MONITORING OF CONTROLLED ACCOMMODATIVE ESOTROPIA*

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ABSTRACT

Purpose: To ascertain an examination interval that will not increase the risk of untimely detection of decompensation of accommodative esotropia whether or not initial nonoperative treatment must be supplemented.

Methods: The records of 63 patients with controlled accommodative esotropia examined at 3- to 6-month intervals were reviewed for age at first control, the occurrence of decompensation, initial refraction and subsequent changes, and the need for increased correction of hyperopia or the addition of bifocals.

Results: Decompensation occurred in 11 patients, not associated with substantial refractive changes toward or away from emmetropia. No instance of decompensation occurred in the first 12 months of observation, and only 11.5% occurred within 2 years. Although 7 of these decompensated patients were among the 18 (28.6%) requiring supplemental nonoperative treatment, their mean initial hyperopia and annual refractive change did not differ significantly from the 11 patients who did not decompensate. Eight (18.6%) of 43 patients who were first controlled earlier than age 48 months later decompensated; 3 (15.0%) of 20 patients with later onset reached this outcome.

Conclusions: Monitoring controlled accommodative esotropia at intervals of 9 to 12 months is adequate for most patients, at least over the first 2 years, other than those requiring treatment for associated conditions such as amblyopia. Refractive error changes and the need for supplemental treatment after initial control are not prominently associated with decompensation. Age at onset of accommodative esotropia earlier or later than 48 months did not influence rapidity of decompensation.

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INTRODUCTION

According to conventional teaching, patients with accommodative esotropia require close follow-up, especially prior to age 6 years.[1] In addition to monitoring for the development of amblyopia, such follow-up is thought to ensure detection of (1) decompensation to a nonaccommodative deviation, a sequel affecting 11% to 48% of these patients,[2-5] and (2) the need for increased treatment measures to continue adequate control[6] or opportunities to incrementally reduce treatment to encourage the expansion of fusional divergence.[7]

In the author's experience, once satisfactory alignment has been obtained, visits at the 3- to 6-month intervals usually recommended often uncover none of these "events." Fewer visits that still accomplish the goals of treatment can simplify the management of such cases, an important advantage in the managed care setting.

This report examines whether lengthening the interval between examinations of patients with accommodative esotropia still allows timely discovery of features determining changes in their treatment.

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SUBJECTS AND METHODS

This retrospective study included 63 patients observed for at least 6 months after initial treatment of accommodative esotropia reduced the deviation to no more than 10 prism diopters (PD), as determined by prism and alternate cover testing, approximately 6 weeks after prescription of the appropriate cycloplegic correction, with a bifocal addition if indicated. Exclusion criteria were major neurologic conditions, prominent nystagmus, extraocular muscle palsy, restricted rotations, and Duane syndrome. Coexisting vertical deviations, oblique dysfunctions, prior surgery for infantile esotropia, and amblyopia were not reasons for exclusion, as the behavior of accommodative esotropia in these settings is similar to that in cases without these additional attributes.[3,8]

Decompensation was defined as a primary position distant esodeviation, originally but no longer reduced to 10 PD or fewer by control of accommodation. No patient entered the study already showing decompensation (ie, all entered as cases of accommodative esotropia responsive to treatment). Thirty-three patients were initially evaluated while under successful treatment that had been instituted elsewhere. This precluded reliably establishing a date of onset of the deviation. Duration from the author's verification of control to either decompensation or the

need for more intense treatment was determined for each subject.

The author performed all examinations. Retinoscopy was accomplished 45 to 60 minutes after 2 instillations of 1% cyclopentolate hydrochloride. Measurements of refractive error refer to the eye preferred for fixation. Refractive error determinations usually were made at approximately annual intervals. Changes in hyperopia were annualized by extrapolation over the longest interval from initial examination up to age 8 years (the typical peak for any increases[9]) for which a subsequent measurement was available.

Initial treatment consisted of prescription of the full, or within 0.50 D of the full, cycloplegic retinoscopic findings. When a high near-to-distance alignment comparison was present,[3,4] a bifocal addition of +2.50 D sphere to both eyes was employed. Some patients subsequently required a stronger refractive correction or inclusion of a bifocal for residual uncompensated esotropia.

Means, standard deviations, and significance determinations were calculated using the 2-tailed *t* test for unpaired data. Although results at further intervals are included, emphasis is on outcomes within the first 2 years of observation.

