Early Intervention Memorandum 1999-2

To: Early Intervention Officials
    Providers of Early Intervention Services
    Families
    Other Interested Parties

From: Donna M. Noyes, Ph.D.
      Director
      Early Intervention Program

Date: December 10, 1999

Subject: Reporting of Children’s Eligibility Status Based on Diagnosed Conditions with High Probability of Developmental Delay

This memorandum provides guidance on the manner in which children's eligibility status should be reported, and subsequently recorded in the Kids Data Management System. This information is applicable whether the results of a multidisciplinary evaluation indicate that the child is eligible for early intervention services based on a documented developmental delay or based on a diagnosed physical or mental condition that has a high probability of resulting in developmental delay. This memorandum also provides guidance on the types of diagnosed conditions that are considered to have a high probability of developmental delay in very young children; and, the qualified professionals who can diagnose these conditions according to the practice acts of New York State.

Please note that this guidance is not a change in eligibility requirements under the Early Intervention Program. Rather, the goal of this guidance document is to improve the information about the population of children receiving services from the Early Intervention Program. Better information on the basis for all children’s eligibility is essential to evaluating the program’s effectiveness in identifying children as early as possible. Better information on each child's eligibility is essential to ensuring children receive high quality evaluations and appropriate early intervention services. Thus, whenever a diagnosis is made or becomes available, the information should be documented in the child’s record and recorded in the KIDS Data Management System.
Review of Eligibility Requirements Under the Early Intervention Program

Children’s eligibility under the Early Intervention Program (EIP) can be established in one of two ways: (1) presence of a developmental delay which meets the state definition of developmental delay\(^1\) or (2) a diagnosed physical or mental condition that has a high probability of resulting in developmental delay\(^2\). In an effort to clarify conditions with a high probability of developmental delay, the EIP and Data Committee of the Early Intervention Coordinating Council (EICC) convened a group of expert clinicians to identify categories and types of diagnosed conditions that - when diagnosed in children under three years of age - have a high probability of resulting in developmental delay. A compendium of these conditions, including a description and the appropriate ICD-9 code, has been included in the Appendix A of this guidance memorandum. A table has also been included which describes those licensed and certified professionals who are permitted under state law to make such diagnoses under their scope of practice as defined in Education Law (Appendix B).

It is important to remember that although a diagnosed mental or physical condition with a high probability of developmental delay establishes the child's eligibility for the Early Intervention Program, all decisions about appropriate services, including

- types of services that are necessary to address the child’s strengths and needs and the family’s priorities, resources and concerns about their child’s development;
- frequency, intensity, and duration of services; and,
- settings in which services are to be delivered

can only be determined by reviewing the results of the child’s complete multidisciplinary evaluation and in collaboration with the full IFSP team.

Review of the Evaluation Process

A child’s eligibility for the Early Intervention Program must be established by the approved, multidisciplinary evaluation team selected by the parent to complete the child’s multidisciplinary evaluation, regardless of whether the child is referred as having:

- a suspected developmental delay only;
- a suspected diagnosis of a condition with a high probability of developmental delay; or,
- a diagnosed condition with a high probability of developmental delay.

When a child is referred as having a suspected developmental delay, the child’s eligibility for the Early Intervention Program is established if a developmental delay as defined at 10 NYCRR Section 69-4.1(g) is documented by the multidisciplinary team. In

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\(^1\) 10 NYCRR Section 69-4.1(g)
\(^2\) 10 NYCRR Section 69-4.1(h)
some instances, the child may have a developmental delay consistent with the state
definition of developmental delay.

When a diagnosed condition with a high probability of developmental delay is
suspected at the time of referral, or suspicion of such a condition emerges during the
multidisciplinary evaluation, it is important to assist the family in obtaining an accurate
diagnosis while also ensuring that an IFSP is developed within the forty-five day
timeframe for children who are eligible based on a developmental delay. For example, a
child may be referred with a suspected motor delay and demonstrate characteristics that
constitute clinical clues for cerebral palsy during the evaluation process. If the
multidisciplinary evaluation team determines that a child is experiencing a developmental
delay which qualifies him or her for the Early Intervention Program, an initial
Individualized Family Service Plan should be developed and may include a supplemental
evaluation as appropriate to obtain a diagnosis. Under these circumstances, the
multidisciplinary evaluation team and service coordinator should also work closely with
the child’s primary care physician and medical community in an effort to assist the family
in obtaining an accurate diagnosis. If feasible within the forty-five day timeframe, the
initial multidisciplinary evaluation team may also be expanded to include a professional
qualified under the state practice acts to make an accurate diagnosis (for example, a
physician or nurse practitioner with an appropriate area of specialty and competence).

When a child is referred with a diagnosed condition with a high probability of
developmental delay, the approved multidisciplinary evaluation team must, with parental
consent, obtain the documentation substantiating the diagnosis from the professional who
made the diagnosis. This documentation may include appropriate medical records and/or
the medical reports or summary reports from the diagnosing professional. The
multidisciplinary evaluation team approved to evaluate children for the Early Intervention
Program is then responsible for incorporating this information into the child’s
multidisciplinary evaluation report and summary.

To summarize, all children referred to the Early Intervention Program who are
thought to be eligible, must, with parental consent, be evaluated by an approved
evaluator. The multidisciplinary evaluation team may, with parental consent, screen a
child to determine what type of evaluation, if any, is necessary unless the child has a
diagnosed condition with a high probability of developmental delay. If a child has a
diagnosed condition with a high probability of developmental delay, a screening is
unnecessary and the multidisciplinary evaluation team should proceed with an evaluation
with parental consent. The multidisciplinary evaluation is necessary to:

- determine whether a child is eligible for the Early Intervention Program;
- assess the status of the child’s physical, cognitive, social-emotional,
  communication, and self-help development;
- identify areas of developmental strengths and needs; and,

3 10 NYCRR Section 69-4.8(2)(i)
- determine the parent’s resources, priorities, and concerns related to their child’s development.

The complete multidisciplinary evaluation forms the basis for decisions about all aspects of the services included in the Individualized Family Service Plan to meet the child’s developmental strengths and needs, and the priority, resources, and concerns of the family related to their child’s development.

Upon completion of a multidisciplinary evaluation, the child’s evaluator must prepare and submit an evaluation report that includes the following information: the names, titles, and qualifications of the persons performing the evaluation and assessment; a description of the assessment process; the child’s responses; the family’s belief about whether the responses were optimal; measures and/or scores that were used; and, an explanation of these measures or scores. In addition, the evaluation report must include a statement of the child’s eligibility (a diagnosed condition with a high probability of developmental delay and/or developmental delay in accordance with the state definition of developmental delay). Early Intervention Officials should review the qualifications of the members of the multidisciplinary evaluation team to ensure that appropriately qualified personnel made the indicated diagnosis.

Please note that the diagnosed conditions with a high probability of developmental delay included in this guidance document do not constitute an exhaustive list of diagnosed conditions that may establish a child’s eligibility for early intervention services. When an evaluator identifies a child as having a condition with a high probability of developmental delay not included in Appendix A, a clear explanation and supportive documentation from the medical research literature must be provided as to why the condition is considered to have a high probability of developmental delay. This information should be submitted as part of the child’s multidisciplinary evaluation report.

It is recommended that the multidisciplinary evaluation team submit a summary form to accompany the evaluation report to assist in ensuring that data concerning the child’s eligibility form are entered accurately in the Kids Data Management System. A suggested form is provided in Appendix C of this document.

1. What if a child is referred to the Early Intervention Program with a diagnosed condition with a high probability of developmental delay—should a multidisciplinary evaluation still be provided?

Yes. Children referred to the Early Intervention Program with a diagnosed condition with a high probability of developmental delay that connotes eligibility for the Early Intervention Program must have a multidisciplinary evaluation by an approved evaluator. The evaluation provides the basis for decisions about appropriate services to meet the identified needs of the child and family.

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4 10 NYCRR Section 69-4.8 (9)(ii)
5 10 NYCRR Section 69-4.8(9)(iii)
2. If a child has a diagnosed condition, does the evaluator still have to include developmental status in each area of development?

Yes. Even when a child's eligibility is based on a diagnosed condition with a high probability of developmental delay, he or she must also be evaluated in all five developmental domains in accordance with Section 69-4.8(a) (4) of the Early Intervention Program regulations. Section 69-4.8(a) (4) indicates that evaluation and assessment must include the child's level of functioning in each of these areas: cognitive development; physical development, including vision and hearing; communication development; social or emotional development; and adaptive development.

Section 69-4.8(a) (9) (iii) of the regulations requires that the evaluation report and summary include a statement of the child’s eligibility, including diagnosed condition with a high probability of delay, if any and/or developmental delay in accordance with the definition of developmental delay at Section 69-4.1(g). The definition of developmental delay provides for qualified personnel to make diagnoses based on informed clinical opinion, appropriate diagnostic procedures and/or instruments and specifies the criteria for eligibility based on a developmental delay. These criteria include: a twelve month delay in one functional area; or a 33% delay in one functional area or a 25% delay in each of two areas.

The statement of eligibility required in both the evaluation report and summary is intended to clearly indicate whether the child is eligible to receive services under the Early Intervention Program and, if eligible, the child's diagnosis or degree of developmental delay present which qualifies them to receive early intervention services. Determining the degree of developmental delay present must be done using diagnostic instruments and informed clinical opinion and these results must be interpreted and included within the statement of eligibility. When the evaluators feel that results from standardized instrument(s) are not reflective of a child's true abilities or cannot be obtained at all, the evaluators must use informed clinical opinion to document the child's eligibility by demonstrating that the child is evidencing a delay of either 33% in one developmental domain or 25% in two or more domains. When the evaluation report and summary clearly documents a child's eligibility for early intervention services in accordance with the regulatory language, the family should receive information that will help them better understand their child's developmental abilities and needs, and to participate more meaningfully in discussions about services for their child.

Information obtained as a result of the multidisciplinary evaluation is necessary to inform the Individualized Family Service Plan process, resulting in functional outcomes and services that best meet the needs of the eligible child and family. Additional concerns or needs may be recognized by the parent or service provider once the IFSP is in place. Should this occur, the need to amend the IFSP can be established through on-going assessment or through a supplemental evaluation. The decision regarding whether to pursue on-going evaluation a supplemental evaluation is contingent on the judgement of the IFSP team, considering the nature of the additional information being sought and the resources available to obtain the specific diagnostic information needed.
3. **Is a child with a diagnosed condition still eligible for early intervention services even if he/she is currently developmentally age-appropriate?**

   Yes. Children with some diagnosed conditions (e.g., those contained in Appendix A) are eligible for early intervention services, even if delays are not evident at the time of the evaluation. For certain conditions, developmental delays may not be initially evident, but are likely to emerge as the child ages (e.g., Down syndrome, certain hearing or vision impairments, etc.).

