A CASE FOR VERTICAL VERGENCE THERAPY

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ABSTRACT

Vertical vergence therapy as an option in treating vertical diplopia is not normally a first-choice treatment option. A 68-year-old patient that presented with vertical diplopia caused by a right superior oblique palsy has been successfully using vertical vergence therapy to decrease her frequency of diplopia. This paper examines some reasons that vertical vergence therapy should be used as an option in treating symptoms of vertical diplopia. Also included in the report is a review of possible etiologies of superior oblique palsy.

KEY WORDS

vertical vision training, vertical vergences, superior oblique palsy

INTRODUCTION

The occurrence of binocular diplopia following retinal detachment surgery is well known, but is less widely recognized following cataract surgery, according to Hamed et. al.[1] Five of the 38 patients they studied (13%) were diagnosed with a superior oblique palsy following cataract surgery. Several sources say that superior oblique palsy is the most commonly encountered acquired extraocular muscle palsy.[2,3] Most practitioners would not treat the symptoms of a palsy such as this with orthoptics, as they would agree with Parks that usually vertical vergence amplitude training does not produce improvement.[4] However, Petito recently showed that in studying forced vertical vergence fixation disparity curves, the presence of a flat portion in the curve may indicate that similar mechanisms as those shaping horizontal disparity curves are active in vertical vergences. He elicited this sigmoid-shaped vertical fixation disparity curve using small prism increments of 0.5^{Δ} BU and BD alternately. The variation in slope across the vertical curve was found to be very similar to the change of slope found across horizontal fixation disparity curves, which is attributed to the interplay between fast and slow fusional vergence mechanisms. Petito suggests that testing only a small number of points on the vertical fixation disparity curve does not give an accurate picture of the patient's vergence mechanisms.

This paper will examine some recent findings concerning the vertical vergence system and make a case for having vertical vision therapy as an option in treating symptoms of vertical diplopia. We will also review etiologies of superior oblique palsy.

CASE REPORT

A 68-year-old white female was referred to the Ferris State College of Optometry for evaluation of her vertical diplopia. The patient complained of vertical diplopia at distance in primary gaze, first noticed after a cataract extraction in her right eye in March, five months previously. Her habitual Rx was O.D. +0.25-2.00 x 107, O.S. +2.00 sphere with a +2.50 add O.U. Distance acuity was 20/20 O.D. and 20/25 O.S. A cover test showed a 6 right hypertropia at distance. The Hess-Lancaster and Parks procedures indicated a right superior oblique paresis with a well-established spread of comitance. Internal and external exams were unremarkable. The right eye was pseudophakic with a centered intraocular lens prosthesis.

At the strabismus evaluation in September, her deviation was 8^{Δ} right hypertropia. Her range of fusion on the synoptophore was 1.5 to 12^{Δ} BD O.D. 4^{Δ} BD before her right eye allowed her to fuse objects across the room. With such a small amount of prism necessary to fuse, we decided to try vision therapy for one week to increase and strengthen her range of vertical fusion before considering a spectacle prism correction. Prism insertion and removal beginning with 8^{Δ} BD O.D. and vertical vectographs were prescribed to increase vertical fusion ranges and train the disparity detectors.

At the one week follow-up visit, her right hypertropia had increased to 12^{Δ}. She was having trouble keeping objects fused through 8^{Δ} BD O.D. Her exercises were indeed helping, but the diplopia encountered throughout the day undid any training because suppression mechanisms were being learned. After refraction we ordered a new Rx with a cosmetically acceptable amount of prism and planned to use press-on Fresnel prisms in the distance portion only to obtain single binocular vision while doing vision therapy. The ultimate goal is no Fresnel prism.

The new Rx was O.D. +0.50-2.50 x 115, O.S. +2.00-0.75 x 050, add power +2.50, with resultant acuities 20/20 O.D. and O.S. at both distance and near. When the spectacles were dispensed, her hypertropia had again increased leading to the necessity of an additional 6^{Δ} BD O.D. with press-on Fresnel, in the distance portion only. The patient was instructed to continue her therapy at least three times a day, although she claimed to do it more often than that.

