Published in final edited form as:

Ophthalmology. 2011 September ; 118(9): 1859–1864. doi:10.1016/j.ophtha.2011.02.026.

Prevalence and Characteristics of Abnormal Head Posture in children with Down syndrome – a 20 year retrospective, descriptive review

Alina V Dumitrescu, M.D.[1], **Daniela C Moga, M.D.**[2,3], **Susannah Q Longmuir, M.D.**[1], **Richard J Olson, M.D.**[1], **Arlene V Drack, M.D.**[1]

[1]Pediatric Ophthalmology, Department of Ophthalmology and Visual Science, University of Iowa Hospital and Clinics

[2]Department of Epidemiology, College of Public Health, University of Iowa

[3]Biostatistics Epidemiology Research Design (BERD), Institute for Clinical and Translational Science, University of Iowa

Abstract

Purpose: To characterize the abnormal head posture (AHP) in children with Down syndrome (DS). Our study has three aims: to estimate the prevalence of AHP, to describe the distribution of different causes for AHP and to evaluate the long-term outcomes of AHP in children with DS evaluated at the University of Iowa Hospitals and Clinics (UIHC) between 1989 and 2009.

Design: Retrospective, chart review.

Participants: 259 patient records.

Methods: The study data was analyzed using chi-square tests (Fisher exact test when appropriate) to describe the relationship between the outcome of interest and each study covariate. A predictive logistic regression model for AHP was constructed including all the significant covariates.

Main outcome measures: AHP

Results: Over the study period, 259 records of patients with DS were identified. Of these, 64 (24.7%) patients had AHP. The most frequent cause of AHP was incomitant strabismus in 17 (26.6%) of 64 patients. The second most frequent cause of AHP was nystagmus, in 14 (21.8%) of 64 patients. For a substantial number of patients with AHP the cause could not be determined. They represented 12 (18.8%) of all the patients with AHP in this study and 12 (4.6%) of all patients with DS examined. When compared with patients with AHP from a determined cause, this

Corresponding author: Arlene V Drack, M.D. arlene-drack@uiowa.edu.
Address: Dr. Alina V. Dumitrescu The University of Iowa Ophthalmology Clinic 4120 MERF Iowa City, IA, 52246

Conflicts of Interest: No conflicting relationship exists for any author.

subgroup has a statistically significantly (p=0.027) higher percentage of atlantoaxial instability, by Fisher exact test. In our population 9/64 (14.1%) of patients with AHP had more than one etiology for AHP. Refractive errors, ptosis, unilateral hearing loss and neck and spine musculoskeletal abnormalities were responsible for AHP in a small percentage of patients. Of all the patients with AHP, 23 (35.9%) improved their head posture with treatment (glasses or surgery). An additional 6 (9.4%) of patients improved their posture spontaneously, over time without treatment.

Conclusions: The prevalence of AHP in the children with DS evaluated is 24.7%. From our analysis, having strabismus of any kind and particularly incomitant strabismus, and/or nystagmus is highly correlated with the developments of an AHP. Almost 19% of DS patients with AHP had no definitive etiology which could be determined.

Précis:

About one quarter of children with Down syndrome develop an abnormal head posture; in almost 19% of these children, no etiology can be found.

Introduction

Down syndrome (DS) is caused by trisomy of human chromosome 21 (Hsa21). The incidence of trisomy is influenced by maternal age and differs between populations (between 1 in 319 and 1 in 1000 live births are trisomic for Hsa21)[1, 2]. Many of the patients referred to a Pediatric Ophthalmology practice have the diagnosis of DS and are seen for either complete eye examination or for the presence of one or more ophthalmologic abnormalities. Numerous ophthalmic features found in children with DS have been reported in the literature[3, 4]. These include ocular findings not affecting vision, the most common of which were upslanting palpebral fissures, Brushfield spots and epicanthic folds, and ocular abnormalities with important impact on vision, such as malformation of the eyelids, cornea, iris and crystalline lens in addition to ametropia, strabismus, nystagmus, glaucoma and amblyopia. These abnormalities affect visual function by distorting or blurring the retinal image. Moreover, patients with DS may experience other functional and structural abnormalities at a higher frequency when compared to the general pediatric population. Considerable variability exists among infants and children with DS with regard to the degree of disability and the specific features affected. They are known to have, with increased frequency, atlantoaxial instability (AAI), scoliosis, musculoskeletal disorders, sensorineural hearing impairment, seizure disorder and mental retardation[5, 6]. The neck instability (symptomatic or not) increases the risk of spinal cord injury during the examination and clinical maneuvers in a child with Down's syndrome[7]. Greater joint range of motion, presumably attributable to laxity of the ligaments, delayed development of postural reactions and myelination, low muscle tone, and congenital heart defects all contribute to delayed motor skills.

