Does Overcorrecting Minus Lens Therapy for Intermittent Exotropia Cause Myopia?

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**Background:** Overcorrecting minus lens therapy has been used as a treatment for intermittent exotropia. It is based on the principle that an exotropic deviation will be decreased by stimulating accommodative convergence with additional minus power in spectacles. Because excessive accommodation has been implicated as a cause of myopia, there is theoretical concern that overcorrecting minus lens therapy for exotropia may cause myopia.

**Objective:** To investigate the effect of overcorrecting minus lens therapy for exotropia on the progression of myopia.

**Design:** A retrospective chart review.

**Subjects and Methods:** Seventy-four patients with intermittent exotropia were treated with overcorrecting minus lens therapy for at least 6 months (6-month treatment group), and a 34-patient subset of them received overcorrecting minus lens therapy for 5 years (5-year treatment group). The mean change in refractive error (spherical equivalent of the fixing eye) of these 2 groups 5 years after initial examination was compared with the mean change in refractive error of a control group of 45 patients with intermittent exotropia who did not receive overcorrecting minus lens therapy.

**Results:** At the time of initial examination, the mean (±SD) refractive error was 0.00 ± 1.40 dipters (D) in the control group, 0.00 ± 1.50 D in the study group, and −0.10 ± 1.50 D in the 5-year study group, all of which were essentially identical. Five years after initial examination, the mean change in refractive error was −1.40 ± 2.80 D in the control group, −1.52 ± 1.80 D in the 6-month treatment group, and −1.54 ± 1.80 D in the 5-year treatment group. These differences in the change in refractive error (myopic shift) were not statistically significant (t test), and the differences are clinically unimportant.

**Conclusion:** Overcorrecting minus lens therapy for intermittent exotropia does not appear to cause myopia.

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**OVERCORRECTING** minus lens therapy has been recommended as a treatment for intermittent exotropia.1-3 By fitting myopic spectacles on hyperopic patients, or spectacles that overcorrect the myopia on myopic patients, accommodative convergence will be stimulated. This should reduce the angle or frequency of an exotropic deviation. It is my practice to try overcorrecting minus lens therapy on most patients with intermittent exotropia if they are young enough that the additional accommodation will not be expected to cause symptoms of asthenopia, typically under 7 years of age. I have had the subjective impression that patients undergoing this treatment program appear to become more myopic than other patients. However, because patients with exotropia are more likely to be myopic than esotropic patients, it is possible that this impression is erroneous.3,6

Because excessive accommodation has been implicated as a cause of myopia,7-11 and the wearing of full hyperopic correction by esotropic patients has been suspected of retarding emmetropization,12 it is logical to ask the question “Does overcorrecting minus lens therapy for intermittent exotropia cause myopia?” The purpose of this study was to investigate that question.

**RESULTS**

During the course of this study, overcorrecting minus lens therapy was prescribed...
SUBJECTS AND METHODS

This study was based on a retrospective chart review. Therefore, the patients were treated according to my best clinical judgment as opposed to a rigidly defined treatment protocol. However, to avoid “data snooping” and the formation of a data-generated hypothesis, the specific outcomes evaluated and statistical tests used were chosen according to a protocol that was written before the data were collected.

This series consists of all consecutive patients seen by me in my private practice for the first time between January 1, 1982, and December 31, 1992, with a diagnosis of intermittent exotropia subject to certain exclusions. Patients were excluded if they had any major ocular disease other than intermittent exotropia, severe myopia (greater than −6.00 diopters [D]), severe hyperopia (greater than +5.00 D), age at initial examination younger than 1 year or older than 7 years, or if it was evident at the onset that I would not be able to observe the patient beyond 1 or several examinations.

My normal routine for treating new patients with intermittent exotropia is to begin treatment with 1 to several months of part-time alternate occlusion for antisuppression. This usually decreases the frequency and magnitude of the deviation. The patient is then tested with additional minus lenses during an office examination. If the additional minus lenses seem beneficial in controlling the deviation, spectacles are prescribed that will be equal to the cycloplegic refraction for myopic patients, plus an additional −1.00 D to −2.00 D. For hyperopic patients, spectacles are prescribed that fully correct the cycloplegic refraction plus additional minus power to make the final spherical equivalent between −1.00 and −2.00 D. In addition, the spectacles often incorporate a total of 4 to 6 prism diopters of ground-in base-in prism divided equally between the 2 eyes. This amount of prism will have the same effect as an additional 1 to 2 D of overcorrecting minus power.