Her last follow-up visit before her winter in Florida occurred in early November. Therapy was going well and the patient was able to remain fused through 2^{Δ} BU O.D. over her Rx and Fresnel. The 6^{Δ} BD Fresnel was replaced with a 4^{Δ} BD Fresnel with resultant single binocular vision. She was also given a 2^{Δ} BD Fresnel to use in 2-3 months after continuing therapy. She will be seen again at our clinic in the spring upon her return from Florida. Patient's motivation is high and by April it is expected that she will be using 2^{Δ} BD Fresnel or possibly no Fresnel prism at all.

DISCUSSION

The most common cause of cranial nerve IV paresis is undetermined (36%), followed by head trauma (32%), vascular, such as in diabetes and hypertension (18.6%), neoplasm (4%) and aneurysm (1.7%), according to a study by Rush and Younge.[6] Other possible causes include encephalitis, multiple sclerosis, herpes zoster, and iatrogenic etiologies. Rush and Younge's study also found that the fourth nerve is slightly more likely to recover compared with the sixth and third nerves.[6] In Nemet's sudy, all thirteen cases of superior oblique palsy with an undetermined etiology spontaneously resolved within ten weeks. He points out that intracranial space-occupying masses are extremely rare in superior oblique palsy and when present are associated with other neurological signs. This rarity can be explained by the intracranial protection of the fourth nerve's pathway by the touch margin of the tentorium, protecting from pressure by tumors or aneurysms.[2]

To expand upon the iatrogenic causes, in the study by Hamed et. al., they give two possible reasons for the superior oblique palsies that were diagnosed following cataract extraction. The palsy may either be "new" and was not diagnosed before surgery because a dense cataract prevented diplopia, or an old palsy may have been compensated for by supranormal vertical fusional reserves. A prolonged asymmetry of input because of a monocular cataract may bring about a reduction in fusional reserves. Since ocular motility and alignment is not routinely assessed in the cataract patient's pre-operative exam, a small deviation may not be detected before cataract surgery.[1]

Parks and Mitchell suggest that the usual treatment plan of acquired fourth nerve palsy during the first six months consists of waiting to determine the degree of spontaneous recovery.[7] Ellis and Helveston point out that many of the patients with an undetermined etiology of acquired palsy received extensive neurological workups, yet those neurological studies were "invariably" nonproductive if the symptoms had been present for four to six months without associated history of trauma or another disease process.[3]

Since our patient came to us four months after she first noticed diplopia, we were quite sure it would no longer resolve spontaneously.No neurological workup was ordered since no history of trauma or disease or neurological signs were present. Her vertical vergence ranges were large (1.5 -12 BD O.D.) at initial testing which Schor and Ciuffreda suggest help to differentiate a pre-existing disorder from a recently acquired neurological disorder.[8] They also claim that any individual with a congenital or longstanding hyperdeviation who is capable of intermittent fusion has by definition a large vertical fusion range. Our patient seems to have been able to fuse intermittently because of the vertical anisometropia created in her spectacles by the minus cylinder near axis 090 in the right eye, creating a base down effect when looking down to read. She claimed she never saw diplopically at near, indicating that the extra 2^{Δ} base down effect O.D. was sufficient to aid her fusional reserves at near and thus prevent diplopia.