RESULTS

Decompensation was observed in 11 patients, at a mean interval from first observation of 45.1 months (range, 13 to 169 months). Of 60 patients followed up for at least 1 year, none showed decompensation within that period. Two (18.1%) of the 11 patients decompensating did so within 18 months, and 6 (54.5%) within 2 years (Table I). This represents 3.7% and 11.5%, respectively, of the patients observed at these intervals.

As in most retrospective studies, follow-up was not uniform. A modified life table approach was used to further define decompensation rates. The pooled decompensation rate of 21.4% determined in prior studies[1,3,4] suggests that no more than 1 of the 3 patients (1.5% of the entire series) not examined up to 1 year after establishment of control should be assumed to have decompensated in that interval. Use of this method of correcting for later follow-up loss leads to the estimate of 15 patients decompensating, with only 3 (20%) occurring within 18 months and 8 (53.3%) within 24 months (Table I).

Eighteen of the 63 patients whose refractive correction initially had been sufficient later required increased treatment to maintain control, as a first event. Eleven patients received this assistance in the first year after initial observation. They accounted for only 3 (27.3%) of those who eventually decompensated. Six patients (3 in the second year) needing stronger treatment after the first year later furnished 3 to the group later decompensating (Table II).

TABLE I: INTERVAL TO DECOMPENSATION

INTERVAL FROM FIRST OBSERVATION (MO.)	PATIENTS OBSERVED	PATIENTS DECOMPENSATING	
		OBSERVED*	ASSUMED*
0-6	63	0	0
7-9	61	0	0
10-12	60	0	1
13-15	58	1	2
16-18	54	2	3
19-21	54	4	5
22-24	52	6	8
25-27	50	6	8
28-30	44	6	9
31-33	42	7	10
34-36	36	8	12
37+	33	11	15

*Cumulative.

There were 40 patients in this series whose first event had been weakening of the refractive correction with maintenance of satisfactory alignment. Only 2 (5.0%) of these patients eventually decompensated, at 19 and 54 months, respectively. Moreover, of 9 patients in this group who required a reinstatement of stronger accommodation control as a second event after this first reduction, only 1 decompensated, 62 months after this measure (not tabulated).

Initially determined hyperopia was distributed normally in the series. For the decompensated patients and for patients remaining controlled, mean initial hyperopia, standard deviation, and range were clinically and statistically the same (Table III). This result is consistent with a

TABLE II: DECOMPENSATION AFTER STRONGER TREATMENT

INTERVAL FROM FIRST OBSERVATION (MO)	PATIENTS REQUIRING* INCREASED Rx*	PATIENTS LATER DECOMPENSATING*
0-6	6	2
7-9	9	3
10-12	11	3
13-15	15	5
16-18	16	5
19-21	17	6
22-24	17	6
25-27	17	6
28-30	17	6
31-33	17	6
34-36	17	6
37+	18	7

*Cumulative.

prior study.[3]

The mean annual change in hyperopia for the decompensating patients was clinically similar to the results of other studies of decompensation[3] and of unselected, principally nonstrabismic patients.[9] The same determination for patients remaining controlled is clinically comparable, and statistically the differences are not significant (Table IV).

Initial hyperopia for the subgroup of patients whose first event was stronger treatment and who later decompensated was similiar for those whom stronger treatment re-stablized (Table V). Likewise, both segments of this group showed only very modest, statistically and clinically similar annual increases in hyperopia (Table VI).

Because the tendency to decompensate presumably is aggravated by the more precise requirement for sustained accommodative (despite optical compensation) and convergence effort in older children as their intellectual maturity increases, the possible influence of age at initial control on the subsequent appearance of decompensation was examined. Forty-eight months was selected as the reference age. Of 43 patients first controlled earlier than this age, 8 (18.6%) decompensated. Three (15.0%) of 20 patients first controlled at later than 48 months of age reached this outcome. The difference in these rates is not significant. For both age-groups, the interval from control to decompensation also was not significantly different (Table VII). A similar analysis based on age 30 months, the reported mean age at onset of accommodative esotropia,[1(p99)] gave comparable results (not tabulated).

TABLE III: INITIAL HYPEROPIA

PATIENTS	MEAN (D)	SD (D)	RANGE (D)
Decompensated (N = 11)	3.77	1.56	2.00 to 7.00
Controlled (N = 52)	4.00	1.82	1.25 to 8.00
		P = 0.35	

D, diopters.

TABLE IV: ANNUAL CHANGES IN HYPEROPIA

PATIENTS	MEAN (D PER YR)	SD (D)	RANGE (D PER YR)
Decompensated (N = 11)	+0.07	0.58	-1.00 to +0.88
Controlled (N = 50*)	-0.05	0.51	-1.44 to +1.37
		P = 0.53	

D, diopters.
*Information unavailable for 2 patients.