   The diagnosis of a condition with a high probability of developmental delay must be made by personnel qualified under state law to do so. The evaluation report must provide *adequate documentation* that the diagnosed condition is present. For conditions included in Appendix A, it is not necessary to include additional documentation from the medical research literature indicating why the condition is considered to have a high probability of resulting in a developmental delay. As noted above, for diagnosed conditions not included in Appendix A, the evaluation report must include such additional documentation.

   The recently issued clinical practice guidelines on assessment and intervention for children with autism and children with communication disorders provide recommendations for the effective evaluation and assessment of children with these conditions and are excellent resources.

4. **What if the Early Intervention Official (EIO) believes that an evaluation does not establish a child’s eligibility?**

   If a diagnosis of developmental delay (Early Intervention Program regulations, 10 NYCRR Section 69-4.1(g)) or a condition with a high probability of developmental delay (Section 69-4.3(e), also see Appendix A), was made by an individual(s) qualified to do so under state law, the child is eligible for early intervention services. If, however, the Early Intervention Official believes that the evaluation report does not establish a child’s eligibility, the EIO or Service Coordinator (SC) can request additional supporting documentation or that an additional evaluation(s) be completed. Section 69-4.8 (a) (12) of the Early Intervention Program regulations state that “with parental consent, certain evaluation and assessment procedures may be performed or repeated and costs may be reimbursed as a supplemental evaluation in accordance with this subpart, if deemed necessary and appropriate by the early intervention official….”

   Parents must receive notification when an additional evaluation(s) is being arranged for their child and must give written consent before any evaluation is administered. When a child’s eligibility has been clearly established, a second evaluation cannot be used to refute eligibility. Only in those instances when eligibility has not been definitively established is it appropriate for an EIO to request additional information.
5. What if a child enters the Early Intervention Program with an established delay, and someone suspects the child has a diagnosed condition with a high probability of developmental delay?

When a child's initial eligibility for the Early Intervention Program is based on an established delay, and a diagnosed condition is suspected, the EIO or service coordinator should be contacted regarding the need for further evaluation to confirm the diagnosis. Under these circumstances, the service coordinator should also work closely with the child’s primary care physician and medical community in an effort to assist the family in obtaining an accurate diagnosis. A supplemental evaluation may be added to the child's Individualized Family Service Plan (IFSP) and the child should be referred - with parental consent- to an appropriately qualified individual (see Appendix B, attached) in order to establish a diagnosis. Section 69-4.30(c)(2)(ii) provides for supplemental evaluations, which include supplemental physician or non-physician evaluations and are provided upon the recommendation of the multi-disciplinary team conducting the core evaluation and agreement of the child’s parent. A supplemental evaluation must be stated in the child’s Individualized Family Service Plan, and must include the type of supplemental evaluation, and the date and evaluator if known.6

In the event a diagnosis is established, this information should be recorded in the KIDS data management system. This up-dated information does not alter the child's eligibility status, but may necessitate changes in the services delivered to the child and his or her family. When new diagnostic information becomes available, the IFSP team should confer to discuss whether any changes in early intervention services are appropriate.

6. What if a child is referred based on risk of having a disability according to Section 69-4.3(f), Referral, of the Early Intervention Program regulations and the risk factor connotes a possible diagnosis with a high probability of developmental delay?

Under Section 2542 of New York State Public Health Law (Comprehensive Child Find and Public Awareness System), the Early Intervention Program’s child find system must provide for the identification, tracking, and screening of children at risk of developmental delays using available resources. Section 69-4.3(f) of the Early Intervention Program regulations includes a list of risk factors that require referral to the Early Intervention Official unless a parent objects to such referral.

Some of the risk factors included in Section 69-4.3(f) have been further clarified as factors that warrant a referral for a multidisciplinary evaluation. These include suspected hearing impairment and suspected vision impairment and very low birth weight (birth weight less than 1,000 grams). Children referred to the Early Intervention Program with any of the diagnosed conditions with a high probability of developmental delay should receive a multidisciplinary evaluation, as discussed under question one. Children

6 10 NYCRTR Section 69-4.8(a)(13)
whose histories include a risk factor(s) included in Section 69-4.3(f) should be referred to the Early Intervention Officials for tracking purposes and should receive developmental surveillance and screening from their primary health care provider. Any child for whom questions arise regarding their developmental progress (e.g., through a positive developmental screen) should be referred to their Early Intervention Official for a multidisciplinary evaluation.

Further Guidance

For more information regarding eligibility for the Early Intervention Program or any other aspect of the program, please contact:

New York State Early Intervention Program
New York State Department of Health
Room 287 Corning Tower Building
Albany, New York 12237-0660
(518) 473-7016

Attachments
ICD Codes
Diagnosis Table
Evaluation Summary Form
## Appendix A – Early Intervention Memorandum 1999-2

### Updated ICD Codes

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I. Syndromes/Conditions

Conditions

Cleft Palate: 749.0 (Cleft Lip: 749.1; Cleft Palate and Lip: 749.2)

Description:
Cleft palate is a congenital fissure in the median line of the palate (bony roof of the mouth) that may extend through the uvula, soft palate, and hard palate. Cleft lip may or may not be involved. Clefts involving the palate and/or lip are classified several ways. Classification systems differ in terms of anatomical references (e.g., the American Cleft Palate Association differentiates between clefts of the prepalate [lip and alveolar process], while Davis and Ritchie consider the position of the cleft relative to the alveolar process). Regardless of the specific classification system used, clefts may be unilateral, bilateral, complete or total, and incomplete, partial, or subtotal. One other type is called a submucous cleft. These are further defined below.

- Complete or total: cleft palate in which the cleft extends from the lip through the alveolar process, hard palate, and soft palate.
- Incomplete, partial, or subtotal: cleft palate in which the cleft can be limited to the lip, alveolar process, hard palate, or soft palate, or a combination of these structures.
- Bilateral: failure of the palate on the right and left sides to fuse to the nasal partition or septum.
- Unilateral: fusion of the palate to the vertical nasal septum only on one side.
- Submucous: condition in which the surface tissues of the hard or soft palate unite, but the underlying bone or muscle tissues do not (also called occult cleft palate).

Effects/Prognosis:
Cleft palate is associated with feeding and swallowing problems, failure to thrive (poor growth), aspiration, recurrent ear infections, and hypernasal, dysarticulate speech. Specific speech/language characteristics also include nasal emission of air during production of fricative sounds and delayed development of language skills. Undesirable facial distortions or mannerisms may also accompany speech.

Extreme Prematurity (Preterm Infant)

Less than 500 grams: 765.01
500-749 grams: 765.02
750-999 grams: 765.03

Description:
Infants weighing less than 1500 grams are referred to as “very low birth weight” (VLBW) babies and comprise 1.5 percent of all births in the U.S. However, this rate is increasing primarily due to the greater numbers of multiple birth babies who are more likely to be born early and weigh less. Survivability correlates with gestational age for infants whose weight is appropriate for gestational age. Statistics from 2002 show that the rate of survival for babies born weighing less than 500 grams was 13.8%, 51% for birth weights of 500-749 grams, and 84.5% for birth weights of 750-1000 grams for the
first year of life. While the mortality rate has diminished with the use of surfactants, the proportion of surviving infants with severe complications has not.

Effects/Prognosis:
Outcomes for this population are variable. VLBW babies are at increased risk for neurodevelopmental complications. Surviving VLBW infants are affected by major deficits including spastic motor dysfunction (cerebral palsy) and associated mental retardation. These deficits appear to result largely from two defined lesions: intraventricular hemorrhage (IVH) and periventricular leucomalacia (PVL). IVH is classified into 4 grades ranging from Grade I to Grade IV. The outcome in infants with grades I and II is good; as many as 40% of infants with grade III IVH have significant cognitive impairment, and as many as 90% of infants with grade IV IVH have major neurologic sequelae. Outcomes for PVL are correlated with outcomes for Grades III and IV IVH. Other complications in VLBW babies may include breathing problems (hyaline membrane disease or respiratory distress syndrome), feeding difficulties, seizure disorders, hydrocephalus, retinopathy of prematurity (also called retrolental fibroplasia), and increased risk for serious or protracted illness. Other associated problems of prematurity include heart abnormalities, renal problems, and vision and hearing problems. Rehospitalization during the first year of life is not uncommon. The average number of physician visits is also higher than for infants of normal birthweight. The presence of a congenital anomaly or developmental delay was a determining factor in physician use. These infants typically have complicated medical courses and often go home with multiple treatments and medications. Longer-term problems for these infants may include behavior problems at preschool age and decreased performance on standardized IQ tests.

Syndromes/Chromosomal Abnormalities

Angleman’s syndrome (congenital malformations affecting multiple systems): 759.89

Description:
Angleman’s syndrome is a disorder characterized by severe mental retardation with marked delay in attaining motor milestones, episodes of inappropriate laughter, and limited or absent speech. Other signs include ataxia and jerky arm movements, said to resemble a “puppet gait.” Seizure activity is most severe at about age four years and may stop by age 10 years. Decreased need for sleep between ages two and six years has also been reported.

Effects/Prognosis:
Severe difficulties with speech; most individuals communicate using alternate means (e.g., sign language). Receptively, simple commands may be understood. Most individuals become toilet trained by day and some by night. All require a supported living arrangement.
**CHARGE Syndrome: 759.89**

**Description:**
CHARGE syndrome is an association of multiple congenital malformations including absence of part of the eye or retina (coloboma), heart disease, nasal blockage (choanal atresia), retarded growth and development with mental retardation ranging from mild to profound, genital anomalies, ear anomalies, and sensorineural hearing loss. Visual and auditory problems may further compromise cognitive function. Other findings may include but are not limited to a small jaw (micrognathia), cleft lip, cleft palate, multiple cranial nerve abnormalities, facial palsy, feeding difficulties resulting from poor suck and velopharyngeal incompetence, kidney (renal) anomalies, and growth hormone deficiency.

**Effects/Prognosis:**
Effects depend on the severity of the defects. In some cases, death occurs shortly after birth due to respiratory insufficiency or heart disease. In less severely involved individuals, most show some degree of mental deficiency and/or CNS defects, along with visual and auditory deficits. Feeding difficulties, facial palsy, and sensorineural hearing loss are related to cranial nerve abnormalities.