In order to optimize visual acuity or maintain binocularity, children with one or more of the aforementioned abnormalities may develop a compensatory abnormal head posture (AHP). The purpose of head posturing has been identified as serving to enhance bifoveal fixation, promote single binocular vision, or improve visual acuity. Patients tend to adopt an AHP which can minimize diplopia and achieve fusion. Alternately, in some cases of acquired

AHP, patients may tilt their head to the side of the affected eye to exaggerate the separation of the two images in order to make one of the images easier to ignore. The AHP may also result from the patient trying to achieve the null-point of nystagmus. In those patients with nystagmus, with a null-point, head posturing provides the best visual acuity with smallest amplitude or highest frequency nystagmus. The null-point may be stable and unidirectional, bidirectional or variable. Less common causes of AHP include compensation for refractive errors, visual field defects, eyelid anomalies and cosmetic reasons. Non-ocular torticollis in childhood is most commonly the result of abnormalities of the central nervous system, hearing, the neck musculature, or the skeletal system.

Often, the ocular and non-ocular potential causes of AHP coexist (and may be more frequent in children with DS) and this multi-factorial cause of AHP affects the treatment options and prognosis. Ophthalmologists may be asked to evaluate such children to identify possible ocular etiologies for an AHP.

In a review of the medical literature from 1963 to 2010 using Pubmed, we were unable to identify any previous research conducted to characterize, extensively, the etiology and the evolution of AHP in children with DS, in contrast with literature describing the general ocular features encountered in association with this syndrome. The purpose of this paper was to evaluate retrospectively our experience over the last 20 years. We designed a descriptive study with three main aims: (1) to estimate the prevalence of AHP in children with DS evaluated in the Pediatric Ophthalmology Clinic at UIHC between 1989 and 2009, (2) to describe the distribution of different causes of AHP in our study population, and (3) to evaluate the long-term outcomes in this population and perform an statistical analysis of our findings.

Material and Methods

Institutional Review Board Committee approval was obtained and retrospective chart review was performed in accordance with Health Insurance Portability and Accountability Act regulations and adhering to the tenets of the Declaration of Helsinki. Between January 1989 and December 2009, records for 259 children with DS who were evaluated in the Pediatric Ophthalmology Clinic at the University of Iowa were identified. All these records were included in our retrospective analysis. Anonymous data on all of the patients was collected from medical charts using a standardized medical record abstraction form. From this retrospective cohort, we gathered information from all patients on the following:

- Demographic characteristics (e.g., gender, age at first presentation).
- Presence of comorbidities like hearing loss, spine and neck abnormalities, AAI, prematurity, heart malformations and gastrointestinal diseases.
- Presence of AHP at the first visit and during the follow-up period and age at the time of diagnosis if the AHP was not present at the first visit
- All known etiologic factors for AHP, as described in the literature, at the first visit and during the follow-up period, including those identified by other specialties (e.g., pediatrics, orthopedics, ear, nose, throat)

- Results from ophthalmological examination.
- Results of the imaging investigations (e.g., Magnetic resonance imaging [MRI], spine radiography-Rx, computed tomography [CT]).
- Treatment for AHP (medical and/or surgical) and evolution after treatment.
- The duration of follow-up.
- The number of visits after the first referral.