TABLE V: INITIAL HYPEROPIA AND STRONGER TREATMENT

PATIENTS	MEAN (D)	SD (D)	RANGE (D)
Decompensated (N =7)	3.82	1.68	2.00 to 7.00
Controlled (N = 11)	5.05	2.04	2.25 to 8.00
		P = 0.20	

D, diopters.

TABLE VI: ANNUAL REFRACTIVE CHANGES AND STRONGER TREATMENT

PATIENTS	MEAN (D)	SD (D)	RANGE (D)
Decompensated (N = 7)	+0.17	0.61	-1.00 to +0.88
Controlled (N = 11)	+0.12	0.36	-0.38 to +0.82
		P = 0.89	

D, diopters.

TABLE VII: AGE AND DECOMPENSATION

AGE (MO)	NO. OF PATIENTS	NO. (%) DECOMPENSATING	INTERVAL (MO) FROM CONTROL (MEAN, SD)
≤48	43	8 (18.6)	47.4 ± 53.5
>48	20	3 (15.0)	39.0 ± 20.2
			P = 0.81

DISCUSSION

Management of these patients had been carried out according to usually advocated principles.[1,6] The study questions were prompted by the retrospective observation that very often 1 or more periodic examinations had been superfluous, resulting only in continuation of treatment without change.

Extension of the time between examinations is desirable if it does not compromise care. These results indicate that decompensation is unlikely to occur in less than 12 months after control of accommodative esotropia is initially established. Moreover, the need for supplemental treatment, even if arising in the first year of monitoring, usually is not a warning that closer follow-up is necessary. Not answered by this study is whether, if follow-up intervals are extended from 6 to even 12 months, a relatively short delay in detecting decompensation adversely affects the final sensory and motor outcome.

This does not support the classic notion that progressive, substantially increasing hyperopia requiring

continued exertion of accommodation and its associated convergence is among the prominent causes of decompensation. Further, the refractive change findings seen here suggest again that emmetropization, considered by some to be a universal tendency, is not typically found in accommodative esotropia patients younger than 8 years of age.[3]

CONCLUSIONS

Omitting examination for 1 year after verifying control of the accommodative esotropia would not have delayed the detection of any patient's decompensation. Deferral of follow-up even to 18 months would not have prevented detection for the majority of these patients.

While less than optimal control of accommodative esotropia may logically be thought to invite decompensation, patients with unstable alignment due to the need for stronger treatment were not more susceptible to decompensation in the first year after initial treatment compared to later intervals.

Observation at an interval of less than 18 months following reduction of initial treatment, whether or not remaining entirely adequate to maintain control, would not have identified decompensation, a result consistent with that of a prior study.

Initially determined hyperopia was not predictive of decompensation.

Since decompensation after control of accommodative esotropia usually is not due to large, rapid increases in hyperopia, closer follow-up for the purpose of repeated refraction would not have improved the identification of future decompensation.

Age at onset of accommodative esotropia was not determined to be a risk factor for early decompensation.

Unless there are coexisting problems, such as amblyopia, inferior oblique overaction, or DVD, follow-up for controlled accommodative esotropia, at least over the first 2 years following the attainment of satisfactory control, can be extended to 9 to 12 months.

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DISCUSSION

DR PAUL R. MITCHELL. Dr Raab has presented a new concept in the monitoring of accommodative esotropia, at intervals of 9 to 12 months, over the first 2 years after control has been established, unless there are associated conditions such as amblyopia, inferior oblique overaction, and dissociated vertical deviations. The records of 63 patients with accommodative esotropia examined at 3 to 6 month intervals were reviewed in a retrospective study, for the occurrence of decompensation, changes in refractive error, and the need for increased hyperopic correction or the addition of bifocals. The 63 patient group included 33 patients who were initially evaluated while under successful treatment which had been instituted elsewhere, which precluded the opportunity of establishing accurately a date for the onset of the eye deviation.

Dr Raab excluded those patients with major neurologic conditions, prominent nystagmus, extraocular muscle palsy, restricted rotations, and Duane syndrome. But, he did not exclude coexisting vertical deviations, oblique dysfunctions, prior surgery for infantile esotropia, and amblyopia.