This study consisted of 2 treatment groups. The 6-month treatment group included patients who received overcorrecting minus lens therapy for at least 6 months. A second group, the 5-year treatment group, included patients who received overcorrecting minus lens therapy and were still wearing spectacles that provided at least 1 D of overcorrecting minus power 5 years later. Thus, the 5-year treatment group was a subset of the 6-month treatment group. If patients began overcorrecting minus lens therapy and did not show satisfactory control over the exotropia at the first follow-up visit, patching for antisuppression (in addition to the spectacles) was typically resumed. Consequently, all patients who received overcorrecting minus lens therapy received that treatment for at least 6 months before a decision for surgery was made. Although 6 months of excessive accommodation might not be expected to have an effect on the progression of myopia, I chose to analyze patients who received overcorrecting minus lens therapy for that length of time to permit a modified form of an “intention-to-treat” analysis. Many patients treated with overcorrecting minus lens therapy show an initial improvement in their exotropia. Often by 5 years later, they no longer need spectacles with overcorrecting minus power for satisfactory alignment. Alternatively, some show deterioration and need surgery. Studying the 6-month treatment group addresses the effect of attempting overcorrecting minus lens therapy on patients with intermittent exotropia. Studying the 5-year treatment may give a better indication of the actual effect of overcorrecting minus lens therapy on the development of myopia.

A control group consisted of patients who met the same inclusion criteria as the treatment groups except they did not receive overcorrecting minus lens therapy. I decided not to use that treatment modality either because the patient did not show an improvement during the office trial of overcorrecting minus lenses, or because of parental refusal.

All refractions were performed approximately 40 minutes after the instillation of 2% cyclopentolate hydrochloride administered once, or 1% administered twice. I performed all the refractions by means of streak retinoscopy with subjective refinement performed by patients who were old enough to do so.

For this study, refractive errors were recorded as the spherical equivalent of the dominant eye for patients with a fixation preference. For patients with free alternation, the eye with the lower refractive error (spherical equivalent) was chosen. For the rare patient in whom there was absolute symmetry of the refractive error and free alternation, the right eye was arbitrarily chosen.

Age, deviation, and refractive error were compared between each of the 2 study groups and the control group by means of a 2-tailed t test. Because this statistical test assumes independence of the 2 groups, and because the 5-year treatment group is a subset of the 6-month treatment group, the 2 treatment groups were not compared with each other statistically. Such a comparison would be complicated by the lack of independence of those 2 groups and is not essential to this study. The main outcome determination was a comparison of the change in refractive error (myopic shift) in each of the study groups with that in the control group 5 years after treatment was instituted. A secondary outcome was to evaluate the same end points after 10 years.

for 86 patients. Two were eliminated because they did not wear the spectacles satisfactorily, and 10 were eliminated because they were not available for follow-up 5 years later. This left 74 patients (44 female and 30 male) who received overcorrecting minus lens therapy for at least 6 months; they compose the 6-month treatment group. Forty of the 74 patients in the 6-month treatment group were still being followed up by me but were no longer receiving overcorrecting minus lens therapy by the 5-year outcome date. Of these 40 patients, 26 showed a deterioration of their deviation and underwent strabismus surgery.

Fourteen patients improved their control so that they no longer needed overcorrecting minus lens therapy for managing their strabismus. The remaining 34 patients in the 6-month treatment group were still receiving overcorrecting minus lens therapy 5 years later and compose the subset designated as the 5-year treatment group. Twenty-three of the 74 patients in the 6-month treatment group were available for a follow-up evaluation 10 years after their initial examination. Sixteen of the 34 patients in the 5-year treatment group were available for a follow-up evaluation 10 years after their initial examination.
The mean amount of overcorrecting minus lens power used (difference between the cycloplegic refraction and the spectacles prescribed) in the 6-month treatment group was $-2.20 \pm 0.90$ D (range, $-6.00$ to $-1.00$ D). The only patients in whom the amount of overcorrecting minus lens power exceeded $-2.00$ D were patients who were hyperopic. For those patients, the actual lens power prescribed was typically between $-1.00$ and $-2.00$ D. The mean length of time patients in the 6-month treatment group wore overcorrecting minus lenses was $35.5 \pm 34.2$ months (range, 6 to 156 months).