The parameters evaluated most constantly in our population were the measurement of the eye deviation in AHP and forced primary position, the behavior of the AHP with each eye occluded, the amplitude and frequency of nystagmus in AHP and forced primary position, and, where it was possible, the amount of fusion achieved in AHP versus forced primary position. The alignment of the eyes was examined using single and alternate cover test, Krimsky method and Hirschberg corneal reflex test.

Our statistical analyses were conducted as two-sided, at a level of significance $\alpha=0.05$ using SAS software Version 9.2 of the SAS System for Windows. Chi-square (Fisher exact) tests were used to characterize the distribution of AHP with regard to demographic characteristics (e.g., age at onset), while also evaluating the distribution of possible etiologic causes, duration of AHP, choice of treatment, and response to treatment. In order to evaluate the main factors associated with AHP in this population, we constructed a logistic regression model using a stepwise selection method. We evaluated the association between each independent variable and AHP. All of the variables that were significant in the bivariate analysis were considered for inclusion in our prediction model.

Parallel to the chart review we performed a review of the literature on AHP in patients with DS. We searched Pubmed for terms like “ocular features in DS”, “ocular motility in DS” and “abnormal head posture in DS”.

Results

Over a 20 year period, 259 patients with DS were examined and 64 (24.7%) of them had AHP. For this population group, there was no gender preference with 129 (49.8%) males and 130 (50.2%) females. For the entire group of patients with DS, the average age of presentation for the first visit was 43.1 months (3.6 years) and the average follow-up time was 66.3 months (5.5 years). The patients had on average 8.9 visits.

From all the patients with DS evaluated in our clinic during the study period, 137 (52.9%) had strabismus, 76 (29.3%) had nystagmus, 149 (57.5%) had refractive errors that required correction using glasses, 50 (19.3%) had nasolacrimal duct obstruction (NLDO), 7 (2.7%) had ptosis, 23 (8.9%) had cataract, 16 (6.2%) had corneal pathology, 4 (1.5%) had glaucoma, 62 (23.9%) had amblyopia, 113 (43.6%) had hearing loss, 55 (21.2%) had AAI (documented by spine X-ray), 10 (3.9%) had neck and spine musculoskeletal abnormalities (other than AAI), 33 (12.7%) were premature (with gestational age between 22 and 36 weeks) and 2 (0.8%) had ROP (no treatment required). The patients with cataracts had surgery in18/23 (78.3%) of cases. From those, 13/18 (60.87%) were aphakic after surgery

and 5/18 (17.4%) had primary intraocular lens (IOL) implanted. For 5/23 (21.7%) patients no surgery was performed.

In our sample, 163 (62.9%) patients had a significant (i.e., moderate and severe) degree of mental retardation and behavioral problems. Visual acuity could be quantified using optotypes (Snellen, LEA, Allen and Tellers) in only 86 (33.2%) patients. For the remainder, fixation preference was used to assess vision. Stereo vision can be assessed in only 62 (23.9%) patients.

Among the patients with AHP, 29 (45.3%) of them had AHP at the time of presentation and 35 (54.7%) developed it during the follow-up (table 1).

The 64 patients with AHP had various postures: head tilt 16 (25.0%), head turn 15 (23.4%), chin up 17 (26.6%), chin down 2 (3.1%) and 14 (21.9%) combined two or more of these postures in their head position.

The distribution of overall causes for AHP in our cohort is reflected in Table 2. We consider individual causes when they are exclusively responsible for AHP. There is a subgroup of patients with more than one cause for their head posture; these are in the “Combined” group. Patients for whom it was impossible to determine the cause of the AHP were assigned to the “Unclear” subgroup. These patients’ subgroups were not included in any other subgroup. “Neck and spine abnormalities” refers to scoliosis and muscular torticollis (nonocular); AAI is not included in this category.

