

	Subject	Cover test[c] D	Cover test[c] N	Cover test AC/A[d]	Phor. D/N AC/A	Near gradient AC/A	Synoptophore AC/A	Objective AC/A $\bar{x}$[e]	SD	n	$\bar{x}$[f]	SD
(a)	Preocclusion											
	1[a]	12 X(T)	0	10·8/1	18·0/1	2·8/1	8·1/1	6·2/1	1·6	32	4·4/1	0·8
	2[a]	10 X(T)	0	10·0/1	8·5/1	2·5/1	7·5/1	4·9/1	1·1	23	3·6/1	0·8
	3[b]	17 X(T)	7 X	10·0/1	9·9/1	7·0/1	8·3/1	8·0/1	2·3	24	4·9/1	1·4
	4[b]	16 X(T)	4 X	10·8/1	12·7/1	7·5/1	8·6/1	4·5/1	1·1	47	3·9/1	0·8
$\bar{x}$=		13·8	2·8	10·4/1	12·3/1	5·0/1	8·1/1	5·9/1			4·2/1	
(b)	Postocclusion											
	1	20 X(T)	4 X	12·5/1	Supp.	2·8/1	4·7/1	6·4/1	1·6	18	2·9/1	0·7
	2	16 X(T)	6 X	10·0/1	8·0/1	2·5/1	5·0/1	4·5/1	0·6	10	3·7/1	0·7
	3	18 X(T)	18 X(T)	6·0/1	3·3/1	6·4/1	9·0/1	10·3/1	2·3	7	7·3/1	1·6
	4	16 X(T)	24 X(T)	2·8/1	7·3/1	6·1/1	7·0/1	4·7/1	1·6	17	4·4/1	1·4
$\bar{x}$=		17·5	13·0	7·8/1	6·2/1	4·5/1	6·4/1	6·5/1			4·6/1	

[a]True divergence excess. [b]Simulated divergence excess. [c]Unit is prism dioptres. [d]Unit for all AC/A measures is prism dioptres per dioptre. [e]Objective response AC/A ratio. [f]Objective stimulus AC/A ratio.
D = distance. N = near. Phor. = phorometric.

determine the extent of postocclusion changes, especially with respect to classification of true or simulated divergence excess.[15] Subjects were not permitted binocular stimulation between or during this second set of measurements.

Results

The results are summarised in Table 1. Abnormally high mean stimulus AC/A ratios were found with the distance/near cover (10·4/1) and phorometric (12·3/1) tests. However, with near gradient measures the mean AC/A values were clearly reduced (5·0/1, 8·1/1). Further, based on the objective findings, in which distance/near effects were minimised, response AC/A ratios were generally within normal limits (mean=5·9/1; range=4·5/1 to 8·0/1), although biased toward the high side. However, our objectively determined mean stimulus AC/A ratios were clearly within normal limits (mean=4·2/1, range=3·6/1 to 4·9/1). The most consistent pre- and postocclusion AC/A ratios were obtained with the objective technique. Pre- and postocclusion response AC/A ratios were similar in all 4 subjects, with the mean preocclusion value being within one standard deviation of the mean postocclusion value. On the basis of similarity of distance/near values of the strabismic deviation following occlusion, subjects 3 and 4 were classified as simulated divergence excess. On the basis of consistent dissimilarity of the distance/near value of the strabismic deviation following occlusion, subjects 1 and 2 were classified as true divergence excess.

The responses of 2 subjects representing each diagnostic group are presented in Fig. 2. These records provide an indication of the slight fluctuations of accommodation and of the synkinetically linked accommodative vergence during attempted steady fixation and focus on the target from which objective response and stimulus AC/A ratios were determined. Dynamic overshoots of accommodation were frequently present in the responses of subject 4, but these transients were not included in estimating the steady-state accommodation level.