Sixty-two patients were not treated with overcorrecting minus lenses, of whom 17 were unavailable for follow-up by 5 years. This left 45 patients (28 female and 17 male) who composed the control group. Of them, 23 did not receive overcorrecting minus lens therapy because it did not appear beneficial during the office trial, and 14 had sufficient control over their deviation that no therapy appeared necessary. For 8 patients the parents were opposed to trying overcorrecting minus lens therapy, although it was offered. Twenty-one of the 62 patients in the control group were available for a follow-up evaluation 10 years after their initial presentation.

The Table presents data regarding the 6-month treatment group, the 5-year treatment group, and the control group. Although the patients in the control group were slightly older at initial examination than both treatment groups and had slightly larger angles of exotropia at the first examination, the differences were clinically unimportant and not statistically significant ($t$ test). The refractive errors in each of the groups were essentially identical at the initial examination. At the 5-year and 10-year outcome dates, the mean changes in refractive error in the control group and each of the treatment groups showed a similar myopic shift. The small differences in myopic shift between each of the 3 groups were not significant ($t$ test) and seem clinically unimportant.

Because many large studies of refractive errors in children all show that the shift toward myopia is much slower in hyperopic children than in myopic children, hyperopic and myopic patients were also analyzed separately. Eight of the 17 control group patients and 3 of the 10 study group patients who were unavailable for follow-up by the 5-year outcome date were initially hyperopic. Between the initial examination and the last refraction performed, there was a mean shift of $-0.14$ D per year in the hyperopic patients in the control group who were unavailable for follow-up and $-0.16$ D per year in the hyperopic patients in the study group who were unavailable for follow-up. Nine of the 17 control group patients and 7 of the 10 study group patients who were unavailable for follow-up by the 5-year outcome date were initially myopic. Between the initial examination and the last refraction performed, there was a mean shift of $-0.41$ D per year in the myopic patients in the control group who were unavailable for follow-up and $-0.47$ D per year in the myopic patients in the study group who were unavailable for follow-up. These numbers, although too small to permit meaningful statistical analysis, appear essentially identical. They are also similar to normal values reported for children in this age range.

These data suggest that overcorrecting minus lens therapy as a treatment for intermittent exotropia does not cause...
myopia, at least in the manner used in this series. My approach is to use overcorrecting minus lenses by an amount that is somewhat smaller than the amount that has been recommended by others. Possibly because I precede overcorrecting minus lens therapy with a course of antisupression patching, which tends to decrease the angle of strabismus and improves control, my patients were able to achieve adequate control with less overcorrecting minus lens power than has been used by others. Also, because 4 to 6 prism diopeters of base-in prism is typically incorporated in the spectacles, less overcorrecting minus lens power is needed.

My subjective impression before this study that overcorrecting minus lens therapy causes myopia appears false. Caltreider and Jampolsky suggested that overcorrecting minus lens therapy did not cause myopia and suggested that patients with exotropia may be more myopic than healthy children of similar age. Accurate determination of this is difficult because of the nature of studies reporting refractive errors in healthy children. Because many healthy children do not undergo complete ophthalmic evaluation, there is an inherent selection bias. Nevertheless, several large studies have suggested that the normal refractive error of children between 1 and 7 years of age is in the range of +1.20 to +1.67 D. This is substantially different than the mean refractive error of 0.00 ± 1.50 D for the patients in this series with intermittent exotropia. It does appear clear that patients with intermittent exotropia are more likely to be myopic than healthy children of similar age. Also, it has been shown in several studies that the shift toward myopia with age is substantially greater in children who are myopic than in children who are hyperopic. Although there is some variability between these studies, in general they show that myopic children show an increase in myopia of approximately −0.50 D per year, and hyperopic children lose approximately +0.15 D of hyperopia per year between 6 and 15 years of age. These factors probably gave rise to my erroneous subjective impression that overcorrecting minus lens therapy may cause myopia. Caltreider and Jampolsky also speculated that perhaps they were more likely to use overcorrecting minus lens therapy in patients who were already myopic than in patients who were hyperopic, because myopic patients were already wearing spectacles. This was not the case in my series. The mean refractive errors at initial examination in the treatment and control groups were essentially identical.