The most frequent cause of AHP was incomitant horizontal and vertical strabismus (lateral rectus muscle palsy, A or V-patterns, DVD, superior oblique muscle palsy). Of 259 patients 137 (52.9%) had strabismus. Of the patients with strabismus 45/137 (32.8%) had AHP, of which 17/45 (37.8%) were exclusively due to the strabismus. Of the 259 patients, 32 (12.4%) had incomitant strabismus but only 17/32 (53.1%) of these had AHP due to strabismus alone. All the patients that had AHP attributed, with certitude, to strabismus had incomitant strabismus, 13/17 (76.5%) horizontal and 4/17 (23.5%) vertical. Of the 45 patients with strabismus and AHP, 5 (11.1%) had unclear cause for AHP, 7 (15.6%) were in the “combined” group, 3 (6.7%) had AHP due to refractive errors, 9 (20.0%) had AHP as null point for nystagmus and 4 (8.9%) had AHP due to ptosis. For strabismus in general, the crude odds ratio (OR) is 2.242 (p-value= 0.0061), but when incomitant strabismus alone is taken into consideration the crude OR increases to 10.589 (p value <0.0001). In other words the odds of developing an AHP is significantly higher if a child has DS and strabismus, even more if the strabismus is incommitant. In 6/17 (35.3%) patients with AHP due to strabismus, extraocular muscle (EOM) surgery was performed and this lead to improvement of the AHP in 5/6 (83.3%) of cases. For the remaining patients the AHP was unchanged after surgery. For patients with strabismus and AHP treated with glasses alone, 3/17 (17.6%) showed improvement in both strabismus and AHP (fully accommodative “A” or “V” pattern esotropia). None of the patients with strabismus and AHP improved in time without treatment.

The second cause of AHP, in this population, was nystagmus. It occurred in 76 (29.3%) out of 259 patients. Of the 76 patients with nystagmus, 34 (44.7%) had AHP. Almost half of

these patients, 14/34 (41.2%), used AHP to “dump” the nystagmus and 5/14 (35.7%) patients had EOM surgery. After the Kestenbaum procedure, 3/5 (60.0%) had significant improvement of AHP without recurrence during the follow-up. Of the remaining, 1/5 (20.0%) had recurrence during the follow-up and 1/5 (20.0%) had unchanged AHP after surgery. Of the patients with nystagmus and AHP 3/14 (21.4%) improved their AHP in time without treatment (glasses or surgery).

For a substantial number of patients with AHP the cause could not be determined. They represented 12 (18.8%) of all the patients with AHP in this study. They also represented 12 (4.6%) of all patients with DS examined. In this group, 5/12 (41.7%) of children had strabismus, 4/12 (33.3%) had manifest nystagmus, 2/12 (16.7%) both, 6/12 (50.0%) had glasses, mostly hyperopia and astigmatism 10/12 (83.3%), but none of these matched the AHP. The children with strabismus, in this subgroup, 3/5 (60.0%) had horizontal comitant strabismus, 1/5 (20.0%) had vertical strabismus and 1/5 (20.0%) had both. Of the patients in this group 4/12 (33.3%) had manifest nystagmus and an additional 1/12 (8.3%) had latent nystagmus. There was no difference in strabismic deviation in the AHP compared to forced primary or other head positions. The manifest nystagmus was not diminished by AHP. The patient with latent nystagmus had a face turn only at distance but not at near with either eye patched. The glasses did not change AHP.

Patching one eye did not correct the head posture. The AHP did not improve the visual acuity nor the streopsis. From the patients in this subgroup, 4/12 (33.3%) had AHP at the first visit and 8/12 (66.7%) developed it later during the follow-up. From this subgroup, 7/12 (58.3%) had AAI documented by spine X-ray, 8/12 (66.7%) had hearing loss documented with audiograms and 10/12 (83.3%) exhibited moderate to severe developmental delay. Of the patients with AHP with an “unclear” cause 2/12 (16.7%) improved their AHP over time without treatment, 1/12 (8.3%) increased their AHP over time and 3/12 (25.0%) were variable in amount and direction. None of the patients in this subgroup underwent surgery. When compared with patients with AHP from a determined cause (all together), by Fisher exact test, this subgroup has the statistically significant ($p= 0.027$) higher percentage of AAI.