Discussion

It is generally believed that the AC/A ratio is abnormally high in individuals having intermittent exotropia of the divergence excess type. Like others we too found abnormally high stimulus AC/A ratios in our subjects, but only if distance/near measurements were used in the calculation. If the near gradient or the objective technique was used, AC/A ratios were generally within normal limits, in agreement with von Noorden.[10] Further, Cooper[22] has questioned the validity of these high AC/A ratio measures, since presence of an abnormally high AC/A ratio cannot explain the following clinical findings associated with divergence excess. First, over 75% of all patients with divergence excess exhibit a dramatic increase in the near deviation following a short period of monocular occlusion.[10] Such an increase in the near deviation results in a dramatic decrease in the calculated stimulus AC/A ratio, and this is evident in our subjects 3 and 4 (Table 1). This change in the stimulus AC/A ratio is surprising, since occlusion does not affect the stimulus to accommodation. Secondly, there is frequently (25% of the cases) a

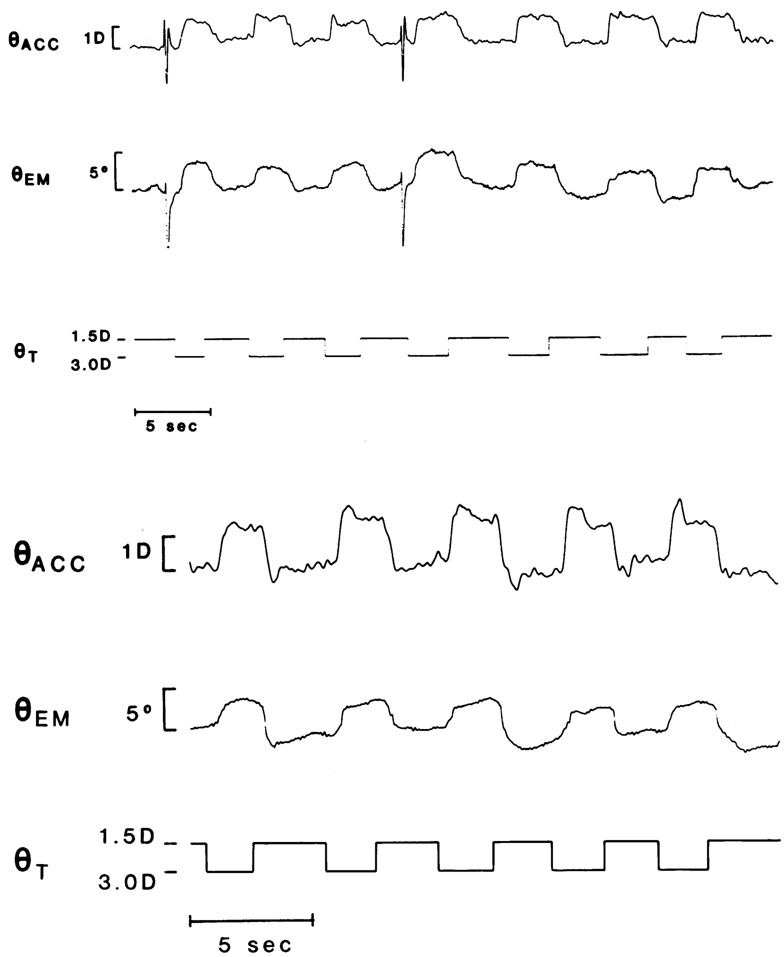

Fig. 2 *Records of accommodation in the viewing left eye (θ_{ACC}), accommodative vergence in the occluded right eye (θ_{EM}), and target position (θ_T). (a) Subject 1. True divergence excess exotropia. Two large deflections are due to blinking. (b) Subject 4. Simulated divergence excess exotropia. Dynamic overshoots of accommodation present in most responses.*

dramatic increase (4 to 10 Δ) in the distance exodeviation when the fixation distance is changed from 20 to 100 feet (6–30 m)[11] (a 0·13 dioptre change in stimulus to accommodation). Such an AC/A-related change in the exodeviation necessitates the presence of an exceedingly high (up to 100/1) AC/A ratio, which is conceptually difficult to accept. Further, it suggests gross nonlinearity of the AC/A ratio in these patients. Thirdly, a patient with an abnormally high AC/A ratio and a large exodeviation should stress his (normal) negative relative accommodation capability during periods of fusion, resulting in a complaint of intermittent blur. However, this is not commonly reported. Lastly, if the deviation is AC/A-dependent, then after surgical correction of the exotropia a large residual esodeviation should be evident at near vision. However, after operation large esodeviations rarely occur at near vision,[23] and the exotropia often recurs.[24] None of the above ideas are consistent with the notion of an abnormally high AC/A ratio in patients with divergence excess.