**Down Syndrome (Trisomy 21 or 22,G): 758.0**

**Description:**
Down syndrome is a chromosomal disorder that includes the following clinical features: hypotonia (decreased muscle tone), short stature, flat facial profile, epicanthal folds, upslanting eye slits, small ears, speckling of the iris, cardiac defects, duodenal atresia, atlantoaxial instability (enlargement of distance between first two neck vertebrae that leaves the individual susceptible to spinal cord compression and neurological involvement), thyroid disorders, hearing loss (may be conductive, mixed, or sensorineural), and mental retardation. Other attributes include poor coordination and relatively slow physical growth in the first 8 years. Sleep-related upper airway obstruction has been reported in about one-third of cases.

**Effects/Prognosis:**
Multisystem disorder with varying degrees of severity. The I.Q. range is reported to be between 25 and 50, with occasional individuals above 50. Varying degrees of hearing loss and speech impairment are also associated. Social performance is usually beyond expectations for mental age.

**Edwards’ Syndrome (Trisomy 18, E3): 758.2**

**Description:**
Edwards’ syndrome is a chromosomal syndrome and the second most common multiple malformation syndrome, having an incidence of 1/3000 births. Clinical findings include clenched hands with overlapping fingers, rocker-bottom feet (rigid flat feet), short sternum, heart disease, severe mental retardation, and failure to thrive (poor growth). Poor sucking capability contributes to failure to thrive and may necessitate nasogastric feeding. Other characteristics include hypertonicity (after the neonatal period) and diminished response to sound.
Effects/Prognosis:
Babies with this syndrome are described as feeble and have limited capacity for survival. Resuscitation is often performed at birth, and episodes of apnea may occur in the neonatal period. Fifty percent die within the first week, and many of those remaining die within the first 12 months. Five to ten percent survive the first year. These individuals are typically unable to walk independently and usually have a very limited expressive vocabulary (a few single words). Some older children with Trisomy 18 reportedly smile, laugh, and interact with their families. All are reported to achieve some psychomotor maturation and to continue to learn.

Fetal Alcohol Syndrome: 760.71
Description:
Fetal alcohol syndrome (FAS) is a syndrome resulting from effects of maternal alcohol ingestion. Malformations are caused in the developing fetus. Clinical findings in FAS include pre- and postnatal growth retardation, mild to moderate microcephaly, cognitive deficits (usually mild to moderate in degree), and characteristic facial features including short eye slits (palpebral fissures) and a smooth indentation in the upper lip below the nose (philtrum). A heart murmur may be present, frequently disappearing by one year of age. Associated difficulties may include myopia, strabismus (cross eye/squint), hearing loss, dental malocclusion, eustachian tube dysfunction, articulation problems, language disorders, specific learning disabilities, and attention deficit/hyperactivity disorder.

Effects/Prognosis:
Individuals with FAS may appear similar to those with “failure to thrive.” They tend to be irritable as infants and hyperactive as young children. A diagnosis of mild mental retardation is frequently reported. In general, the severity of the maternal alcoholism and the extent and severity of the pattern of malformation are predictive of the ultimate prognosis. A follow up study of a group of adolescents and adults with FAS (average age 18 years) revealed academic functioning at a fourth grade level, with difficulty in mathematics noted. Other common behavioral characteristics in this group included poor judgment, distractibility, and difficulty recognizing social cues.

Fragile X Syndrome: 759.83
Description:
Fragile X syndrome is also known as Martin-Bell syndrome, Marker X syndrome, and Escalante syndrome. This is a genetic syndrome characterized by mental retardation ranging from mild to profound in males. Attention problems related to hyperactivity and autism are commonly seen. Other features include macrocephaly (abnormally large head) and macrosomia (large body size) in early childhood, thickening of the nasal bridge extending down to the tip of the nose, large ears, pale blue eyes, epicanthal folds (crescent-shaped folds of skin extending down from the side of the nose to the lower eye lid and partially covering the inner corner of the eye opening), and dental crowding. Hand flapping or biting and poor eye contact are also characteristic. A speech pattern called “cluttering” (rapid speech with some syllables omitted, which may be difficult to understand) is also typical in higher-functioning individuals. Occasional associated abnormalities may include submucous cleft palate and heart disease. In females, involvement tends to be mild; shyness, anxiety and panic attacks are reportedly seen.
Effects/Prognosis:
Effects are dependent on the degree of cognitive involvement and are more pronounced in males. Females typically show milder effects but do have educational difficulties.

**Patau’s Syndrome (Trisomy 13, D1): 758.1**

**Description:**
Patau’s syndrome is a chromosomal disorder with multisystem involvement with an estimated incidence of 1/8000 births. Clinical findings include microcephaly (abnormally small head), incomplete development of the forebrain, severe mental retardation, clefting (lip and palate), hyperconvex nails, and extra fingers or toes (polydactyly). Other characteristic abnormalities include apneic spells in early infancy, deafness, visual deficits, and heart disease in more than 50 percent of cases. Fewer than one child in five survives the first year of life. Advanced maternal age is a contributing factor in the occurrence of this syndrome.

**Effects/Prognosis:**
The average survival for children with this disorder is 2.5 days, with only 5 percent surviving the first 6 months. Survivors reportedly have severe mental defects and fail to thrive. Seizures are also typical.

**Prader-Willi Syndrome: 759.81**

**Description:**
Prader-Willi syndrome is also known as HHHO syndrome (Hypotonia-Hypogonadism-Hypomentia-Obesity syndrome). This is a genetic syndrome characterized by severe obesity, mental retardation (primarily in the mild to moderate range), poor muscle tone in infancy, short stature, small hands and feet, incomplete sexual development, and behavior problems. Feeding problems are associated with this disorder in infancy, and tube feeding is sometimes necessary. The hypotonia may cause respiratory problems as well. Failure to thrive may occur in early infancy, followed by a marked change with the development of an insatiable appetite. Onset of obesity may occur between age 6 months and 6 years, and is related to increased intake with reduced activity. At the same time, the hypotonia generally improves.

Three phases are identified: infancy with decreased muscle tone (hypotonia) and failure to thrive; childhood with features including almond-shaped eyes, small eye slit length, ravenous appetite (hyperphagia), cognitive deficits (ranging from mental retardation to learning disabilities and language impairments), and young adulthood with increased severity of the childhood symptoms along with severe emotional and behavioral symptoms.

**Effects/Prognosis:**
Typical effects include mental retardation, most often in the mild to moderate range. Dietary management is key to prolonging life expectancy and reducing the chances of complications related to morbid obesity (e.g., cardiac complications). Speech problems (articulation) and hypernasal speech are noted. The individual may also perseverate on favorite topics.
II. Neuromuscular/Musculoskeletal Disorders

**Congenital Hereditary Muscular Dystrophy: 359.0**

***Description:***
Congenital hereditary muscular dystrophy is a disease of the muscles. It is present at birth and is manifested in the infant by low muscle tone. All muscular dystrophies are genetically determined. However, the term congenital muscular dystrophy is used to encompass several distinct diseases with a common characteristic of severe involvement at birth.

***Effects/Prognosis:***
Infants often have contractures or arthrogryposis (multiple congenital contractures). Head control is poor. Facial muscles may be mildly involved. The prognosis is variable: the disease may progress or stay the same.

**Other Myopathies: 359.8**

***Description:***
Myopathy is a disease or abnormal condition of striated muscle. Myopathies encompass a widely varied group of muscle diseases, which are characterized by weakness in infancy or childhood. Other myopathies include endocrine myopathies (thyroid and steroid-induced) and metabolic myopathies (potassium-related periodic paralysis, malignant hyperthermia, glycogenoses, mitochondrial myopathies, lipid myopathies, and vitamin E deficiency myopathies).

***Effects/Prognosis:***
The effects and prognosis of other myopathies are variable, depending on the specific underlying disease.

**Werdnig-Hoffmann Disease: 335.0**

*(Infantile Spinal Muscular Atrophy)*

***Description:***
Werdnig-Hoffmann disease is the severe infantile form of spinal muscular atrophy. Spinal muscular atrophies are degenerative diseases of motor neurons that begin in fetal life and continue to be progressive in infancy and childhood.

***Effects/Prognosis:***
Affected infants have severe hypotonia and generalized weakness. Infants who are symptomatic at birth may have respiratory distress and are unable to feed. Most infants demonstrate symptoms by six months of age. The disease is progressive, and two-thirds of severely involved infants die by two years of age.

**Spinal Cord Injury, NOS (unspecified site of spinal cord injury w/o spinal bone injury: 952.9**

***Description:***
Spinal cord injury is a trauma to the spinal cord during the birth process or afterwards. Strong traction exerted during delivery may produce fracture and separation of the vertebrae. Transection of the cord may occur with or without vertebral fractures.
Effects/Prognosis:
Effects are variable, depending on the level and location of the lesion. There may be loss of sensation and paralysis below the level of injury. If the injury is severe, the infant may deteriorate rapidly and die within several hours. The course may be protracted, with symptoms and signs appearing at birth or later in the first week. Immobility and associated brachial plexus injuries may not be recognized for several days. Treatment of survivors of spinal cord injury is supportive, and affected infants often remain permanently injured.

**Lobster Claw (Cleft Hand, Congenital): 755.58**

Description:
Lobster claw is a deformity of the extremities that causes deep clefts in the anterior part of the hand.

Effects/Prognosis:
Fingers may have various degrees of syndactyly (webbed fingers). The prognosis is good when only one extremity is involved. The prognosis may be poor if multiple congenital anomalies are present.

**Arthrogryposis: 728.3**

Description:
Arthrogryposis is not a disease but a descriptive term that signifies multiple congenital contractures. Multiple contractures around the joints of the arms and legs result in wasting of the muscles and loss of function. Involvement ranges from mild deformities to deformities that make functioning almost impossible.

Effects/Prognosis:
The prognosis is variable, depending on the underlying cause.

**Phocomelia (absence of limb): 755.4**

Description:
Phocomelia is a congenital malformation where the proximal portions of the extremities are poorly developed or absent. The hands and feet may be attached to the trunk directly or by means of a poorly formed bone. In complete phocomelia, the hand or foot seems to spring directly from the trunk.

Effects/Prognosis:
Severe deformities of the extremities are often associated with other malformations incompatible with life. The prognosis is variable, depending on the severity of the involvement and associated defects.