Nevertheless, the causes of AHP coexist in many cases and the contribution of each of the causes to the head position is difficult to estimate. In our population 9/64 (14.1%) of patients with AHP were assigned to the “Combined” subgroup. Of them, 7/9 (77.8%) had strabismus and 3/9 (33.3%) had nystagmus. Neck and spine musculoskeletal abnormalities, other than AAI, were found in 3/9 (33.3%), ptosis in 2/9 (22.2%) and refractive errors that require glasses to correct in 8/9 (88.9%). This subgroup had the longest follow-up time and a higher average number of visits. The AHP changed with glasses or surgery for 4/9 (44.4%) of these patients but never disappeared completely. Of those patients 2/4 underwent ptosis surgery, 1/4 underwent strabismus surgery and 1/4 was prescribed glasses.

Refractive errors were responsible for AHP in 5/64 (7.8%) of children in our population. All of them improved their head posture significantly while wearing glasses. Not all the children were compliant with glasses and 1/5 (20.0%) of them improved over time even without wearing glasses permanently.

Ptosis, unilateral hearing loss and neck and spine musculoskeletal abnormalities (nonocular torticollis) were responsible for AHP in only a small percentage of patients. The patients with ptosis and AHP exclusively due to it benefit the most after surgery. In our sample 3/4 (75%) of patients underwent lid surgery and improved their head position significantly after the procedure. None of the patients had spine surgery.

From all 64 patients with AHP, 23 (35.9%) improved their head posture with treatment (i.e., glasses – 9, EOM surgery - 9 and ptosis surgery - 5) and 6 (9.4%) improved over time without treatment. Another 4 (6.25%) out of 64 underwent surgery (EOM - 3 and ptosis - 1) without improving in AHP.

In our data analysis we evaluated all the parameters that may be associated with AHP in the study group. Table 3 presents crude odds ratios with their confidence interval and p values, for all of the independent variables that were significantly associated with AHP in the bivariate analyses for the entire group of 259 patients. The model that best predicted AHP in our study population included incomitant strabismus, nystagmus, ptosis, length of follow-up, developmental delay and neck and spine abnormalities (other than AAI). Table 4 presents this model along with the adjusted odds ratios corresponding to each independent variable.

Discussion

Patients with DS represent an important group of patients in pediatric ophthalmology and some comprehensive ophthalmology practices. Over time they require extensive follow-up and many office visits. A complete diagnostic workup should be undertaken in children with DS who present with AHP, because 35.9% will benefit from treatment. The ocular pathology found in our study population compares with the findings described in the literature[3,4] Our focus was on the AHP subgroup. The most common treatable causes in our series are incomitant strabismus and nystagmus.

The prevalence of AHP in children with DS evaluated in the Pediatric Ophthalmology Clinic at the UIHC between 1989 and 2009 is 24.7%. It is difficult to compare this number with the prevalence in the general population (here defined as children without DS) due to lack of comprehensive studies available, but it is higher than reported in the few limited studies available (1.3% in a Chinese population study, 12% of all newborns during the first six months of life for asymmetric positional preference, 0/143 children in a Swedish study)[8–10]

Regardless of the cause of AHP, children with DS tend to develop it late in childhood, around 40 to 45 months of age (3–3.5y.). The length of the follow-up and the number of visits are statistically significantly correlated with occurrence of AHP and the longer the follow-up the higher the prediction of diagnosing an AHP.

In our population of DS patients the most frequent cause of AHP is incomitant horizontal and vertical strabismus and the second most frequent cause is nystagmus. This is consistent with what has previously been reported as the two most frequent ocular causes of AHP[11–15]. From our analysis, having DS and strabismus of any kind, particularly incomitant strabismus, is highly correlated with the development of an AHP. The same is true for nystagmus, ptosis and neck and spine musculoskeletal abnormalities. Children with DS have

a higher incidence of strabismus and nystagmus than the general population so our findings are concordant with the expectations.