Others have argued in favour of the distance/near type of stimulus AC/A ratio in divergence excess patients on the basis of its clinical usefulness, especially with respect to determination of the appropriate surgical procedure for strabismus.[2–3] We do not deny, nor wish to detract from, its usefulness to the clinician. However, we believe it is important from a scientific point of view to determine the true AC/A ratio with the hope that this information will provide insight into the physiological mechanisms underlying the divergence excess condition. Our objective findings indicated relatively normal AC/A ratios. Thus the lack of a large exodeviation at near vision and the enhanced binocularity at near cannot be accounted for by evoking the use of an abnormally high AC/A ratio to align the eyes for proper binocular sensory integration. A further point supporting the objective determination of AC/A ratios in these subjects can be made with respect to the uncertainty of the clinical measures due to the presence of sensory abnormalities and also to assumptions related to the response of accommodation. Most clinical estimates of the stimulus AC/A ratio depend upon a subjective measure of vergence changes, which demands the presence of normal retinal correspondence and absence of suppression during measurements. Neither condition is always present in these patients.[25] Further, the assumption is made that the accommodative stimulus and response are equivalent, which is not true in normal persons[12] and was not the case in our patients having intermittent exotropia. Our objective recording procedure was not influenced by abnormal direction sense, as found in anomalous retinal correspondence, nor by the suppression of input to the deviated eye, as is frequently found in these patients.[25] Further, the accommodative responses were objectively recorded, which by-passed the need to make assumptions regarding the accommodative stimulus/response relationship.

While some may argue that our population of subjects with divergence excess was atypical, since their mean distance deviation (13·8 ΔXT) was less than commonly reported (29 ΔXT),[6] we believe this not to be the case. First, all subjects met the Burian-Spivey criterion[17] of having at least a 10 Δ preocclusion difference between the distance and near deviation. Secondly, as pointed out by Burian,[26] in many patients with simulated divergence excess the near deviation increases as adulthood approaches, and they begin to lock like a basic exotrope with distance and near exodeviations being similar. Because of the stringent requirements essential for obtaining accurate measures of accommodation and accommodative vergence with our objective recording devices, only co-operative adults could be used, and thus our subject pool was biased in this more basic exotropic direction. Thirdly, all subjects had sensorimotor characteristics typical of the patients with divergence excess.[6] For example, there was increased frequency of exodeviation with inattention, exophoria at near vision, anomalous retinal correspondence and/or suppression while deviating, and presence of normal stereopsis when bifixating. On the basis of these arguments we believe our sample of subjects to be representative of the adult divergence excess population.

We believe that the abnormally high, clinically determined stimulus AC/A ratios are contaminated by at least 2 factors: fusional convergence after-effects and proximal convergence effects. The fusional convergence after-effect[27] refers to a transient (20 seconds or more[28]) change (up to 10 Δ) in the fusion-free position of the eyes following a short period (several seconds to a few minutes) of sustained vergence. Fusional convergence after-effects are commonly observed in normal patients as a change in the phoria position following a determination of relative vergence ranges.[27] A relative increase in esophoria is found following brief binocular fusion through base-out prisms, and a relative increase in exophoria is found following brief binocular fusion through base-in prisms.[19] Thus, in individuals having simulated divergence excess, in which there is a large relative increase in exodeviation after 45 minutes of monocular occlusion, we believe this postocclusion change is a direct manifestation of a fusional convergence after-effect resulting from sustained fusional convergence required to bring the eyes from a divergent position to one of binocular alignment on the object of interest. If the initial cover test is performed, a certain fusion-free position of the eyes is found. However, after 45 minutes of occlusion, which is apparently sufficient for decay of the fusional convergence after-effect in these individuals, a marked increase in exodeviation was revealed. This after-effect appears to be especially pronounced at near vision, where the increased frequency of fusion demand may result from increased presence and value of stimulus parameters related to fusion and processing of depth information, such as small, angular disparities, increased size of fusion contours of objects of interest within the central field, and the association of tactile information with visual information at close distances.