**Spina Bifida without hydrocephalus (unspecified region): 741.9**

Description:
Spina bifida is a disorder of early fetal development resulting in failure of the spinal cord to fuse properly. Spina bifida occulta, which is an opening in the spine but no protrusion of membranes, nerves, or spinal cord is the least serious form. This form does not usually cause loss of body function. Meningocele is the protrusion of the membrane-like coverings of the spinal cord and nerves. Myelomeningocele, characterized by a sac
containing spinal cord and nerves protrudes through the defect in the vertebrae is the most severe type of spina bifida.

Effects/Prognosis:
Depending on the location and severity of the spinal lesion, some or all of the functions made possible by a normal spinal cord and nerves may be decreased or absent below the spina bifida. Spina bifida may affect muscle control, movement, and strength; sensation in the legs and lower body; urinary and bowel function; and neurological function.

**Spina Bifida with hydrocephalus (unspecified region): 741.0**

Description:
Spina bifida is a disorder of early fetal development resulting in failure of the spinal cord to fuse properly. (Please refer to spina bifida without hydrocephalus, 741.9 for a definition of spina bifida.) Hydrocephalus develops in some cases with spina bifida when the normal flow of cerebrospinal fluid is blocked and becomes trapped within the spaces that lie inside the brain.

Effects/Prognosis:
Depending on the location and severity of the spinal lesion, some or all of the functions made possible by a normal spinal cord and nerves may be decreased or absent below the level of the spina bifida. The prognosis is variable, depending on the level of the lesion and how much of the spinal cord is involved. Hydrocephalus usually requires some form of neurological shunt placement to drain cerebrospinal fluid.

III. Central Nervous System (CNS) Abnormalities

**Infantile Cerebral Palsy (not otherwise specified-NOS): 343.9**

Description:
Cerebral palsy (CP) is a static encephalopathy (a generalized disorder of cerebral function) resulting from injury to the brain during its early (fetal, perinatal, and early childhood) stages of development. Cerebral palsy is a condition that involves the brain’s ability to control the muscles. Muscle and nerve function is essentially normal. Cerebral palsy is categorized by a description of the resultant motor handicap: *physiologic* classification identifies the major motor manifestation and *topographic* taxonomy indicates the involved extremities.

*Physiologic:*
1) Spasticity (muscle stiffness - hypertonia) present in 60 percent of all cases of CP;
2) Dyskinesia (involuntary movements) present in approximately 20 percent of all cases of CP;
3) Athetosis – slow writhing movements;
4) Choreoathetosis – abrupt and jerky movements;
5) Dystonia – slow, rhythmic movements; and
6) Ataxia (a broad-based, lurching gait with primary balance difficulties) occurs by itself in about one percent of all CP.

*Topographic:*
1) Diplegia – involvement of the trunk and all four extremities (the legs more so than the arms);
2) Hemiplegia – involvement of one side of the body only;
3) Paraplegia – involvement of the legs only;
4) Quadriplegia – involvement of both arms, both legs, the head, and the trunk;
5) Monoplegia – involvement of one extremity; and
6) Triplegia – involvement of three extremities.

Effects/Prognosis:
Some children have barely detectable problems, while others have severe disabilities. Difficulties in mobility and communication are the major functional manifestations. There also may be associated problems with cognition, vision, and behavioral responses. Hearing impairment, seizures, constipation, and feeding difficulties can be accompanying health problems. Given the comprehensive care they require, children with CP can look forward to an average life span.

Infantile Spasms without Intractable Epilepsy: 345.60
with Intractable Epilepsy: 345.61

Description:
Infantile spasms are brief symmetrical contractions of the muscles of the neck, trunk, and extremities resulting in a jackknifing of the body. The character of the seizure depends on whether the flexor or extensor muscles are predominantly affected and the particular muscle groups involved. Infantile spasms usually begin between the ages of four and eight months and frequently emerge as a new development in a series of neurological abnormalities. Seizure activity typically occurs in clusters and tends to develop when the child is drowsy or immediately upon awakening. Eye movements and a pre- or post spasm “cry” frequently accompany the seizure episodes. The most common EEG finding is hypsarhythmia, a continuous disorganized pattern of high voltage slow waves and spikes. ACTH and corticosteroids are the present treatment options. However, there is no clear evidence that ACTH treatment improves the long-term outcome in terms of mental retardation although there is often an improvement in the social interaction of the infant when spasms are controlled. Infantile spasms will subside in approximately one year in about 25 percent of the cases and in two years in another 25 percent of the cases. The spasms may last five years or more and may be replaced by other types of seizure activity.

Effects/Prognosis:
Infantile spasms are typically classified into two categories: cryptogenic and symptomatic. Cryptogenic infantile spasms are characterized by normal preseizure health history, normal neurological examination, and normal CT scan. Ten to twenty percent of infant spasms are classified as cryptogenic and children with this subtype have a good prognosis. Symptomatic infantile spasms are characterized by abnormal prenatal and postnatal factors including hypoxic-ischemic encephalopathy, congenital infections, inborn errors of metabolism, neurocutaneous syndromes, cytoarchitectural abnormalities, prematurity, central nervous system infections, and head traumas. Children with symptomatic infantile spasms have an 80 to 90 percent risk of mild to severe mental retardation, depending on the severity of the neurological abnormalities before seizures appeared. Children with infantile spasms typically have delayed psychosocial development, and motor deficits such as spasticity and hypotonia. Microcephaly, cortical blindness and/or deafness, and a variety of other central nervous system (CNS) deficits are also present.
Usually these problems are present in differing degrees prior to the onset of infantile spasms. The presence or absence of certain risk factors can significantly alter the prognosis. Death occurs in approximately 29 percent of children with infantile spasms.

**Encephalocele: 742.0**

*Description:*
An encephalocele is a neural tube defect resulting in the herniation of the meninges and portions of the brain (cerebral cortex, cerebellum, or portions of the brainstem) through a bony midline defect in the skull, most commonly in the occipital region. The neural tissue within an encephalocele is often abnormal. This condition is one of two major forms of dysraphism (defective fusion of parts that usually unite). The first form, a cranial meningocele, is less severe and consists of a meningeal sac only.

*Effects/Prognosis:*
The amount of compromised and deformed neural tissue determines the extent of cerebral dysfunction and the resultant disabilities. Even brain tissue not extending into the encephalocele may be structurally and functionally abnormal. The following problems are manifested: hydrocephalus, microcephaly, motor delays with weakness and/or spasticity, ataxia, seizures, and visual problems.

**Microcephalus: 742.1**

*Description:*
Microcephaly is a congenital abnormality of the central nervous system where the head circumference measures more than three standard deviations below the mean for age and sex. Microcephaly is divided into two main groups: primary or genetic and secondary or nongenetic. Primary microcephaly refers to a group of conditions that is associated with specific genetic syndromes (Down syndrome, Cri-du-chat, Cornelia de Lange, Edward-18-trisomy, Rubinstein-Taybi, Smith-Lemli, Optiz); usually has no other malformations, and follows a Mendelian pattern of inheritance (autosomal recessive, autosomal dominant). Secondary microcephaly results from a large number of noxious agents that may affect the fetus in utero or the infant during periods of rapid brain growth, particularly during the first two years of life (radiation, drugs, congenital infections, meningitis, encephalitis, malnutrition, etc.). Occasionally, microcephaly is due to premature closure of the cranial sutures (craniosynostosis) but more often is the result of micrencephaly (a small brain).

*Effects/Prognosis:*
The clinical manifestations and degree of central nervous system (CNS) dysfunction vary, but there is a correlation between the severity of the microcephaly and the degree of mental retardation. Almost 90 percent of children with microcephaly will have mental retardation. Milder decreases in head size have been associated with learning disabilities and language disorders.
**Congenital reduction deformities of the brain: 742.2**

**Holoprosencephaly: 742.2**

**Description:**
Congenital reduction deformity of the brain is an anterior midline defect that occurs during early fetal development (before 23 weeks gestation) of the midface and the forebrain. The consequences of this defect are varying degrees of malformations in midline facial development and in brain development and function. The incidence is 6-12/100,000 live births, and although the cause is unknown, the condition is sometimes seen in conjunction with chromosomal anomalies and as an autosomal dominant and autosomal recessive defect. It is also seen in children of diabetic mothers and as a result of congenital infections (CMV, toxoplasmosis, and syphilis). Holoprosencephaly is categorized into 3 types according to clinical features:

1. Alobar – anophthalmia (congenital absence of one or both eyes), cyclopia, median and bilateral cleft lip and palate, microcephaly, severe mental deficiency, apneic spells, seizures, death;
2. Semilobar - orbital hypotelorism, microphthalmia, coloboma, normal lip and palate, absence of philtrum, median cleft lip, flat nose, single-nostril nose, mild to severely mentally retarded; and
3. Lobar - normal face, single maxillary incisor, minimal handicap, mildly to severely mentally retarded.

**Effects/Prognosis:**
The prognosis is dependent on the severity of the involvement; although most affected individuals die before 6 months of age. Mildly affected individuals may live to adulthood. The degree of facial malformation is usually predictive of brain malformation. This condition is considered the most devastating of the anterior midline defects. Complications may include endocrine abnormalities such as hypopituitarism, ACTH-adrenal axis failure, and diabetes insipidus.

**Lissencephaly: 742.2**

**Description:**
Lissencephaly is a disorder of neuronal migration characterized by the absence of sulcation of the cerebral hemispheres resulting in a “smooth brain.” An insult before 12 weeks gestational age prevents successive waves of migrating neurons from reaching the cerebral cortex. This disorder is associated with various syndromes such as, Miller-Dieker, Walker-Warburg, HARD+/E Syndrome. The following abnormalities are seen depending on the clinical subtype affecting the individual: open Sylvian fossa, absent or hypoplastic corpus callosum, large cavum septi pellucidi, small midline calcifications in the region of the third ventricle, microcephaly, high wrinkled forehead, small nose with anteverted nostrils, micrognathia, slanted palpebral fissures, low-set and/or posteriorly angulated auricles, late eruption of primary teeth, cryptorchidism, pilonidal sinus, polydactyly or syndactyly, transverse palmar crease, cataracts, hypoplasia of optic nerve, microphthalmia, retinal dysplasia, hydrocephalus, congenital heart disease, duodenal atresia, and renal agenesis.
Effects/Prognosis:
This condition may result in symptoms of failure to thrive, repeated aspiration pneumonia, hypotonia (or rigidity and opisthotonos), infantile spasms or other seizure activity, and severe mental retardation. Infants may exhibit brief visual fixation, smiling, and nonspecific responses to stimulation. Developmental skills are minimal, and death usually occurs before 2 years of age.