For the third largest subgroup the cause of the AHP was uncertain even if they presented with at least one of the major predictors for AHP. We suspect a multifactorial etiology for AHP in these children, in which developmental delay and AAI or neck dystonia may play an important role. The only subgroup with an increased percentage of AAI was of patients with an “unclear” cause of AHP. For the entire study population AAI (documented by X-ray or CT) did not prove to be predictive nor statistically significantly associated with AHP. Considering that, further research should be done to determine whether in children with AHP without definitive etiology the AAI plays a role in causation.

Another hypothesis is that these patients may have Sandifer syndrome (SS)[16], which is an uncommon syndrome of abnormal posture of the head, neck, and trunk occurring in patients with gastroesophageal reflux disease (GERD), neural axis abnormalities, and benign paroxysmal torticollis. AHP may be a manifestation of atlantoaxial rotatory displacement resulting from trauma, or oropharyngeal inflammation (Grisel’s syndrome).

Retropharyngeal abscesses and pyogenic cervical spondylitis are unusual infectious causes of torticollis. Intermittent torticollis associated with headaches, vomiting, or neurologic symptoms may be caused by tumors of the posterior fossa. Benign and malignant neoplasms of the upper cervical spine are rare causes of torticollis in children. Torticollis resulting from cervical dystonia is also rare in children but may be seen in older adolescents. All of these are theoretically potential etiologies for AHP of unclear origin. They do not fully apply to our study population or they cannot be disproved in all the cases. Only 3/12 (25%) of children with AHP of unclear etiology had documented GERD, none had trauma or documented infections or tumors.

In a multi-disciplinary study of the ocular, orthopedic, and neurologic causes of AHPs in children without DS[17], in 63 cases congenital muscular torticollis (i.e., neck muscles dystonia, Klippel-Feil anomaly and brachial plexus injury) was found to be the single most common cause of an AHP, accounting for 55.5% patients, followed by incomitant strabismus 27%, nystagmus 12.7%, unclear 12.7% and neurologic (i.e., psychomotor delay and brain tumor) 7.9%.

In our population, a higher percentage of children 5/64 (7.8%) had AHP due to refractive errors as compared to what is reported in the literature for the general population (0.5 to 2.5%)[14,15].

In many children with AHP, more than one possible cause was present, making the diagnosis and treatment more challenging. The improvement of AHP with treatment in this subgroup is modest. In our regression model, not all the patients with strabismus, nystagmus or ptosis had an AHP but these abnormalities are predictive for developing one.

DS is the most common cause of mental retardation in childhood. There is also an increased incidence of other developmental disorders, including attention-deficit/hyperactivity disorder (ADHD), autism, and cerebral palsy. In these circumstances, the examination and accurate

strabismus and visual acuity measurements for patients with DS are challenging. Due to multiple comorbidities such as congenital heart conditions, neck anomalies and respiratory problems, the decision to do surgery is very complex in children with DS. General anesthesia may carry greater risks in this subgroup and the parents decline surgery more often. This may explain why only 17/64 (26.6%) of the patients with AHP in our study had surgical correction for AHP. The rate of success, defined as significant improvement of the AHP without recurrence during the follow-up was 14/17 (82.4%) from the patients who underwent surgery.

Despite a long follow-up and numerous visits, only 23 (35.9%), of all the patients with AHP improved their head posture with glasses or surgery. An additional 6 (9.4%) of patients with AHP spontaneously improved over time without treatment. Of these 3 (50.0%) had nystagmus, 2 (33.3%) had unclear etiology and 1 (16.7%) had AHP due to refractive errors.

A limitation of the current study is that it is retrospective and that it does not include all the patients with DS from a population over a period of time. Children with obvious neurological or orthopedic anomalies were probably missing from this study (as they were not referred to the ophthalmology department). The study sample included children with DS referred solely because of their diagnosis of DS, but also children in whom an eye abnormality was noted. Therefore an ascertainment bias toward higher likelihood of eye problems is probably present. We believe the DS population sample in this study is representative for the children with DS usually referred to a tertiary care facility.