In Table I, Dr Raab describes decompensation in 11 patients, 17.5% of the group of 63 patients. Twenty-one patients were lost to follow-up, and by the end of the period of study of 37+ months, a total of 33 patients had been observed. The exact number of decompensations of the 21 patients lost to follow-up can only be hypothesized. If all 21 decompensated, then the rate of decompensation could be 11+21/63 (51%). If none of the 21 decompensated, then the rate would be 17.5%. The true percentage is somewhere between these 2 numbers. In evaluating the number of decompensations per time interval, the percentage increases with time, as fewer were observed and as more patients were lost. At the 12 months interval, 0% decompensation, at 18 months, 2/54 (3.7%), at 24 months, 6/52 (11.5%), at 30 months, 6/44 (13.6%), at 36 months, 8/36 (22.2%) and at 37+ months 11/33 (33%) decompensation.

In Table II, 18 patients of the 63 required increased hyperopic correction, a bifocal, or supplementary anticholinesterase medication. With each time interval, the percentage of decompensation increased. At 12 months, 3/11 (27.3%), at 18 months, 5/16 (31.3%), at 24, 30, and

TABLE I: INTERVAL TO DECOMPENSATION

INTERVAL FROM FIRST OBSERVATION (MONTHS)	PERCENTAGE	PATIENTS OBSERVED	PATIENTS DECOMPENSATING	PATIENTS LOST
0-6		63	0	0
7-9		61	0	2
10-12		60	0	1
13-15		58	1	2
16-18	2/54=3.7%	54	1	3
19-21		54	2	0
22-24	6/52=11.5%	52	2	3
25-27		50	0	3
28-30	6/44=13.6%	44	0	0
31-33		42	1	6
34-36	8/36=22.2%	36	1	0
37+	11/33=33%	33	3/11	1/21

TABLE II: DECOMPENSATION AFTER STRONGER TREATMENT

INTERVAL FROM FIRST OBSERVATION (MONTHS)	PERCENTAGES	PATIENTS REQUIRING	PATIENTS LATER DECOMPENSATING
0-6		6	2
7-9		3	1
10-12	3/11=27.3%	2	0
13-15		4	2
16-18	5/16=31.3%	1	0
19-21		1	1
22-24	6/17=35.3%	0	0
25-27		0	0
28-30	6/17=35.3%	0	0
31-33		0	0
34-36	6/17=35.3%	0	0
37+	7/18=38.9%	1/18	1/7

36 months, 6/17 (35.3%), and at 37+ months, 7/18 (38.9%). These were 7 of the 11 patients who decompensated in Table I.

There are a number of areas of concern with this paper, not only with the way the data was collected and presented, but also with the conclusion. The paper is not a summary of pure accommodative esotropia and the conclusions should be tempered. Were the patients selected at random, in sequence, or selectively chosen? Were they excluded if their deviation with eyeglass correction was more than 10 diopters? What diagnosis did each patient have, besides accommodative esotropia? Of the 33 patients treated elsewhere and acquired by Dr Raab, more information would be of value. Which patients were they, of the 63 total, and when did they decompensate, relative to patients treated initially by Dr Raab? Was there any difference in the rate of decompensation? If Dr Raab's initial patients did not decompensate for a year, then why did those patients …"evaluated while under successful treatment that had been instituted elsewhere" not show a tendency to decompensate sooner? Or did they decompensate at the same rate? Dr Raab stated that the date of onset of the deviation was not reliable. Should these 33 patients have been included if the date of onset

of the deviation was not reliable? Should they have been tabulated separately?

Pure accommodative esotropia presents intermittently between 7 months of age and 7 or 8 years of age, with an average of 2 1/2 years, typically with binocular vision already established prior to the onset of the deviation. Accommodative esotropia developing after early surgery for congenital esotropia has successfully aligned the eyes, reveals a different sensory and motor presentation, including oblique muscle dysfunction, and the dissociated strabismus complex. The major differences in complexity are not discussed in this paper, by grouping these patients together, and there is no indication in the outcome tables as to which patients have which condition. The manuscript briefly mentions that amblyopia should be treated, and sensory data is not detailed except for a comment in the discussion: "if…a relatively short delay in detecting decompensation adversely affects the final sensory and motor outcome."

Dr Raab's definition of decompensation is "distant esodeviation, originally but no longer reduced to 10 prism diopters or fewer by control of accommodation." In fact, deterioration is a far more serious matter, than just alignment status measured in prism diopters. Deterioration is the replacement of intermittent esotropia with constant esotropia, which has the potential to create a lifelong problem. The loss of bifixation and absence of alternation results in amblyopia. With decompensation of accommodative esotropia and 3 months of constant esotropia, bifixation is lost forever. The monofixation syndrome is the result.