**Congenital Hydrocephalus: 742.3**

Description:
Hydrocephalus is a condition that results from impaired circulation and absorption of cerebral spinal fluid (CSF) or, in rare circumstances, from increased production by a choroid plexus papilloma within the intracranial cavity. Hydrocephalus can be the result of obstruction within the ventricular system (obstructive or noncommunicating hydrocephalus), or the result of obliteration or malfunction of the absorption sites, i.e., the subarachnoid cisterns or the arachnoid villa (obstructive or noncommunicating hydrocephalus). Hydrocephalus is termed either “congenital” (when it exists at birth) or “acquired” (when it occurs as the result of injury to the brain after birth).

Effects/Prognosis:
When there is an excessive accumulation of CSF in the ventricular system, the resultant pressure (intracranial pressure) leads to various symptoms depending on the age of the child, whether or not the cranial sutures have fused, and the treatment rendered. A frequent surgical treatment is the placement of a ventriculoperitoneal shunt, which diverts CSF from a lateral ventricle to the peritoneal cavity. Prognosis depends on the cause of the dilated ventricles as opposed to the size of the cortical mantle at the time of operative intervention. There is an increased risk of developmental disabilities including a reduced mean intelligence quotient, particularly for performance tasks; abnormalities in memory function; visual problems such as strabismus, visual spatial abnormalities, visual field defects and optic atrophy; and aggressive and delinquent behavior in some children.

**Cystic Periventricular Leukomalacia (CPVL): 348.8**

Description:
Cystic periventricular leukomalacia is a softening of the white matter of the brain in the area of the ventricles due to hypoxic ischemic injury. Intraventricular hemorrhage in the premature infant is often a factor in the development of necrosis of the periventricular white matter and the resulting cystic formation.

Effects/Prognosis:
Babies with CPVL are at a high risk for developmental abnormalities. The degree of white matter necrosis influences the severity of the mental and/or motor problems that result. These abnormalities may include spastic diplegia (legs more involved than arms), delays and qualitative problems in motor development, slow mental development, problems with hearing or vision, seizures, attention deficits, poor coordination or balance, problems with eye-hand coordination, learning disabilities, and behavioral difficulties.
**Intraventricular Hemorrhage (grade IV): 772.1**

**Description:**
Intraventricular hemorrhage (IVH) is a bleeding in the tissue surrounding the ventricles of the brain, most common in premature infants. The incidence of IVH increases with decreasing birth weight, with 80-90 percent of the cases occurring between birth and the third day of life. There are four levels of hemorrhage that have been defined as follows:
1. Grade I - bleeding is confined to the subependymal matrix
2. Grade II - indicates intraventricular bleeding
3. Grade III - includes grade II plus intraventricular dilatation
4. Grade IV - includes grade III plus intracerebral bleeding.

**Effects/Prognosis:**
Babies with grade IV bleeds frequently develop serious ongoing neurological problems, although the degree of neurological impairment may be related to a combination of factors such as the initial hypoxic or other insult, the hemorrhage itself, increased intracranial pressure, or the ventricular dilatation following the hemorrhage. Ten to fifteen percent of low birth weight infants with IVH develop hydrocephalus. Leukomalacia and porencephalic cysts are other common complications. The following problems may develop: fine and gross motor delays, cerebral palsy, vision and hearing impairments, and nongenetic learning disabilities.

**Kernicterus: 774.7**

**Description:**
Kernicterus is damage to particular parts of the brain (basal ganglia and brainstem nuclei) due to the accumulation of high levels of unconjugated bilirubin. An increase in bilirubin production or a decrease in bilirubin excretion or both will result in neonatal hyperbilirubinemia (bilirubin concentrations greater than 10 mg/dL in premature infants or 15 mg/dL in full-term infants). Excessive bilirubin accumulation from any cause can produce kernicterus, especially in the preterm or sick newborn. Some possible causes of neonatal hyperbilirubinemia are as follows: fetal-maternal blood group incompatibility, extravascular blood such as pulmonary or cerebral hemorrhage, polycythemia such as fetal to fetal transfusion, obstructive disorders such as a band or tumor, sepsis, intrauterine infection, respiratory distress syndrome, asphyxia, and/or being the child of a diabetic mother.

**Effects/Prognosis:**
Hyperbilirubinemia is treated by early, frequent feedings, phototherapy, and exchange transfusions in an effort to prevent kernicterus. It is also vital to diagnose and treat the underlying cause of the hyperbilirubinemia to prevent or lessen the serious consequences of kernicterus. Kernicterus can result in the following problems: opisthotonos, muscular rigidity, seizures, hypotonia, bilateral choreoathetosis with involuntary muscle spasm, extrapyramidal signs, mental deficiency, dysarthric speech, high-frequency hearing loss, squints and defective upward movement of the eyes, and ataxia. In mildly affected infants, the syndrome may be characterized only by mild to moderate neuromuscular incoordination, partial deafness, or minimal brain dysfunction, occurring singly or in combination.
Multiple anomalies of brain (NOS): 742.4

Congenital cerebral cyst: 742.4
(See True porencephalic cyst below)

Macrocephaly: 742.4

Description:
Macrocephaly is an occipitofrontal head circumference more than two standard deviations above the mean for age. The most frequent causes of macrocephaly are mass lesions (porencephalic cysts, tumors, subdural hematomas, etc.), megalencephaly, and hydrocephalus. Macrocephaly can be familial or associated with a number of syndromes and several storage diseases.

Effects/Prognosis:
Most significant degrees of macrocephaly are likely to indicate the presence of neurodevelopmental disorders. The effects and prognosis will depend on the underlying condition responsible for the increase in head circumference and the availability of prompt and effective treatment.

Megalencephaly: 742.4

Description:
Megalencephaly is an abnormally large and oftentimes malfunctioning brain.

Effects/Prognosis:
Megalencephaly can be a familiar trait not associated with any other deficiencies, but usually is an indication of underlying medical problems such as epilepsy and other neurological conditions.

Porencephaly: 742.4

Description:
Porencephaly is a fluid-filled cyst or cavity within the cerebrum. There are two types of porencephalic cysts:
1.) True porencephalic cysts are cysts that occur as a result of faulty embryonic neurodevelopment. They typically communicate with the ventricles, cerebral cortical surface, and/or subarachnoid space.

Effects/Prognosis:
True porencephalic cysts are associated with more severe neurologic manifestations. Some of the possible manifestations are as follows: hypotonia, seizure disorder, developmental delay, mental retardation, mild to severe motor dysfunction, failure to thrive, optic atrophy, delayed limb growth, hydrocephalus, and supranuclear bulbar palsy.
2.) Pseudoporencephalic cysts typically arise after a well-defined destructive event (of normal brain tissue) such as vascular disruption or infection occurring late in fetal or early infantile life (intraventricular hemorrhage, periventricular leukomalacia, congenital infections). This event leads to a cavitation of the necrotic region and cyst formation within the parenchyma of the cerebral hemispheres. These cysts usually do not communicate with other structures, tend to be unilateral, and are typically not associated with other disorders.
Effects/Prognosis:
Pseudoporencephalic cysts are associated with hemiparesis and focal seizures in the first year of life. Prognosis is variable: some children develop only minor neurological signs and have normal intelligence.

IV. Hearing, Vision, and Communication Disorders

Retinopathy of Prematurity (Retrolental fibroplasia): 362.21 (grades 4 and 5)

Description:
Retinopathy of prematurity is also known as Terry disease. This is an eye disease that is a major cause of blindness. Retinopathy of prematurity (ROP) occurs primarily in premature infants and is more severe with decreasing birth weight. ROP results from abnormal development of the retina (the light sensitive lining of the eye) in premature babies. It occurs when abnormal blood vessels and scar tissue form at the edge of the normal retinal blood supply. The abnormal retina then has a damaging demand for oxygen.

ROP is a progressive disease that starts slowly, usually between the fourth to tenth week of life. Progression may be slow or rapid through Stages 1-5, or the disease may stop at the early stages and disappear completely. Not all premature infants develop ROP. The two critical factors for predicting who will develop ROP are birth weight less than 1500 grams and gestational age at birth less than 32 weeks. High oxygen levels may exacerbate ROP but do not cause the disease. Stages 4 and 5 reflect more severe involvement than the earlier stages.

Stage 4 ROP is caused by the scar tissue formed in Stages 1 through 3 pulling on the retina and causing it to separate from the wall of the eyeball. Stage 4 is subdivided, depending on the location of the detachment. In stage 4A, the detachment is partial and outside the macula (the area of central vision), and it may or may not affect the infant’s vision. Stage 4B is characterized by partial detachment involving the macula. Stage 5 ROP involves a complete retinal detachment. Infants with Stage 5 ROP have essentially no useful vision in that eye.

Effects/Prognosis:
Stages 1, 2, and 3/mild require monitoring. Treatment is instituted at Stage 3/moderate or severe because these infants have a 50 percent chance of proceeding to Stage 4 or 5 and possible blindness. For Stage 4A, the chance for usable vision is relatively good if the retina reattaches. Stage 4B generally results in a more limited prospect for usable vision due to the macular involvement. Treatment options at Stage 5 involve surgery to attempt to reattach the retina. Some vision may be recovered by this surgery, but the individual will most likely be legally blind in the involved eye.

Conductive hearing loss unspecified (NOS): 389.00

Description:
Conductive hearing loss refers to hearing loss arising from failure of sound pressure to reach the cochlea (inner ear) through the normal air conduction channels (outer and/or middle ear). This type of hearing loss results from a problem in the outer and/or middle
ear space and can range from mild to moderate-severe in degree. Causative factors range from fluid in the middle ear space to congenital malformations of the outer and/or middle ears. Depending on the cause, conductive hearing loss may fluctuate. In a conductive hearing loss, hearing sensitivity will be impaired via the air conduction (outer + middle + inner ear) pathway and normal via the bone conduction (inner ear) pathway.

Effects/Prognosis:
Effects of conductive hearing loss are dependent on the extent and duration of the hearing loss. For example, with a 30 dB HL (mild) hearing loss, 25-40 percent of the speech signal may be missed. This can result in difficulty hearing consonant sounds. The individual will generally have to expend greater energy to listen, resulting in fatigue. Typical symptoms include “not paying attention” and “daydreaming.” Depending on the cause, conductive hearing loss may be treated and cured through medical and/or surgical means. If the hearing loss cannot be resolved medically, amplification can be pursued if the individual is otologically cleared. Periodic audiological monitoring of hearing levels and middle ear function will be needed. Speech-language consultation may also be required.

