Article

A Developmental Approach to Congenital Ocular Motor Apraxia: case report and literature review

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Abstract

Introduction: Congenital ocular motor apraxia is a deficiency in horizontal saccades with normal vertical saccades and pursuits (vertical and horizontal). It is not usually diagnosed until the age of 4-6 months when patients develop head thrusting and/or a synkinetic blink. The structural defect in C-OMA is not known, but the vermis in the cerebellum is the most likely site of the neurological defect. Improvement with age usually, but not always, occurs. Various neurological manifestations have been reported in association with congenital ocular motor apraxia. These children will have difficulty with both fine motor and gross motor activities. No definitive treatment has been documented in the literature.

Case Report: A 5-year old male with a history of congenital ocular motor apraxia presented for a full examination with complaints of head turning and head tilting. He was having difficulty in school and avoided near activities. He was diagnosed with a gross motor delay one and a half years earlier. Upon testing, the patient was found to be a latent hyperope with poor horizontal pursuits and saccades. Prescription glasses were prescribed to compensate for the hyperopia. A perceptual examination was performed, which showed numerous delays. A program of vision therapy was initiated to improve perceptual difficulties and ocular motility.

Conclusion: C-OMA is a disease that must be managed by optometrists. Even though no documented treatment has been found, vision therapy should be implemented to enhance basic skills and improve ocular motility.

Key Words

ocular motility, saccade, apraxia, C-OMA

Introduction

In 1952, Cogan described a series of four children that displayed difficulty voluntarily generating horizontal saccades. Vertical saccades were not affected and pursuits were full and smooth in all directions. Patients developed head thrusting and lid blinking as compensatory mechanisms. He termed this condition congenital ocular motor apraxia (C-OMA).[1-8]

Background

Although it may be observed earlier, C-OMA is not usually recognized until after the age of 4-6 months. The pathological state is present throughout the first months of life, but at this age, adequate head control develops, and compensatory mechanisms are adopted.

While most cases occur sporadically, reports in siblings,[9] consanguineous unions and in successive generations have been documented. This suggests the possibility of an autosomal dominant or recessive inheritance pattern. C-OMA is found more frequently in males versus females, indicating a possible X-linked recessive transmission.[10]

Correspondence regarding this article can be e-mailed to Taub@nova.edu or sent to Dr. Marc Taub Nova Southeastern University, Health Professions Division, 3200 S. University Drive, Fort Lauderdale, FL 33328 All statements are the authors' personal opinion and may not reflect the opinions of any particular institution or organization.

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The structural defect in C-OMA is not known. Agenesis or hypoplasia [5,11] of the corpus collosum and malformations of the cerebellum,[5] and cerebrum[9] have been associated. Several other reported abnormalities have been documented in patients with C-OMA. (Table 1.)

Recent studies support the theory that the vermis in the cerebellum is the most likely site of the neurological defect. The vermis is involved with the generation of rapid eye movements, although the exact pathway is unknown. The vermis of the cerebellum forms from the fusion of the hemispheres, which begins superiorly around the 9th gestational week. This progresses inferiorly and is completed by the 15th gestational week.[15] Insult to the brain that occurs during this time span can result in any number of malformations. An insult that takes place after complete fusion of the hemispheres will lead to a destructive lesion in the vermis.[8]

In a study on congenital ocular motor apraxia, Shawkat et. al, reported that 38 out of 62 (61%) patients had abnormal Magnetic Resonance Imaging (MRI) studies. Taking a closer look at the specific defects, they discovered that 45% and 39% had cerebellar and brainstem abnormalities respectively.[2] Sargent et. al found that in a series of 19 subjects, 14 had abnormal findings when investigated with imaging studies. Ten out of the 14 had a distinctive finding of an abnormally small vermis. This was detected on axial CT and MR studies.[16]

Unless imaging studies are directed to specifically look in the cerebellar and brainstem region for this type of abnormality, it can easily be overlooked. It is recommended that a high-resolution sagittal fast-spin echo T_2 and a thin section volumetric coronal T_1-weighted MRI be performed versus a CT scan. This will enable more exact delineation of the anatomy of the vermian lobules.[8]

In contrast to congenital ocular motor apraxia, a variety of acquired causes have been reported. In acquired OMA, both horizontal and vertical eye movements are abnormal. Head thrusting, a major component in congenital cases is much less prominent. Whereas congenital OMA tends to improve with age, acquired OMA rarely improves. Acquired OMA has been associated with lesions in the brain and metabolic diseases. (Table 2.)