Repka and coworkers found that patients with hyperopia and accommodative esotropia who were treated with spectacles incorporating their full cycloplegic correction tended to lose hyperopia at a slower rate than children described in normative data. They believed their data suggested, but did not prove, that the wearing of hyperopic spectacles might retard emmetropization because they decrease the demand for accommodation. It is noteworthy that the mean amount of hyperopia in their patients, all of whom had accommodative esotropia, was substantially greater than the mean amount of hyperopia in the healthy subjects with whom they were compared. Because the loss of hyperopia is inversely related to the initial amount of hyperopia, another explanation is possible. Perhaps their patients showed a slow loss of hyperopia because of the magnitude of their initial refractive error, rather than the fact that they were wearing their full hyperopic correction.

Interestingly, children who start out hyperopic, and become myopic later, show a small shift per year toward myopia while they are hyperopic, but have a more rapid myopic shift per year once they become myopic. This has given rise to the seemingly paradoxical hypothesis that accommodation possibly prevents myopia, because accommodation is occurring to a greater degree in hyperopic children than myopic children. This hypothesis was tested by Goss, who compared overcorrecting minus lenses in myopic children to prevent the progression of myopia. He found essentially no difference between the treatment group and the control.

The efficacy of overcorrecting minus lens therapy for treating intermittent exotropia has been described by others. Although it is outside the scope of this study to formally study the benefits of that treatment, the results do deserve some comment. Of the patients in whom overcorrecting minus lens therapy was instituted, 48 (77%) of 62 ended up with good control over their deviation without needing surgery by 5 years later. Of them, 14 (23%) were no longer in need of overcorrecting minus lens therapy; however, 6 of the 14 still required some base-in prism in their glasses. Based on the size and control of the deviation they manifested when first examined, I believe many would have needed surgery if they had not received overcorrecting minus lens therapy. Of those who did ultimately need surgery, overcorrecting minus lens therapy allowed surgery to be substantially deferred. This has the theoretical advantage of permitting patients a longer time during which they are not actually suppressing. It also permits very young children to mature so that measurements can be obtained more accurately. Also, the likelihood of switching from bifoveal fusion to a monofixation esotropia is lessened if the patient is older, if initially overcorrected after surgery.

In theory, overcorrecting minus lens therapy may predispose patients to persist in manifesting an esotropia after surgery for exotropia, because they have been habituated to exercising excessive accommodation. No such trend was seen in this series. The percentage of persistent overcorrections was essentially identical in each group.

This study needs to be viewed in light of some important limitations. Because treatment was not randomized, there is legitimate concern about selection bias. Patients who received overcorrecting minus lens therapy differed from the control group in that they showed benefit with overcorrecting minus lenses during an office trial; the control group did not (with the exception of the patients who declined treatment). Consequently, the treatment group patients were slightly younger at initial examination, had somewhat smaller deviations, and had better control. As can be seen in the Table, these differences were relatively small and were not statistically significant (t test). More
importantly, there is no obvious reason to believe that having a less severe exotropia should have any effect on the progression toward myopia. Similarly, a higher percentage of the control group patients underwent surgery than patients in the treatment group. But there is no obvious reason why strabismus surgery should affect the progression toward myopia. Thus, although these sources of selection bias should have little impact on these data, the differences between the treatment and control groups nevertheless somewhat diminish the certainty of the conclusions of this study.

Finally, a substantial number of patients in each group were unavailable for follow-up by the 5-year outcome date, and substantially more by the 10-year date. Although all patients who are unavailable for follow-up diminish the power of a study, I believe this issue has minimal impact on the conclusions stated herein. A review of the records of the last examination of the patients who were unavailable for follow-up did not disclose any obvious trends with respect to their myopic progression. Ideally, the effect on overcorrecting minus lens therapy on the progression of myopia should be studied in a prospective randomized manner. Such a study would need to be conducted over a period of approximately 10 years. Given the mobility of our society, and the newer restrictions on patient choice as a result of managed care, it is unlikely that such a study could ever be conducted without a substantial number of patients being unavailable for follow-up. Also, although the effect of overcorrecting minus lens therapy is an important clinical issue, it is probably not sufficiently important to justify the cost of such a study. I believe a nonrandomized retrospective study such as this one is a reasonable way of addressing this issue, if viewed in light of its limitations.

It appears that overcorrecting minus lens therapy, possibly combined with patching for antisuppression and base-in prisms, is a useful tool in treating patients with intermittent exotropia. It may delay the need for surgery, and possibly in some cases prevent it. At least in the manner used in this series, overcorrecting minus lens therapy does not appear to be an important cause of myopia.

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REFERENCES