It should be noted that the Early Intervention Program regulations (Section 69-4.3[e][5]) specify that a hearing impairment qualifying as a diagnosed condition with a high probability of developmental delay is a diagnosed hearing loss that cannot be corrected with treatment or surgery. Only conductive hearing losses that are not amenable to resolution through medical or surgical means, are chronic in nature, and/or have an impact on other areas of development, in particular speech/language/communication development, meet the requirements to determine eligibility in the Early Intervention Program. Consistent with the regulatory language, an occasional or transient conductive hearing loss occurring in isolation, i.e., without concomitant delays in other developmental domains, would typically be managed through the child's primary medical care provider.

**Sensorineural hearing loss (NOS): 389.1**

Description:
Sensorineural hearing loss is a hearing loss resulting from a pathological condition in the inner ear (cochlea) or along the nerve pathway from the inner ear to the brainstem (cranial nerve 8). It may be cochlear or retrocochlear depending on the site of the lesion. Sensorineural hearing loss may range from mild to severe to profound. Causes are varied and include congenital abnormality in the auditory nerve, damage to the cochlea (e.g., from certain antibiotics such as gentamycin), and diseases such as meningitis. Certain syndromes are associated with sensorineural hearing loss. High-risk factors for sensorineural hearing loss include but are not limited to low birth weight (less than 1500 grams), anoxia, jaundice, cranial defects, congenital viral infections (e.g., rubella), and family history of hearing loss.

Effects/Prognosis:
Effects of sensorineural hearing loss depend on numerous factors, including the degree (extent) of hearing loss, age at onset of hearing loss, age at identification of the hearing loss, and amplification (personal hearing aids, FM system) history. For example, an individual with a moderate (41-55 dB HL) sensorineural hearing loss can miss 75 to 80
percent of the speech signal without amplification. Effects on speech-language skills may include delayed or defective syntax, limited vocabulary, imperfect speech production, and an atonal voice quality. With a severe hearing loss (71-90 dB HL), the individual may hear loud voices about one foot away from the ear. Optimal amplification will provide access to environmental sounds and too many speech sounds. Individuals with profound hearing loss (91 dB HL or more) may rely on vision as the primary avenue for communication and learning. If the loss occurs prelingual, (before speech development), oral language and speech may not develop or will be severely delayed. Prognosis varies depending on multiple factors including consistent amplification and intervention emphasizing development of language, speech, and auditory skills. Regular audiological consultation to monitor hearing levels and amplification will be required.

Mixed Conductive and Sensorineural Hearing Loss: 389.2  
**Description:**  
Mixed hearing loss is a combination of conductive (outer and/or middle ear) and sensorineural (inner ear) hearing loss occurring simultaneously. The hearing loss may range from mild to severe to profound.  

**Effects/Prognosis:**  
Effects of the hearing loss depend on numerous factors as described above. Management of the hearing loss will include medical consultation regarding the conductive (outer and/or middle ear) component and may include amplification to address the remaining hearing loss once the individual is otologically cleared. Regular audiological consultation to monitor hearing levels, middle ear status, and any amplification needs will be required. Speech-language consultation may also be required.

Unspecified congenital anomaly of the ear with impairment of hearing: 744.00  
**Description:**  
Anomalies of the ear generally include external ear changes that make these syndromes easier to diagnose. The hearing loss may be congenital or slowly progressive. Syndromes include but are not limited to:

1. Atresia (closure) of the external auditory canal with conductive hearing loss;
2. Ear malformations, persistent periauricular pits (depressions around the external ear), sinuses or nodules, and mixed hearing loss;
3. Preauricular pits, persistent branchial (Gill) clefts or fistulas, and sensorineural hearing loss;
4. Malformed low-set ears and conductive hearing loss;
5. Small external ear, meatal (ear canal) atresia and conductive hearing loss; and
6. Lop ears, small lower jaw, and hearing impairment of mixed type.

When a middle ear deformity is present, the stapes, which develops from different embryological origins than does the malleus and incus, is usually involved. Other structural lesions producing hearing impairment involving congenital inner ear anomalies have been described. These include the following:

1. The Michel type;
2. The Mondini-Alexander type;
3. The Bing-Siebenmann type;
4. The Scheibe or cochleo-saccular type (which is the most common type of congenital abnormality and accounts for 70 percent of cases);
5. The Siebenmann type; and
6. Type VI exhibits microtia (abnormally small external ear) and atresia (absence or closure) of the external meatus (ear canal).

Effects/Prognosis:
Effects are variable and dependent on numerous factors including the extent of hearing loss. Otologic management will be required to address the medical aspects and audiological management will be required to maximize the use of the individual’s residual hearing. Speech-language consultation may also be required.

**Dyspraxia Syndrome (Developmental coordination disorder): 315.4**

Description:
Dyspraxia is a less severe form of apraxia. Apraxia is defined as a disruption in the ability to transmit or express a motor response along a specific modality. It involves disruption of voluntary or purposeful programming of muscular movements while involuntary movements remain intact. Dyspraxia is characterized by difficulty in articulation of speech, formation of letters in writing, or in movements of gesture and pantomime. In speech, it is a nonlinguistic sensorimotor disorder of articulation characterized by impaired capacity to program the position of speech musculature and the sequencing of muscle movements (respiratory, laryngeal, oral) for the volitional production of phonemes. Synonymous terms include oral, speech, or verbal apraxia.

Effects/Prognosis:
Effects vary, depending on the severity of involvement. If severely affected, the treatment may include accessing other modes of communication, e.g., sign language or an augmentative communication device.

**Blindness, both eyes: 369.00**

Description:
A person is termed “blind” when there is corrected visual acuity less than 20/200 in the better eye. It has been recommended that the definition of the term “blind” be restricted to the absence of light perception and that “visual impairment” and “low vision” be extended to describe persons with vision less than 20/200 but who are able to retain light perception.

Effects/Prognosis:
Effects vary depending on such factors as the onset of blindness. Individuals with congenital blindness (born blind) may be developmentally delayed because the process of learning through the visual channel has been severely limited. Those with adventitious blindness (blindness occurring after birth) may be developmentally similar to others in their age group depending on when the vision loss occurred. Blind infants may show delays in the areas of gross motor development related to the development of locomotion, in prehension (physical grasp) skills, and in the development of attachment.
Blindness, one eye, low vision other eye: 369.10

Description:
See definition for blindness above. Low vision generally refers to severe visual impairment and is not necessarily limited to distance vision. Low vision applies to all individuals who are unable to read a newspaper at a normal viewing distance, even with the aid of corrective lenses. The rate of visual handicaps is higher among individuals with multiple handicaps.

Effects/Prognosis:
Effects depend on the age of onset and amount of residual vision. Screening for potential visual abnormalities during infancy or early childhood is important because this is the optimal time for preventing or minimizing visual impairments. Signs of eye problems include frequent squinting or rubbing of the eyes, lack of attention, or irritability.

Low vision both eyes (moderate to severe): 369.20

Description:
See definition for low vision above.

Effects/Prognosis:
See information under low vision (369.10) above.

Optic nerve coloboma (bilateral), congenital (Specified congenital anomalies of optic disc): 743.57

Description:
Colobomata are areas of absent tissue and are found where fetal clefts or fissures fail to close. In the eye, these may involve portions of the optic disc (the circular tip of the optic nerve).

Effects/Prognosis:
Involvement of the optic disc results in depressed central vision. Depending on the extent and location of the coloboma, there may be decreased visual acuity, nystagmus, strabismus, photophobia, and a loss of visual fields. Treatment options include cosmetic contact lenses and/or sunglasses for colobomas of the iris. Optical aids may be helpful. When a coloboma of some part of the inner eye is suspected, visual fields measurement is suggested.

Optic nerve coloboma (bilateral) acquired (Coloboma of optic disc): 377.23

Description:
A coloboma is a defect in the eye, usually a fissure or cleft of the iris, ciliary body (thickened part of vascular tunic of the eye between the base of the iris and the anterior part of the choroid), or choroid (dark brown vascular coat of the eye between the sclera and the retina extending from the ora serrata to the optic nerve and consisting of blood vessels united by connective tissue). This condition may be a result of surgery.

Effects/Prognosis:
See above.
Aniridia: 743.45

Description:
Aniridia is congenital absence of all or part of the iris. This defect is usually accompanied by photophobia, nystagmus, and defective vision. Other associated conditions include glaucoma, progressive corneal degenerative changes, cataracts, macular hypoplasia, and optic nerve hypoplasia. Transmission of this condition may be familial or sporadic. Sporadic aniridia is associated with Wilms tumor in 1/70 patients. In Aniridia-Wilms Tumor Association, abnormalities include moderate to severe mental deficiency in most patients, growth deficiency and microcephaly in one-half of patients, craniofacial abnormalities, and aniridia in most patients, and Wilms tumor (a malignancy of the kidney) in one-half of patients.

Effects/Prognosis:
It is estimated that one-third of individuals with sporadic aniridia develop Wilms tumor, while 50 percent of those with combined aniridia, genitourinary anomalies, and mental retardation develop Wilms tumor. Effects of this disorder depend on the severity of the deficits. Associated eye abnormalities in addition to aniridia include congenital cataracts, nystagmus, ptosis (upper eyelid droops below its normal level), and blind spot. With aniridia, there is usually decreased visual acuity (circa 20/200), photophobia, possible nystagmus, cataracts, displaced lens, and underdeveloped retina. Visual fields are usually normal unless glaucoma develops. Because the macula (the most sensitive part of the retina) does not fully develop ("macular hypoplasia"), reduced vision occurs. The macula is used for fine vision, such as for reading. Treatment strategies include pinhole contact lenses, tinted lenses and/or sunglasses, corrections for refractive errors, optical aids, and lower illumination levels to control glare. Magnification may also be helpful. Long-term prognosis is poor if glaucoma develops.

Albinism: 270.2

Description:
Albinism is a genetic absence of pigment of the skin, hair, and eyes (or eyes only) resulting from a metabolic defect. Visual impairment (refractive errors), strabismus (cross eye, squint), nystagmus (involuntary eye movements), and photosensitivity are common.