Any decrease in the frequency of fusion in individuals with divergence excess results in an increase in the exodeviation. This may be noted by observing the effects of fixation disparity measurements, cover test findings, and prolonged occlusion on the magnitude of the deviation. When true (full-field) dissociation does not occur, as during the measurement of fixation disparity, these patients exhibit eso- or minimal exofixation disparity.[29] The initial findings of the rapid cover test reveal an exodeviation. Prolonged occlusion results in uncovering the full extent of the exodeviation. Thus, the more complete and the longer the monocular occlusion, the greater the exodeviation. This is consistent with the notion of a decay in the fusional convergence after-effect.

The role of the fusional convergence after-effect is further supported by the results of orthoptic treatment in these patients. There is frequently a dramatic decrease in frequency and magnitude of deviation, as measured by the routine cover test, after successful orthoptic treatment.[30 31] However, prolonged occlusion will reveal the original deviation. Thus monocular occlusion appears to influence the fusional vergence system only, without affecting accommodative, tonic, or proximal vergence factors. The fact that proximal factors probably do not play a role in simulated divergence excess is evidenced by the similarly high AC/A ratios obtained with distance/near as well as gradient-type measures.

Individuals with the true divergence excess also showed abnormally high stimulus AC/A ratios by distance/near measure. However, since the *difference* in exodeviation was not affected by prolonged occlusion, a fusional convergence after-effect at near only, as was speculated in the case of simulated divergence excess, cannot explain this finding. We suggest rather that proximal convergence factors predominate, as near gradient stimulus AC/A ratios were typically much reduced relative to the distance/near values. However, we do believe that fusional convergence after-effects are also involved, but affect the vergence system at all distances approximately equally, as shown by the increase in exodeviation of the fusion-free position of the eyes at both distance *and* near. This difference in influence of a fusional convergence after-effect may be related to differences in frequency of fusion at distance and near vision in the simulated and true divergence excess patients. This idea warrants further careful investigation.

The results of our investigation lead one back to Maddox,[16] who defined the 4 components of vergence: tonic, proximal, accommodative, and fusional. He believed that accommodative vergence was primary, with fusional vergence serving only a supplementary role. Proponents of the abnormally high (stimulus) AC/A ratio in patients with divergence excess likewise suggest that accommodative vergence is the primary, and perhaps the sole, component used to align the eyes from the distance unfused (deviated) position to the near fused position. However, our finding of relatively normal (response) AC/A ratios in these patients forces one to accept a less dominant role for accommodative vergence. More recent investigations in normal persons[32 33] provide strong evidence that fusional rather than accommodative vergence is the dominant component. On the basis of our findings in individuals with divergence excess we would like to extend this concept and suggest that fusional as well as proximal vergence play important roles in the near fusion response. However, the exact proportion of each vergence component used to achieve fusion awaits further investigation.

This work was supported in part by NIH grant EY03541 to K.J.C.

References

1 Duane A. A new classification of the motor anomalies of the eye based upon physiological principles. *Ann Ophthalmol* 1897; **6:** 84–122.

2 Jampolsky A. Ocular divergence mechanisms. *Trans Am Ophthalmol Soc* 1970; **68:** 730–822.

3 Parks MM. *Ocular Motility and Strabismus*. New York: Harper and Row, 1975.

4 Allen MJ. How do you treat intermittent exotropia? *Optom Weekly* 1968; **59:** 298–9.

5 Brown HW. Accommodative convergence in exodeviation. *Int Ophthalmol Clin* 1971; **11:** 39–45.

6 Bair DR. Diagnosis and incidence. *Am Orthopt J* 1952; **2:** 12–7.

7 Ogle KN, Martens TG, Dyer JA. *Oculomotor Imbalance in Binocular Vision and Fixation Disparity*. Philadelphia: Lea and Febiger, 1967.

8 Ogle KN, Mussey F, Prangen A de H. Fixation disparity and the fusional processes in binocular single vision. *Am J Ophthalmol* 1949; **32:** 1069–87.