Only very recently has there been considerable investigation into the effects of vertical vergence training. Normal vertical fusional amplitude is 3^{Δ} to 6^{Δ}, although Metz claimed that one patient in a military hospital had built up 30^{Δ} of vertical fusional vergence by working several hours daily for many months on a major amblyoscope.[9] Balliet and Nakayama have shown it is possible to increase a patient's voluntary cyclotorsional capabilities up to 30

degrees.[10] Crone claimed that a vertical divergence as large as 9$^{\Delta}$ can be developed by orthoptics.[11] Robertson and Kuhn studied the effect of vertical vergence training given to three patients with a vertical deviation, two patients having a paretic deviation. These patients significantly increased their vertical fusional amplitudes.[12] And finally, to cite yet another support for vertical vergence training, Hiatt evaluated 25 patients with early onset paretic vertical deviations and found that their vertical fusional amplitudes were in the range of 15 prism diopters.[13] These last two sources make specific mention of paretic vertical deviation. Concerning this, Duke-Elder and Wybar report that vertical vergence training can be successful in paretic casses that are mildly incomitant and in the stage of almost complete recovery.[12] However, these stringent qualifications need not be added to the statement that vertical vergences can be improved by orthoptics.

There seems to be a flexibility among patients in altering vertical vergences by training, wherein some patients are quite capable of increasing their vertical vergences and others seem not to be. Robertson and Kuhn found that three of six symptomatic patients exhibited an increase in their vertical vergences after training, but that subjects with no vertical heterophoria did not increase their vertical vergences with training. They claimed that their study implied vertical vergence training is effective in extending the fusional range in only one direction.[12] Rutstein et. al. claimed that the results of their study indicated the vertical fusional amplitudes were not altered at a clinically valuable level by vertical vergence therapy, but they did admit that in contrast to their findings, there appeared to be patients whose vertical amplitudes could be increased by orthoptics.[15] Balliet and Nakayama's remarkable study on training voluntary cycloversions demonstrated that the oculomotor system has more plasticity than generally assumed.[10]

Some specifics of training include an adequate sensory feedback loop, an important factor in Balliet and Nakayama's training procedure, and a highly motivated patient. Other sources suggest that it's better to train at slower rates where vertical vergences are more effective,[16] to use short, frequent training sessions (shown more effective in training positive fusional vergences),[17] and that stepwise phasic training is more effective than smooth tonic activities, but that these tonic activities should not be altogether excluded to train only with phasic techniques.[18]

A way to mnitor the improvement in the binocular field of view with diplopia was suggested by Soden and Cohen.[19] The patient was given red-green glasses and instructed to follow a moving target on an arc perimeter. There are three possible responses. If luster is seen, fusion is indicated. Diplopia is indicated if two lights are seen (one red, one green). If either a red or a green light is seen, then either the patient is suppressing or the light is beyond the patient's binocular field of vision. An arc perimeter chart is used to record the patient responses. Using this monitoring technique, Soden and Cohen showed that vision therapy on a patient with a right superior oblique palsy increased his 10-15 field of fusion followed by a 5-10 field of diplopia into a 35-40 field of fusion followed by a 5-8 field of diplopia. Since our patient did not complain of constant diplopia, this may have been a useful technique to monitor her progress during therapy.

The prescribed therapy for our patient seems to fulfill the recommended requirements in that she is using both phasic (prism insertion and removal) and tonic (vectographic) training techniques, and in short, frequent intervals during the day. Gradually her Fresnel prism "crutch" is being reduced to increase her adaptation ability, as recommended by Sethi and North.[20] With hindsight that is, as usual, 20/20, one other test that would have been helpful initially and throughout training is a vertical fixation disparity curve. This also would help monitor progress, since vertical vergence therapy flattens the vertical fixation disparity curve which increases the subject's ability to adapt to vertical prism.

To conclude, I would agree with the suggestion of Rutstein et. al. that a study would be beneficial that investigates the effect of vertical vergence training on patients having a vertical deviation associated with a neurogenic or myogenic cause.[15] Since these types of

patients especially seem to have supranormal vertical fusional reserves and a more plastic oculomotor system, they may prove to be very successful with vertical vision therapy. More data on individual successes or failures using carefully planned vertical vergence therapy is definitely needed before this type of therapy can be unequivocally accepted or rejected.

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