Pratt-Johnson admonished all ophthalmologists to develop a special routine in treatment of accommodative esotropia. One should consider that any child with the onset of intermittent esotropia, which after workup appears to have accommodative esodeviation, deserves treatment as soon as possible after onset, to prevent losing bifixation and having to resort to monofixation. For this reason, the only logical treatment is for the children to be examined as soon as possible after the onset of the intermittent esodeviation, and followed closely to detect a trend toward decompensating to comitant esotropia after originally being compensated by anti-accommodative therapy.

Perhaps the data presented in the manuscript suggests that because decompensation did not occur in the first year of treatment, that the frequency of examinations should then be increased in subsequent years, when decompensation is more likely to occur. The lack of compliance in wearing eyeglasses certainly rates as one of the leading causes of decompensation. Children's eyeglasses are prone to damage or loss, often resulting in the absence of wearing them for lengthy periods of time. Repeated parental instruction about the importance of constant eyeglass wear is part of the responsibility assumed by the ophthalmologist caring for these vulnerable young patients. Increasing the intervals between visits, as Dr Raab's conclusion in this paper seems to advocate, must be weighed against the potential problems this policy could cause.

Thank you for the opportunity to discuss this paper, and I thank Dr Raab for providing the manuscript and tables in a timely fashion.

[Editor's note] DR MALCOLM R. ING asked about the role of compliance in the incidence of decompensation. DR DAVID L. GUYTON asked about the effect of residual accommodation and whether an adequate cycloplegic refraction was obtained. DR JOHN F. O'NEIL asked if the difference in time of onset of the esotropia influenced the rate of decompensation. DR ALLAN J. FLACH asked about the duration of follow-up and the problems presented by patients who do not keep their follow-up appointments.

DR EDWARD L. RAAB. I am pleased that the Program Committee included this presentation in our meeting, and that it has led to a substantial amount of thoughtful comment.

Dr Mitchell mentioned the imprecision in establishing a date of onset for the many patients that were already under care successfully when I became their ophthalmologist. He is correct, but if it influences the results at all, it actually extends the time they remained under initial control and I think strenghthens my observations.

Dr Mitchell also discussed follow-up loss. The manuscript will reflect that I handled follow-up loss by deriving an average decompensation rate of 21.4% from prior reported studies, so that if 3 patients were not observed over the entire first year, and rounding up to the nearest whole patient, I assumed that one of those would have decompensated. Therefore, only one of 63 patients would have been missed if not reexamined for an entire year. Employing the same analysis at later intervals leads to the same conclusion.

As I stated, my particular interest was in the first 2 years. Beyond that, or once there are signs of impending decompensation, whether or not it is advisable to maintain an extended follow-up interval is undetermined. I agree with Dr Mitchell that this question is examined best in a randomized, controlled trial comparing outcomes with shorter and longer intervals, but I cannot offer that information based on this work. The series was obtained from a section of my files and were identified by a color code. I have enough material for a much larger series, but it would be no less retrospective.

Dr Ing asked whether compliance was an issue here. Rather than how many patients decompensated or why

they decompensated, I was looking at *when* they decompensated. That was the practical question I was addressing, so I can't really give an answer on the compliance factor.

Dr Guyton queried about adequacy of cycloplegia. I was taught that the definition of adequate cycloplegia is not based on the drug that was used, but on the residual accommodation in the particular patient with whatever agent was given. If there is no more than about a diopter of residual accommodation on dynamic retinoscopy, i.e., with distant fixation and then with near fixation, this would be considered clinically adequate cycloplegia. None of these patients had atropine. All had cyclopentolate 1%, and none were examined before 45 minutes after the first instillation. Although I did not investigate this in every patient in this study, my habit in the ordinary course of practice is to "spot check" periodically, and of those that I did check, I thought there was adequate cycloplegia.

I did not examine the emmetropization question, so I cannot answer Dr Guyton's question about whether "pushing plus" retards this phenomenon. My concern was the downside risk of decompensation, and I would be looking to push rather than cut back when I thought this was about to occur.

Dr O'Neill asked about early v. late onset and more difficult management of patients presenting very early. Yes, in general that has been my experience, although not overwhelmingly. I did not analyze it for the purposes of this study.

Dr Flach suggests that a longer follow-up interval could imply an attitude on the practioner's part that returning is not important. I have not noticed this since adopting my conclusion, but it is a possibility. As to his other question, I have not been contacting accommodative esotropia patients about follow-up unless they are under treatment for amblyopia or threatening to decompensate. I certainly do this for such conditions as congenital glaucoma, aniridia or Sturge-Weber syndrome where glaucoma is a prominent possibility, and acquired extraocular muscle palsies, and I agree that insuring adequate follow-up of whatever we are treating is a worthwhile goal.

Thank you again for your interest.