Effects/Prognosis:
Due to absence of normal protection by melanin in the skin, individuals with albinism are predisposed to damage from ultraviolet light. They require use of sunscreen during exposure to sunlight. Eye abnormalities require ophthalmological evaluation and treatment/correction based on the diagnosed condition.

Visual deprivation nystagmus: 379.53

Description:
Nystagmus is an oscillatory motion of the eyes that may be congenital or acquired. Congenital nystagmus is pendular (back-and-forth movements that are at roughly the same speed) at rest, with irregular jerking when the eyes are deviated to the sides. It is usually associated with poor visual acuity and is thought to be due to failure of development of visual fixation in infancy. Congenital nystagmus often accompanies
congenital visual impairment (e.g., corneal opacity, cataract, albinism, aniridia, optic atrophy, or chorioretinitis).

Nystagmus may also involve jerk-type movements in which there is a rapid movement in one direction followed by a slower recovery movement in the opposite direction. The character of the oscillation may change in different positions of gaze, and there may be a null point at which the nystagmus is minimal. Patients with a null point tend to maximize their vision by assuming a head position in which the nystagmus is least marked. This head-turning to position eyes at a null point is a natural and effective means of improving vision.

Nystagmus is classified according to the position of the eyes when it occurs. Grade I nystagmus occurs only when the eyes are directed toward the fast component; Grade II occurs when the eyes are in their primary position; Grade III occurs even when the eyes are directed toward the slow component.

**Effects/Prognosis:**
Reduced visual acuity is caused by the inability to maintain a steady fixation. Head nodding often accompanies congenital nystagmus. Patients with a null point tend to maximize their vision by assuming a head position in which the nystagmus is least marked. This head-turning to position eyes at a null point is a natural and effective means of improving vision. Certain types of jerky nystagmus (usually Grade I types) show spontaneous improvement in childhood (up to age 10). This type may also be amenable to muscle surgery (essentially a repositioning of muscles to take advantage of the point of least nystagmus or position of relative rest).

Nystagmus reduces vision more at a distance than at close range. Therefore, children with nystagmus may hold objects close to see them and will do better on near visual acuity tests than on a wall chart test. Educationally, children with nystagmus may tend to lose their place in beginning reading instruction and may be helped through the use of a typoscope (card with a rectangular hole to view one word or line at a time) or an underliner (card or strip of paper to “underline” the line being read). As children with nystagmus mature, they seem to need these support devices less often.

**V. Psychiatric/Emotional/Behavioral Disorders**

**Infantile Autism active state: 299.0**

**Description:**
Infantile autism usually becomes evident before 30 months of age and is characterized by a qualitative impairment in verbal and nonverbal communication, imaginative activity, and reciprocal social interactions.

**Effects/Prognosis:**
The most notable symptoms and signs which are consequences of impaired communication and social reciprocity are nondeveloped or poorly developed verbal and nonverbal communication skills, abnormalities in speech patterns, impaired ability to sustain a conversation, and abnormal social play. Stereotypic body movements, a marked need for sameness, narrow interests, and preoccupation with parts of the body are also frequent. Eye contact is minimal or absent. If speech is present, echolalia, pronomial reversal and other idiosyncratic language forms may predominate. Prognosis is guarded. Some children, especially those with language, may become marginally self-sufficient.
better prognosis is associated with higher intelligence, functional speech, and less bizarre symptoms and behavior. Symptoms may often change as children grow older.

**Pervasive Developmental Disorder (PDD): 299.8**

Description:
Pervasive developmental disorder includes autism as its major diagnostic entity. Pervasive developmental disorder *not otherwise specified* refers to children who have autistic features but do not formally qualify for that diagnosis. Some children have a qualitative impairment in the development of reciprocal social interaction and verbal and nonverbal communication but do not have the quantity of symptoms necessary for a diagnosis of autism. These individuals may be diagnosed as having a schizoid personality disorder or Asperger syndrome, which generally refers to a higher functioning form of autism. However, this distinction remains somewhat controversial.

Effects/Prognosis:
Pervasive developmental disorder is a pattern of atypical development that can coexist with mental retardation. Children may be somewhat socially aware but appear to others to be peculiar and eccentric. Prognosis for these individuals would be similar to that of children with autism who have higher intelligence and functional speech.

**Posttraumatic Stress Disorder: 309.81**

Description:
Posttraumatic stress disorder (PTSD) is a psychophysiological syndrome that may follow trauma. It can be either episodic (as in a single extremely stressful event like rape, fire, or flood), or prolonged (wartime events/conditions), or cumulative (so-called "strain trauma," ongoing child abuse, incest, neglect). The symptoms and behavioral manifestations vary with the developmental level of the child.

Effects/Prognosis:
Very young children may present with what appear to be developmental delays and/or behavior and anxiety disorders. Symptoms can be grouped as hyperarousal (anxiety states, especially separation anxiety, hyperactivity, sleep disturbances, hypervigilance), hypoarousal (numbness, inattention, difficulty concentrating, "withdrawn" behavior, avoidant/phobic behavior), and reexperiencing (repetitive reenacting play).

**Emotional Disturbance of Childhood (unspecified): 313.9**

Description:
The categories of emotional disorder that apply to children include three that are specific to children—overanxious disorder, avoidant disorder of childhood, and separation anxiety disorder and a number from the adult nosology—obsessive compulsive disorders, phobic disorders, somatoform disorders, and depressive disorders.

Effects/Prognosis:
The symptoms of emotional disorders in very young children are frequently not as well differentiated as they are in adults. As a result, it is sometimes difficult to categorize an emotional disorder in a child because there is no predominant type of symptom that colors the clinical picture or appears to be specifically related to functional impairment. Many children with emotional disorders grow up to be healthy adults, with the likely
exceptions of those with definite childhood diagnoses of obsessive-compulsive, severe separation anxiety, and severe depressive disorders.

**Attention Deficit Disorder of Children with Hyperactivity: 314.01**

Note: texts dated 1996 and later do not give separate definitions and descriptions for Attention Deficit Disorder (ADD) with and without hyperactivity. The current terminology describes a "predominantly inattentive type", a "predominantly hyperactive/impulsive type", and a "combined type."

**Description:**
Children with attention deficit/hyperactivity disorder (ADHD) commonly exhibit elements of both inattention and hyperactivity-impulsivity in varying degrees and combinations.

**Effects/Prognosis:**
Characteristics of this disorder may include poor ability to attend to a task, heightened distractibility, motoric overactivity, and impulsivity. Children with attention deficit hyperactivity disorder may have difficulty following instructions and sustaining attention, shift rapidly from one uncompleted activity to another, talk excessively, intrude on others, often seem to not listen to what is being said, lose items regularly, and often engage in physically dangerous activities without considering possible consequences. A program that gives structure to a child’s environment decreases the effects of the handicap. Although hyperactivity may be short-lived, other symptoms of ADHD may persist into later life.
Sources


Children’s Hospital of The King’s Daughters Very Low Birthweight – High-Risk Newborn http://www.chkd.org/HealthLibrary/Content.aspx?pageid=PO2424


National Center for Health Statistics (NCHS) and The Centers for Medicare & Medicaid Services (CMS). The International Classification of Diseases, 9th Revision (ICD-9 CM)


Pediatric Database (PEDBASE) http://www.iconda.com/health/pedbase/files


## Appendix B

### Table of Personnel Qualified to Diagnose EIP Eligible Conditions

<table>
<thead>
<tr>
<th>Syndromes/Conditions</th>
<th>Licensed Physician</th>
<th>Nurse Practitioner *</th>
<th>Audiologist</th>
<th>Optometrist</th>
<th>Psychologist (licensed)</th>
<th>Speech-Language Pathologist</th>
<th>Licensed Clinical Social Worker</th>
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<td>Blindness, both eyes</td>
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<td>X</td>
<td>X</td>
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<tr>
<td>Blindness one eye, low vision other eye</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Low vision both eyes (moderate to severe)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Optic nerve coloboma (bilateral), congenital</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
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<tr>
<td>Optic nerve coloboma (bilateral), acquired (Coloboma of optic disc)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Aniridia</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Albinism</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Visual deprivation nystagmus</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychiatric/ Emotional/ Behavioral Disorders</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Infantile Autism active state</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<td></td>
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</table>

* indicates the role of a Nurse Practitioner.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Licensed Physician</th>
<th>Nurse Practitioner *</th>
<th>Audiologist</th>
<th>Optometrist</th>
<th>Psychologist (licensed)</th>
<th>Speech-Language Pathologist</th>
<th>Licensed Clinical Social Worker</th>
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<tbody>
<tr>
<td>Pervasive Developmental Disorder (PDD)</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Posttraumatic Stress Disorder</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Emotional disturbance of childhood</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>(unspecified)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attention deficit disorder w/o hyperactivity</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Attention deficit disorder with hyperactivity</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>

* A nurse practitioner may diagnose these conditions provided that the diagnosis is related to the nurse practitioner’s specialty and competency. Otherwise, the nurse practitioner must refer the child and family to a licensed qualified professional with the training and expertise needed to make an appropriate diagnosis.
## EARLY INTERVENTION PROGRAM
### MULTIDISCIPLINARY EVALUATION SUMMARY FORM

**Child’s Name:**

<table>
<thead>
<tr>
<th>Last</th>
<th>First</th>
<th>Middle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**DOB:** ________/_______/_______

**Date of Evaluation Establishing Eligibility** ________/_______/_______

### NOT ELIGIBLE

Write V79.3 – Not Eligible

Attach evaluation report.

Attach Core/ Supplemental Evaluation Summary Sheets

### ELIGIBLE - BASED ON DIAGNOSED CONDITION

Sufficient to determine eligibility. Submit the following to assist in developing service plan:

1. Indicate Diagnostic Condition in Part A. Attach documentation of diagnosis.
2. Attach Core Evaluation - Data Entry Form, Supplemental Data Entry Form(s), and Narrative Summary of Evaluation.
3. Attach all evaluation reports

### ELIGIBLE - BASED ON DELAY

Submit the following to assist in developing service plan:

1. This page.
2. Core Evaluation-Data Entry Form, Supplemental Evaluation-Data Entry Form(s), and Narrative Summary.
3. Attach all evaluation reports.
4. Indicate ICD 9 Code in Part B.

### A. Diagnosed Physical and Mental Conditions With a High Probability of Developmental Delay

Complete this section only if child is eligible based on diagnosed condition. Attach documentation of diagnosis by physician or clinician.