C-OMA is often unrecognized and misdiagnosed. Even after testing has been completed, these cases have been labeled as "a habit," autism, spasmodic torticollis, behavior problems, cerebral palsy, myoclonic or atypical petit mal epilepsy, dystonia, psychomotor retardation,[10] cortical blindness and Leber's congenital amaurosis.[11]

Table 1.
C-OMA associated with congenital malformations

Agenesis/hypoplasia of the Corpus Callosum [2, 5, 11,18]
Cerebellar Hypoplasia/Aplasia of the Cerebellar Vermis[2,8,15,16]
Immature development of the putamen[13]
Heteropia of grey matter[13]
Porencephalic cyst[12]
Hamartoma near the foramen of Monroe with dilated lateral ventricle[12]
Macrocephaly[12,14]
Cystic lesion of the posterior fossa with hydrocephalus[10]
Chodrodystrophic dwarfism[10]
Hydrocephalus[10]
Encephalocele[10]

Table 2.
Causes of acquired ocular motor apraxia

Acquired Immune Deficiency Syndrome (AIDS) [28]
Bilateral frontal and parietal lesions [17,18]
Huntington's chorea [11]
Niemann-Pick disease [11]
Gaucher's disease [11]
Balint's syndrome [11, 17]
Progressive Supranuclear Palsy [11]
Multiple Sclerosis [11]
Wilson's disease [11]
Central Nervous System Neoplasms, Infarctions [11]
Joubert syndrome [19]
Cerebellar medulloblastoma [20]
Dilated fourth ventricle with hypoplastic vermis [21]
Midline lipoma displacing the vermis [22]
Ataxia telengiectasia [13, 23-25]
Olivopontocerebellar degeneration [23, 26, 27]
Spinocerebellar degeneration [11]

Due to confusion in diagnosis, inappropriate and unnecessary drug therapies may be employed.

A characteristic head thrust is adopted by the patient, in order to generate a motor response to break what is described as a "spasm of fixation."[29] When the patient is asked to fixate, the head is turned and vestibular input (vestibular ocular reflex) causes the eyes to contravert in the opposite direction of the turn. It becomes necessary for the head to overshoot, literally dragging the eyes along to the point of fixation. Once fixation is made, the head turns back until the eyes are straight ahead in the primary position.[29-31] (Figure 1.)

Improvement with age usually, but not always occurs. With better motor control, patients can learn to direct their eyes, reducing the need for head thrusting.[12] This finding is not exclusive to C-OMA, but can occur in children with gaze palsies, slow saccades, visual field defects and poor eccentric gaze holding.[7] If a child is hypotonic or has poor head control, head thrusting may appear later in life or may be absent.