9 Moore S, Stockbridge L, Knapp P. A panoramic view of exotropias. *Am Orthopt J* 1977; **27:** 70–9.

10 Noorden GK von. Divergence excess and simulated divergence excess: diagnosis and surgical management. *Doc Ophthalmol* 1969; **26:** 719–28.

11 Burian HM, Smith DR. Comparative measurement of exodeviations at twenty and one hundred feet. *Trans Am Ophthalmol Soc* 1971; **69:** 188–96.

12 Alpern M, Kincaid WM, Lubeck MJ. Vergence and accommodation: III. Proposed definitions of the AC/A ratios. *Am J Ophthalmol* 1959; **48:** 141–8.

13 Ogle KN, Martens TG. On the accommodative convergence and the proximal convergence. *Arch Ophthalmol* 1957; **57:** 702–15.

14 Hokoda S, Ciuffreda KJ. Theoretical and clinical importance of proximal vergence. In: Schor CM, Ciuffreda KJ, eds. *Basic and Clinical Aspects of Vergence Eye Movements*. Boston: Butterworths, in press.

15 Burian HM, von Noorden GK. *Binocular Vision and Ocular Motility*. St Louis: Mosby 1974.

16 Maddox E. Investigations on the relationship between convergence and accommodation of the eyes. *J Anat* 1886; **20:** 475–505, 565–584.

17 Burian HM, Spivey BE. The surgical management of exodeviation. *Trans Am Ophthalmol Soc* 1964; **62:** 276–302.

18 Cooper J, Feldman J. Random-dot-stereogram performance by strabismic, amblyopic, and ocular pathology patients in an operant-discrimination task. *Am J Optom Physiol Opt* 1978; **55:** 599–609.

19 Borish IM, *Clinical Refraction*. 3rd ed. Chicago: Professional Press, 1970.

20 Kruger PB. Infrared recording retinoscope for monitoring accommodation. *Am J Optom Physiol Opt* 1979; **56:** 116–23.

21 Stark L, Bahill AT, Ciuffreda KJ, Kenyon RV, Phillips S. Neuro-optometry: an evolving specialty clinic. *Am J Optom Physiol Opt* 1977; **54:** 85–96.

22 Cooper J. Intermittent exotropia of the divergence excess type. *J Am Optom Assoc* 1977; **48:** 1261–73.

23 Dyer JA, Martens TG. Surgical treatment of divergence excess. *Am J Ophthalmol* 1960; **50:** 297–302.

24 Cooper EL. Purposeful overcorrection in exotropia. In: Arruga, ed. *International Strabismus Symposium*. Basel and New York: University of Giessen, 1968: 311–8.

25 Cooper J, Feldman J. Panoramic viewing, visual acuity of the deviating eye, and anomalous retinal correspondence in the intermittent exotrope of the divergence excess type. *Am J Optom Physiol Opt* 1979; **56:** 422–9.

26 Burian HM. Exodeviations: their classification, diagnosis and treatment. *Am J Ophthalmol* 1966; **66:** 1161–6.

27 Alpern M. The after effect of lateral duction testing on

subsequent phoria measurements. *Am J Optom Physiol Opt* 1946; **23:** 442–7.
28 Ludvigh E, McKinnon P, Zaitzeff L. Temporal course of the relaxation of binocular duction (fusion) movements. *Arch Ophthalmol* 1964; **71:** 389–99.
29 Ogle KN, Dyer JA. Some observations on intermittent exotropia. *Arch Ophthalmol* 1965; **73:** 58–73.
30 Goldrich SG. Optometric therapy of divergence excess strabismus. *Am J Optom Physiol Opt* 1980; **57:** 7–14.
31 Goldrich SG. Oculomotor biofeedback therapy for exotropia. *Am J Optom Physiol Opt* 1982; **59:** 306–17.
32 Fincham EF, Walton J. The reciprocal actions of accommodation and convergence. *J Physiol* 1957; **137:** 488–508.
33 Semmlow JL. The oculomotor near response. In: Zuber BL, ed. *Models of Oculomotor Behavior*. Miami: Chemical Rubber Pub. Co., 161–91.