<table>
<thead>
<tr>
<th>Code</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>270.2</td>
<td>Albinism</td>
</tr>
<tr>
<td>759.89</td>
<td>Angleman’s</td>
</tr>
<tr>
<td>743.45</td>
<td>Aniridia</td>
</tr>
<tr>
<td>728.3</td>
<td>Arthrogryposis</td>
</tr>
<tr>
<td>314.00</td>
<td>Attention Deficit Disorder w/o Hyperactivity</td>
</tr>
<tr>
<td>314.01</td>
<td>Attention Deficit Disorder of Childhood with Hyperactivity</td>
</tr>
<tr>
<td>369.00</td>
<td>Blindness, both eyes</td>
</tr>
<tr>
<td>369.10</td>
<td>Blindness one eye, low vision other eye</td>
</tr>
<tr>
<td>749.0</td>
<td>Cleft Palate</td>
</tr>
<tr>
<td>749.1</td>
<td>Cleft Lip</td>
</tr>
<tr>
<td>749.2</td>
<td>Cleft Palate and Lip</td>
</tr>
<tr>
<td>759.89</td>
<td>CHARGE Syndrome</td>
</tr>
<tr>
<td>389.00</td>
<td>Conductive Hearing Loss unspecified - NOS</td>
</tr>
<tr>
<td>742.3</td>
<td>Congenital Hydrocephalus</td>
</tr>
<tr>
<td>359.0</td>
<td>Congenital Hereditary Muscular Dystrophy</td>
</tr>
<tr>
<td>742.2</td>
<td>Congenital Reduction deformities of brain (Holoprosencephaly/Lissencephaly)</td>
</tr>
<tr>
<td>348.8</td>
<td>Cystic Periventricular Leukomalacia (CVPL)</td>
</tr>
<tr>
<td>315.31</td>
<td>Developmental Language Disorder</td>
</tr>
<tr>
<td>315.4</td>
<td>Dyspraxia Syndrome (Developmental coordination Disorder)</td>
</tr>
<tr>
<td>758.0</td>
<td>Down (Trisomy 21 or 22,G)</td>
</tr>
<tr>
<td>758.2</td>
<td>Edwards’ (Trisomy 18 E3)</td>
</tr>
<tr>
<td>313.9</td>
<td>Emotional Disturbance of Childhood (Unspecified)</td>
</tr>
<tr>
<td>742.0</td>
<td>Encephalocoele</td>
</tr>
<tr>
<td>760.71</td>
<td>Fetal Alcohol</td>
</tr>
<tr>
<td>759.83</td>
<td>Fragile X</td>
</tr>
<tr>
<td>299.0</td>
<td>Infantile Autism active state</td>
</tr>
<tr>
<td>343.9</td>
<td>Infantile Cerebral Palsy (unspecified) - NOS</td>
</tr>
<tr>
<td>345.60</td>
<td>Infantile Spasms w/o intractable epilepsy</td>
</tr>
<tr>
<td>345.61</td>
<td>Infantile Spasms with intractable epilepsy</td>
</tr>
<tr>
<td>772.1</td>
<td>Intraventricular Hemorrhage (grade IV)</td>
</tr>
<tr>
<td>774.7</td>
<td>Kernicterus</td>
</tr>
<tr>
<td>765.01</td>
<td>Less than 500 grams - Low Birth Weight</td>
</tr>
<tr>
<td>765.02</td>
<td>500 - 749 grams - Low Birth weight</td>
</tr>
<tr>
<td>765.03</td>
<td>750-999 grams - Low Birth Weight</td>
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<tr>
<td>755.58</td>
<td>Lobster Claw (Cleft Hand, Congenital)</td>
</tr>
<tr>
<td>369.20</td>
<td>Low vision both eyes (moderate to severe)</td>
</tr>
<tr>
<td>742.1</td>
<td>Microcephalus</td>
</tr>
<tr>
<td>389.2</td>
<td>Mixed conductive and sensorineural hearing loss</td>
</tr>
<tr>
<td>742.4</td>
<td>Multiple anomalies of brain - NOS</td>
</tr>
<tr>
<td>377.23</td>
<td>Optic nerve coloboma (bilateral), Acquired Congenital</td>
</tr>
<tr>
<td>743.57</td>
<td>Optic nerve coloboma (bilateral), Congenital</td>
</tr>
<tr>
<td>359.8</td>
<td>Other Myopathies</td>
</tr>
<tr>
<td>758.1</td>
<td>Patau's (Trisomy 13 D 1)</td>
</tr>
<tr>
<td>299.8</td>
<td>Pervasive Developmental Disorder (PDD)</td>
</tr>
<tr>
<td>755.4</td>
<td>Phocomelia (absence of limb)</td>
</tr>
<tr>
<td>759.81</td>
<td>Prader-willi</td>
</tr>
<tr>
<td>309.81</td>
<td>Posttraumatic Stress Disorder</td>
</tr>
<tr>
<td>362.21</td>
<td>Retinopathy of prematurity (Retrorenal fibroplasia) (grades 4 &amp; 5)</td>
</tr>
<tr>
<td>389.1</td>
<td>Sensorineural Hearing Loss - NOS</td>
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<tr>
<td>741.0</td>
<td>Spina Bifida with hydrocephalus</td>
</tr>
<tr>
<td>741.9</td>
<td>Spina Bifida w/o hydrocephalus</td>
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<tr>
<td>952.9</td>
<td>Spinal Cord Injury, NOS</td>
</tr>
<tr>
<td>744.00</td>
<td>Unspecified congenital anomaly of the ear with Impairment of hearing</td>
</tr>
<tr>
<td>379.53</td>
<td>Visual deprivation nystagmus</td>
</tr>
<tr>
<td>335.0</td>
<td>Werdnig-Hoffmann Syndrome (Infantile Spinal Muscular Dystrophy)</td>
</tr>
</tbody>
</table>

### B. Indicate Diagnostic Condition and ICD Code(s) below if eligible due to delay or if different from above.

1. ____________________________________________
2. ____________________________________________
## EARLY INTERVENTION PROGRAM
### CORE EVALUATION SUMMARY FORM

**INSTRUCTIONS:** This form must be accompanied by Multidisciplinary Evaluation Summary form, Supplemental Evaluation Data Entry form (when applicable), and Narrative Summary. Please print or type.

<table>
<thead>
<tr>
<th>Name of Child:</th>
<th>Last:</th>
<th>Middle</th>
<th>First:</th>
</tr>
</thead>
<tbody>
<tr>
<td>DOB:</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>EI Evaluator</th>
<th>Name:</th>
<th>Provider ID#:</th>
<th>Contact Person:</th>
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</thead>
<tbody>
<tr>
<td>Phone#:</td>
<td></td>
<td>Fax#:</td>
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</tbody>
</table>

### Core Evaluation - Individuals Involved

<table>
<thead>
<tr>
<th>Name:</th>
<th>Specialty:</th>
<th>Instrument(s):</th>
</tr>
</thead>
<tbody>
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</table>

[ ] Check if Bilingual Evaluation Performed

Language:

Summary of evaluation must be translated.

Dates of Core: From / To / |

<table>
<thead>
<tr>
<th>Name:</th>
<th>Specialty:</th>
<th>Instrument(s):</th>
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<tbody>
<tr>
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</tbody>
</table>

[ ] Family Assessment Offered & Refused

[ ] Family Assessment Completed and Attached

### Disciplines involved in Core Evaluation

<table>
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<tr>
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</thead>
<tbody>
<tr>
<td></td>
<td>Audiologist</td>
<td>Other Physician</td>
<td>Nurse</td>
<td>Physician Assistant</td>
<td>Nurse Practitioner</td>
<td>Psychologist</td>
<td>Nutritionist</td>
<td>Social Worker</td>
<td>Occupational Therapist</td>
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</tbody>
</table>

### Method

P - Informed Clinical Opinion

T - Standardized Test

(1) Developmental Status Codes

A - No Delay (development within acceptable ranges)

B - 2.0+ SD below the mean (sufficient alone for eligibility)

C - 1.5+SD below the mean (similar delay in another functional area needed to establish eligibility)

D - 12 month delay (sufficient alone for eligibility)

F - 33% or more delay (sufficient alone for eligibility)

G - 25% or more delay (similar delay in another functional area needed to establish eligibility)

### EVALUATION SUMMARY

<table>
<thead>
<tr>
<th>Functional Area</th>
<th>Developmental Status</th>
<th>Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adaptive</td>
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</tr>
<tr>
<td>Cognitive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Communication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social/Emotional</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical</td>
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</tr>
</tbody>
</table>

Diagnosed Condition(s) | ICD 9 Code
**Name of Child:**_____________________________________________________________________
**DOB:**_______/_______/_______

**EI Evaluator**
**Name:**________________________________________
**Provider ID#:___________________________________
**Contact Person:**_________________________________
**Phone : (_____)________________**
**Fax : (_____)________________**

**Supplemental Evaluation**
[ ] Bilingual Evaluation  Evaluation Type:_______
[ ] Physician  [ ] Non-Physician
**Dates:** From:____/____/____ To:____/____/____
**Name:**__________________________________
**Discipline:**_______________________________

**Functional Area** | **Developmental Status (1)** | **Method (2)** | **Functional Area** | **Developmental Status (1)** | **Method (2)**
--- | --- | --- | --- | --- | ---

**Supplemental Evaluation**
[ ] Bilingual Evaluation  Evaluation Type:_______
[ ] Physician  [ ] Non-Physician
**Dates:** From:____/____/____ To:____/____/____
**Name:**__________________________________
**Discipline:**_______________________________

**Functional Area** | **Developmental Status (1)** | **Method (2)** | **Functional Area** | **Developmental Status (1)** | **Method (2)**
--- | --- | --- | --- | --- | ---

**Developmental Status Codes**
A - No Delay (development within acceptable ranges)
B - 2.0+ SD Below the mean (sufficient alone for eligibility)
C - 1.5+SD Below the mean (similar delay in another functional area needed to establish eligibility)
D - 12 month delay (sufficient alone for eligibility)
F - 33% or more delay (sufficient alone for eligibility)
G - 25% or more delay (similar delay in another functional area needed to establish eligibility)

**Method of Determination**
P - Informed Clinical Opinion  T - Standardized Test

**Evaluation Type Code**
A - Assistive Technology  J - Psychological Services
B - Audiology  L - Social Work
F - Nursing  M - Special Instruction
G - Nutrition  N - Speech and Language
H - Occupational Therapy  Q - Vision
I - Physical Therapy

**List Diagnosis and ICD Numbers:**
1________________________________________________ 2_______________________________________