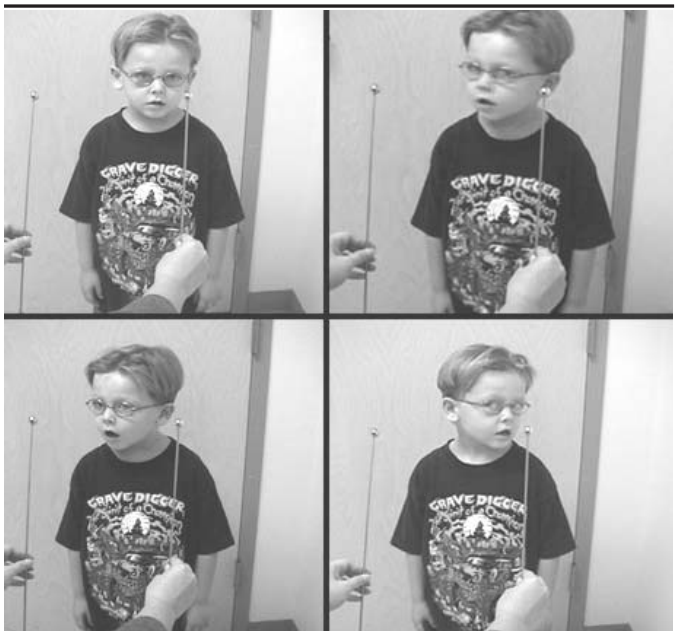

Figure 1. A) Top left-The patient is fixating on the right (gold) wand. B) Top right-He attempts to saccade to the left (silver) wand. The head thrusts to the left, but the eyes remain fixated on the right wand. C) Bottom left-The patient is fixates on the left (silver) wand. D) Bottom right-He attempts to saccade to the right (gold) wand. The head thrusts to the right, but the eyes remain fixated on the left wand.

A synkinetic blink is another compensatory behavior that is found in older patients or younger patients when the head is restrained. Due to the time and energy involved in head thrusting, blinking can be adopted. Two possible explanations for the blinking are put forth in the literature. One theory proposes that blinking breaks fixation, thus allowing the saccade to initiate.[2] Another possibility is that the blink lowers the gain of the vestibulo-ocular reflex, reducing the amount of head thrusting required.[31]

The historical definition of congenital ocular motor apraxia indicates that saccades will be deficient only in the horizontal plane, but the vertical plane cannot be disregarded in these cases. Several reports of vertical ocular motor apraxia have been published.[7,11,14,32,33] One commonality among these cases is the indication that pre- or peri-natal insult was a contributing factor.[11] The etiology for acquired OMA must be differentiated between degenerative and metabolic disorders (e.g. neuro-visceral lipidosis) and stable disorders (e.g. birth injury, perinatal hypoxia and Leber's congenital amaurosis).[30]

Pursuit movements when tested may also appear abnormal. This differs from the original definition that C-OMA only involves voluntary saccadic eye movements. Shawkat et al. in a review of 62 cases of C-OMA showed a significant difference in pursuit eye movements between diagnostic (those that had an associated abnormality on a brain scan) and idiopathic groups (those which did not show an abnormal brain scan). The idiopathic group, had essentially normal smooth pursuits in contrast to diagnostic groups.[2]

A simple in office method available to confirm a diagnosis of C-OMA is made by spinning the infant around the examiner. Due to impaired saccades, the eyes move in the direction of the spin, but a corrective fast horizontal saccade will not occur.[30]

Various neurological manifestations have been reported in association with C-OMA such as developmental delay, poor coordination and gait abnormalities.[10] Most affected children are slow in attaining early developmental milestones and were later classified as clumsy. This association was first documented by Cogan in 1966. He stated, "The general development has nearly always been slow and the patients are typically characterized as 'clumsy', 'awkward', and prone to 'stumbling'. The probability is of course that many symptoms were due to the ocular motor defects."[34]

These children will have difficulty with both fine motor and gross motor activities. They will do poorly in sports, tend to misjudge openings such as doors and frequently fall. Games and activities that a "normal" child would have no difficulties participating in, especially those that involve climbing and balancing, may be considered dangerous for a child with congenital ocular motor apraxia.[3]

Rappaport et al. demonstrated the concurrence of ocular motor and oral motor deficiencies. Both defect types were measured in 80% (8 of 10) of his study cases. In contrast to ocular motor signs that tend to subside with age, speech production problems continued to manifest and affect many children with C-OMA later in life.[34]

Case Report

A 5 year,10 month old young Caucasian male presented for a comprehensive vision exam. The chief complaints included head tilting or turning when "looking at anything," avoidance of near work, and becoming easily frustrated and giving up too easily. His mother stated that he absolutely hates to read, write, or color. Her goal was to help him with his writing, eye/hand coordination, and to assess his visual skills so that he could achieve his best potential reading abilities.