It is important that when a child with DS is evaluated for an AHP, an array of possibilities is considered. Neurologic and ocular causes must be evaluated in addition to congenital neck and spine abnormalities, as well as developmental delay. When the diagnosis is not clear, a multidisciplinary approach may be appropriate to help plan the appropriate treatment protocol. Parents of children with DS may be told that 24.7 % of DS children develop an AHP based on our data. Of these, 32.8% will improve with treatment. In 18.8% of DS patients with AHP (or 4.6% of all DS patients) a definitive cause cannot be detected, but it is suspected to be related to musculoskeletal abnormalities and developmental delay. Up to 9.4% of patients with AHP may improve spontaneously over time.

Financial Support:

Dr. Drack is supported by a Marjorie Carr Adams Career Development Award from the Foundation Fighting Blindness and a Hope for Vision award. This publication was made possible by Grant Number UL1RR024979 from the National Center for Research Resources (NCRR), a part of the National Institutes of Health (NIH). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the CTSA or NIH.

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Table 1:

The characteristics of age at diagnosis.

	Gender	Avg. age at presentation	Avg. follow-up	Avg. # of visits	Avg. age AHP diagnosis
AHP at presentation	12 (41.4%) M	41.8 mo.(3.5y)	64.6 mo.(5.4y)	122	41.8 mo.(3.5y)
	17 (58.6%) F				
AHP in follow-up	18 (51.4%) M	12.0 mo.(1y)	100.9 mo.(8.4y)	145	45.5 mo.(3.8y)
	17 (48.6%) F				

AHP= abnormal head posture

Table 2:

The distribution of etiologic causes for abnormal head posture and their diagnostic timing.

	N (%)	Avg. age of presentation	Avg. follow-up	Avg. # of visits	Avg. age AHP diagnosis
Strabismus	17 (26.6%)	22.1 mo.(1.8y)	63.4 mo.(5.3y)	12.9	33.5 mo.(2.8y)
Nystagmus	14 (21.8%)	16.1 mo.(1.3y)	77.3 mo.(6.4y)	9.7	43.6 mo.(3.6y)
Unclear	12 (18.8%)	32.6 mo.(2.7y)	90.0 mo.(7.5y)	12.0	57.9 mo.(4.8y)
Combined	9 (14.1%)	20.6 mo.(1.7y)	134.1 mo.(11.2y)	18.7	35.0 mo.(2.9y)
Refractive errors	5 (7.8%)	28.1 mo.(2.3y)	36.0 mo.(3y)	7.8	40.5 mo.(3.4y)
Ptosis	4 (6.3%)	67.0 mo.(5.6y)	66.0 mo.(5.5y)	10.5	73.5 mo.(6.1y)
Neck and spine abnormalities	2 (3.1%)	6.0 mo.(0.5y)	153.0 mo.(12.8y)	34.5	10.5 mo.(0.8y)
Hearing loss	1 (1.5%)	18.0 mo.(1.5y)	24.0 mo.(2y)	3.0	18.0 mo.(1.5y)

AHP= abnormal head posture

Table 3:

Crude odds rations with their confidence interval and p values for independent variable significantly associated with abnormal head posture

Parameter	Odds ratio	95% confidence interval	p-value
# of visits	1.041	1.014 to 1.068	0.0021
Length of follow-up	1.006	1.001 to 1.010	0.0091
Strabismus	2.242	1.259 to 3.995	0.0061
Incomitant strabismus	9.699	4.272 to 21.983	<.0001
Amblyopia	2.230	1.200 to 4.143	0.0112
Developmental delay	2.321	1.218 to 4.422	0.0105
Glasses	2.236	1.211 to 4.129	0.0101
Nystagmus	4.129	2.270 to 7.508	<.0001
Ptosis	8.170	1.545 to 43.193	0.0134
Neck and spine abnormalities	4.939	1.348 to 18.103	0.0159

Table 4:

Logistic regression model, odds ratio estimates.

Effect	Point Estimate	95% Wald Confidence Limits	
Incomitant Strabismus	10.598	4.276	26.268
Follow-up	1.005	1.000	1.010
Developmentally delay	2.020	0.943	4.325
Nystagmus	5.272	2.630	10.568
Ptosis	10.579	1.399	79.979