The patient was diagnosed with ocular motor apraxia at 7 weeks old. His birth weight was 7lbs, 13 oz and his peri-natal history was unremarkable. The patient first sat-up without support at 8 months, crawled at 10 months, walked at 18 months, and spoke his first words at 7 months. His developmental history was normal until age 4. At that time, he was diagnosed with a gross motor delay by a pediatric neurologist. He had a history of

Table 3.
Examination Findings

Uncorrected VA's: 20/50 OD/OS, 20/40 OU
Habitual RX
OD: +3.00 sph
OS: +2.75 sph
Corrected VA's
Dist: 20/20^{-} OD, OS
Near: 20/40 OD, 20/32 OS
Cover Test
Unilateral: Orthophoria (dist & near)
Alternating: Orthophoria (dist & near)
NPC: 8cm/10cm (break/recover)
Amplitudes of Accommodation: 16.5D OD, OS
Color: 13/14 OD, 12/14 OS
Stereo: Global 250 arc seconds, Local 40 arc seconds
Confrontation Fields: Full to finger count
Pupils: Equal, round, reactive to light
EOMs: FROM, jerkiness and fixation loss
Dry Retinoscopy
OD: +6.50-0.25 X 180
OS: +5.50
Cycloplegic Retinoscopy
OD: +6.50-0.50 X 180
OS: +6.50-0.50 X 180
Vergences
Dist: BO(X/10/8), BI(4/14/6)
Near: BO(6/30/18), BI(X/25/16)
MEM: +1.00 OD/OS (over habitual)

chronic ear infections starting at 19 months old and bilateral hip dysplasia which required him to wear a harness for 8 weeks. Hearing tests, revealed no deficits. He received 10 weeks of physical therapy in 2003 to help him with his gross-motor coordination, but occupational therapy has not been attempted. His mother reported that he is allergic to some dairy products, soy, and peanuts with no known allergies to medications. Currently, he is taking Claritin for seasonal allergies.

The patient's last eye exam was a year ago and he was prescribed a full-time prescription to compensate for latent hyperopia. With his habitual prescription of +3.00 DS OD and +2.75DS OS, his corrected entering distance visual acuities were 20/20- OD, OS, OU and his corrected near visual acuities were 20/40 OD and OS and 20/32 OU. The uncorrected distance visual acuity was 20/50 OD, OS and 20/40 OU. The entire examination is summarized in Table 3. The anterior and posterior segment ocular health was unremarkable.

He was given a new bifocal prescription of +3.00D DS OU with a +1.50D Add. The Add was issued to facilitate plus acceptance since the patient would not tolerate higher levels of the hyperopic prescription. At a 6 week follow-up, he reported that he could see better without his glasses, but was compliant with wearing them full time. His distance visual acuity with the new prescription was $20/40^{-2}$ OD, OS and $20/30^{-2}$ OU and his near visual acuity was 20/25^{-} OD, OS and 20/20^{-}OU. His NPC was 6 cm break/ 8 cm recovery and there was no improvement in stereopsis, which measured 40 arc seconds with Wirt Circles. MEM with his new prescription revealed a lag of +0.75 D OD, OS. A single vision eyeglass prescription of +4.50 DS OU, which was the maximum plus accepted, was given for full time wear. A bifocal was not used in these lenses due to his eye movements and the more permanent nature of this prescription. The patient was then scheduled for a visual perceptual/visual information processing evaluation. The visual perceptual results are displayed in Table 4.

Smooth pursuit and saccadic eye movements were tested with the NSUCO eye movement battery and were very poor for his age. He had numerous fixation losses, excessive head and body movement when attempting to track a slow moving target. He also had excessive fixation losses when looking back and forth between two targets with a head jerk and a loss of balance during this task. The patient's ability to fixate was adequate for his age. (Figure 1.)

Optokinetic nystagmus (OKN) was tested monocularly and binocularly in both the horizontal and vertical directions. OKN was absent during all testing in the horizontal direction. Upon vertical testing, movement patterns were erratic. While each direction (drum rotating up and down) was tested several times, OKN was both absent and present, often appearing and disappearing during one "run." While the patient does have wandering attention, this was not the case during the OKN evaluation. OKN testing cemented the deficiency of both horizontal and vertical eye movements.

Visual-Spatial Skills include the following: body awareness, bilateral integration (ability to use both the right and left side of the body in coordination), laterality (ability to know right and left on one's own body), and directionality (awareness of one's body in relation to objects in space, on the chalkboard or on paper). These skills were probed with tests such as Jordan Left Right Reversal, Piaget Right Left Awareness Test, and Gardner Reversal Frequency Tests. Standing Angels tests for body knowledge and control, body awareness, body image (an understanding of one's body and its individual parts), and body schema (understanding how the body works in relation to itself). The patient's visual analysis skills were tested using the Visual Memory, Visual Sequential Memory, Visual Discrimination, Visual Figure Ground, Visual Closure and Visual Form Constancy subtests of the Test of Visual Perceptual Skills (TVPS).

His ability to integrate different sensory and perceptual modalities was tested by the Beery Test of

Table 4.
Perceptual Examination Findings

	TEST	CLASSIFICATION	RAW SCORE	PERCENTILE/ AGE EQUIVALENT	INTERPRETATION
Visual Spatial Skills	Standing Angels	Responsive motions to touched body parts -gross motor/ body awareness/bilateral integration	Stage 3	5 yrs old	Weak Unable to complete Contralateral Movements Excessive Gross Motor Overflow
	Piaget Right-Left Awareness Test	Right-Left Awareness Test	A	5yrs-6yrs	Average
	Gardner Reversal Frequency-Execution	Requires child to write numbers & letters as called out in random order	Unable to Complete	<1%	Very Weak
	Gardner Reversal Frequency-Recognition	Requires child to recognize letters & numbers written backwards/reversed Laterality/directionality	Unable to Complete		Very Weak
	Gardner Reversal Frequency-Matching	Requires child to match a given symbol to the identical symbol/visual discrimination	10 errors	8%	Very Weak
	Jordan Left-Right Reversal Test	Part I- requires child to recognize printed letter reversals	Unable to Complete due to poor letter and number knowledge		Very Weak
Visual Analysis Skills	TVPS VD	Visual Discrimination	2	<1% <4 yrs-0mos	Very Weak
	TVPS VC	Visual Closure	7	53% 5 yrs-11 mos	Average
	TVPS VFC	Visual Form Constancy	2	<1% <4yrs-0 mos	Very Weak
	TVPS VM	Visual Memory	2	1% < 4yrs-0mos	Very Weak
	TVPS VSM	Visual Sequential Memory	8	66% 6 yrs-6mos	Above Average
Oculo-motility	DEM (all parts)	Developmental Eye Movement Test -saccadic eye tracking for reading and automaticity of number calling	Horz-Not scoreable Vert-Not scoreable Ratio-Not unscoreable		Oculomotility dysfunction Poor Automaticity of number calling
	NSUCO Eye Movement Test	Northeastern University College of Optometry Eye Movement Test -Evaluates Quality of Pursuits and Saccades by comparing accuracy, ability, head movement and body movement	Pursuits OD OS OU Saccades OD OS OU	Ability/ Accuracy/ Head/Body 4/1/1/1 4/1/1/1 4/1/1/1 4/1/1/1 4/1/1/1 4/1/1/1	Excessive head and body movement with pursuit and saccadic activity
Visual-Integration Skills	Beery-VMI	Visual Motor Integration -simultaneous processing -eye-hand coordination	10	14% 4yrs – 10 mos old	Very Weak
	Birch Belmont	Auditory Visual Integration Test - visual recognition of tapped out sound patterns	Unable to Complete	1%	Very Weak
Gross Motor	3 X 3 Alternate Hop	Measures child's balance and coordination	Poor-Gross motor overflow		Very Weak

Visual Motor Integration (VMI) and Six Figure Divided Form Board. The Six Figure Divided Form Board is a three dimensional wooden puzzle that evaluates a child's eye-hand coordination, developmental stage of visual-motor integration, visualization, and visual thinking. It took the patient 8 minutes to complete this puzzle, which is below expected based upon age norms. He would not look at the puzzle pieces directly but would view the pieces with his head turned to the side. He would try to force the pieces in the spaces without trying to visually match the shapes to the spaces. This indicates that he is very motor based on the visual-motor integration hierarchy. He was very attentive to this task and he did not frustrate easily. However, he had a difficult time with problem solving and mental flexibility to try different solutions.

The patient was easily distracted and very inattentive during testing. He had to be told repeatedly to stay on track, focus, and concentrate. He was unable to sit still throughout the examination. The non-dominant hand was used for support to hold the paper down during tests involving writing tasks. The exam was performed at an unusually close working distance. He used an irregular pencil grip when performing written tasks and seemed to be very impulsive with his answers. He was extremely happy, friendly and talkative. He always tried his best and wanted to perform well and frequently looked at the test giver for approval.

Perceptual deficiencies were found in the following areas:

Oculomotor Skills

Visual Spatial Skills-

Poor Body Awareness, Poor Bilateral Integration Laterality, Directionality

Visual Analysis Skills-

Visual Form Constancy, Visual Memory, Visual Discrimination

Visual-Integration Skills-

Visual-Motor Integration, Auditory-Visual Integration, Visual-Verbal Integration

Visualization and Visual Thinking

A vision therapy program was recommended. An occupational therapy, physical therapy, speech-language evaluation with possible remediation and a psycho-educational evaluation were discussed and recommended. Since C-OMA affects the ability to control voluntary saccades, a vision therapy program was developed to enhance steady and accurate fixation, improve accuracy of smooth pursuit eye movements without head and body movement, increase peripheral awareness to reduce the need to scan or make voluntary saccades, and to improve gross motor development and spatial organization.[35] Ocular motility training will consist of a sequence of fixation-pursuit-saccadic therapy, monocular then binocular, with eye-hand reinforcement progressing to eye movements without head and body movement.[36] An outline for the goals of this program and examples of activities detailed in Table 5.

Developmental delays including gross motor delays, the inability to balance on either leg for more than three seconds and poor internal spatial organization may be due to the C-OMA. Improvements in gross motor skills, balance and coordination, and better spatial organization will help reduce head and body movements that occur during fixations, pursuits, and saccades. It is more advantageous for our patient to develop a synkinetic blink, reducing reliance on head-thrusting. Enhancing saccadic ability without head movement will be a main objective of therapy.

Discussion

The initial presentation may be to the pediatrician or eye care provider indicating poor fixation and or a choppy/jerky motility pattern. Common initial complaints include: 'having to move head to move the eyes,' poor vision, 'head and eye movements don't coordinate,' and 'looking at objects in a weird way'.[5] The parents may indicate that the child has learning or reading problems, developmental delays, speech and hearing deficiencies and gross motor dysfunctions.

The lack of coordinated eye movements may lead to a diagnosis of blindness and needs to be further investigated with the use of visual-evoked potential (VEP) testing. In contrast to most assessments of vision that depend upon subjective or objective motor responses (e.g. Teller Preferential Looking Cards) from the patient, the VEP is independent of abnormalities of the motor system.[6] Pupil testing, funduscopic evaluation and electroretinography must also be investigated. [37]

An important aspect of testing is the use of the optokinetic nystagmus (OKN) drum. Whether the deficiency is horizontal, vertical or both, an abnormal or absent OKN will be revealed.[2,7,14] OKN evaluation should consist of an assessment of the gain on the slow phases and the objective quantification of the saccade failure on the fast/quick phase during the return sweep. When the quick phase is missed, the eyes remain in a lateral deviation "lock-up" in the direction of the slow phase.[2] When the quick phase spontaneously resumes, it may be accompanied by a synkinetic blink or a head thrust. In a study of 74 children, Harris et al. documented "some degree of locking up" during optokinetic and/or vestibular nystagmus.[7]

Our case demonstrated many aspects of the disease that have been documented in the literature. Like many

Table 5.
Goals and Activities for Vision Therapy

Goal #1 Bilateral Integration

Phase 1: Achieve motor planning by executing isolated, simultaneous and sequential movements of both sides of the body.
Activities: Snow angels, Standing angels, Randolph shuffle, Bi-manual circles, Chalkboard Circles, Flashlight walk, Body rolls, Creeping, Crawling, Walking rope, Walking rail, and Balance board.

Goal #2 Laterality

Phase 1: Achieve the concepts of laterality by orally or motorically identifying his own body's right and left.
Activities: Snow Angels, SUNY Visual Motor Series (hands, feet, and hands and feet), Standing angels, Randolph shuffle, and Chalkboard circles. These will be done by calling out the patient's right and left and also having additions and dropouts of body parts told by the doctor.
Phase 2: Achieve the concept of using his internal reference to guide himself through space.
Activities: Floor Maze Level I (the patient would guide himself through the maze)

Goal #3 Directionality

Phase 1: Achieve the concept of being aware of other objects in three-dimensional space in relationship to himself.
Activities: Floor Maze Level II (the patient will guide the doctor through the maze).
Phase 2: Achieve the concept of the patient to be aware of two-dimensional symbols in space in relationship to himself.
Activities: Kirschner arrows, Kirschner arrows with a Balance Board.

Goal #4 Oculomotility

Phase 1: Fixate on a target monocularly then once they are equal in each eye then proceed same activities binocularly.
Activities: Stick in straw (begin with a tube of about 1 inch in diameter with a marker as the target then as improve\decrease the size of the diameter), AN star fixation , Dive bombing, Flashlight fixations, Dotting O's (starting with bigger O's then down to smaller O's), Stationary Pegboard.
Phase 2: Develop fine pursuits monocularly, once equalized then do binocularly.
Activities: Marsden ball and calling out letters on ball, Michigan tracking, Visual tracing, Mazes, Rotating pegboard, Flashlight tag and Pie tin rotations.
Phase 3: Develop large excursion pursuits monocularly, once equalized then do binocularly. The goal here is to eliminate head movement along with the eye movements.
Activities: Flashlight pursuits, Marsden ball pursuits, thumb pursuits, four corners pursuits.
Phase 4: Developing peripheral awareness.
Activities: Wayne Saccadic Fixator, Lowman board (focusing on a central target while simultaneously asking questions about the periphery), Chalkboard Circles
Phase 5: Develop gross monocular saccades followed by gross binocular saccades.
Activities: Four corner saccades, Flashlight tag, and Wayne saccadic fixator.
Phase 6: Develop fine monocular saccades followed by fine binocular saccades. The goal is to eliminate head and eye movements.
Activities: Hart chart saccades, Saccadic workbooks, Computer visual scan, Computer visual search, Computer directionality, and Pencil saccades with an accommodative target.
Phase 7: Integrate accurate saccades and pursuits with accommodation and vergence.
Activities: Brock string, Vectograms, Aperture rule, and Lifesaver cards.

Goal #5 Visual Integration Skills

Phase 1: Integrate the visual and motor skills by combining visual information processing skills with the motor system to execute complex visual patterns.
Activities: Bean bag toss, Michigan tracking, Mazes, Circle and Square, Rosner dots, Connect the dots, Geoboard, Lite Brite, Beading, X's and O's, Haptic writing, and Ball bunting.
Phase 2: Integrate the visual and auditory skills.
Activities: Kirschner arrows with a metronome, Walking rail (with auditory stimulus), Computer visual tracing (with auditory feedback), and Hart chart with metronome.

Goal # 6 Visual Analysis Skills

Phase 1: Develop Visual Discrimination skill (the ability to distinguish different forms by their size, shape, and orientation).
Activities: Perceptive cards (Visual Discrimination), Parquetry blocks, Geoboard, and same/different.
Phase 2: Develop the skills of Visual Memory by training the short-term memory. First by recalling the spatial characteristics of the forms using only visual information. Secondly, recalling the sequential order of the forms using only visual information. Lastly, recalling the forms as the amount of information is increased and the viewing time is decreased.
Activities: Tachistoscope in Computer, Parquetry Blocks by memory, Geoboard by memory, Beading a color sequence by memory, and Balance Board (calling out a sequence of shifting in different directions).

Adapted from references 38, 40-44

other diseases, C-OMA is a variable condition. Not every case will appear exactly the same. For example, our patient had the characteristic head thrust, but lacked the synkinetic blink reflex. This may change as the child ages. As mentioned earlier, historically the condition involves only horizontal saccades. The patient we examined demonstrated a deficiency with both horizontal and vertical eye movements. While this has been documented in the literature, it is a rare finding.

Gross motor and fine motor delays are found with C-OMA and were present in our patient. A physical therapy, occupational therapy, speech and language and a psycho-educational evaluation were recommended to address the patient's non-ocular needs, such as building core body strength and posture, and addressing some of the auditory and sequential processing deficits. The psycho-educational evaluation would allow the school to assess the student's academic potential. If our patient's school performance is below his expected, specific actions including classroom and testing modifications may be mandated through an Individualized Education Plan (IEP).

Several simple classroom accommodations can be made that may have an immediate impact on his school performance. It is recommended that he be placed in the front of the classroom to reduce distraction by surrounding students. He should also be placed in the center of the classroom to reduce the need for head turning. Extra time and frequent breaks for all testing and assignments, including federal and state standardized assessments, should be sought after for this patient. Cognitively, he is a normal child. However, the energy and effort he expends during reading and school tasks to keep his eyes tracking along a page and scan in a busy classroom, is having a negative effect on his ability to comprehend and learn. He easily fatigues and is perceived by his classroom teachers as being "immature, off-task, and disruptive". It is very important to educate his classroom teachers about his visual condition of C-OMA and to expect the child to have funny head-thrusts, appear that he may not be looking at them directly or at his book, and that he will fatigue quickly and need frequent visual breaks.

A motor-based developmental approach is being implemented for our C-OMA patient.[29,38] From birth to his diagnosis at age 7 weeks old till now, he developed impaired visual spatial skills due to his inability to generate an accurate fixation and saccade, reducing his movement through space, thereby distorting his ability to localize objects in space.[39] Combined with the bilateral hip dysplasia, the C-OMA has negatively affected his gross-motor development, which is common among children with C-OMA. Our patient can be classified as "Lost in Space" or according to Skeffington, he is weak in the "Centering" and "Anti-gravity" circles.[40,41] He had very poor body awareness and bilateral integration and was unable to hop on one leg. The inability to aim his eyes accurately or localize himself in space has negatively affected his attention and cognition in the classroom, which may also explain his difficulties with speech and language skills.[42]

Our main goal is to stabilize and secure his body in space and provide him with more accurate visual-motor perceptual experiences. When his body becomes more stable and he has improved with overcoming gravity, he will develop the ability to center himself. This will eventually allow him to visually project his eyes into space. Once this happens, improvements should be seen with his fixation and smooth pursuit ability. Also by developing his bilateral integration and peripheral awareness skills, this will help him reduce the need to scan the environment. The use of vision therapy in association with congenital ocular motor apraxia has not previously been documented in the literature. We are attempting to improve gross motor skills, bilateral integration, balance and coordination combined with traditional ocular motility therapy, to achieve maximum results in this patient with a neurological inability to generate a voluntary saccade. Any improvement in his visual perceptual abilities and oculomotor abilities, either subjective or objective, will help facilitate learning and improve his quality of life.

Conclusion

Reading problems are pervasive in children with C-OMA. It is not an inability to learn to read, but rather a reduction in reading speed and efficiency. Unfortunately, if C-OMA is not diagnosed or detected early, a child may be labeled with dyslexia. Since these children are literate, the reading difficulty may actually represent a false or pseudo-dyslexia. In these cases, it is poor eye movements that lead to the inability of the patient to shift his eyes from word to word.

Reading in its most basic form is the saccadic movement of the eyes along a page from fixation to fixation. When the end of the line is reached, the eyes move down to the next line, a return sweep is made, and the rhythmic saccadic eye movement pattern across the line starts again. For an individual with normal ocular motility patterns this task is effortless and natural. However, for a child that suffers from ocular motor apraxia, reading becomes a fatiguing and frustrating task. The eye care community, especially developmental optometry, must be at the forefront in the diagnosis, treatment, and prevention of C-OMA in children.

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