

CEREBRAL PALSY—DON'T DELAY

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Cerebral palsy (CP) is the most severe physical disability within the spectrum of developmental delay. CP is an umbrella term describing a group of motor disorders, accompanied by many associated impairments. The disability is a result of injuries to the developing brain occurring any time from the first trimester of pregnancy through to early childhood. However, for the great majority, their full etiological causal pathway remains unclear. It is important to discriminate as early as possible between: (a) mild or nonspecific motor delay, (b) developmental coordination disorder, (c) syndromes, (d) metabolic and progressive conditions, and (e) CP with its various motor types and distributions. The most promising predictive tool for CP is the general movements assessment, which assesses the quality of spontaneous movements of infants in the first 4 months of life. We propose a change in diagnostic practice. We recommend a shift away from referral for intervention following a formal (most often late) description of CP, to one of referral for intervention which occurs immediately once an infant is considered "at risk" of CP.

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INTRODUCTION

Global developmental delay is an umbrella term that describes two or more delays in the area of speech and language, social and emotional, cognitive and motor development. Children with cerebral palsy (CP) often fall under the umbrella of global developmental delay, but CP cannot be considered "delay," as children do not "grow out of it." Health professionals need to understand what clinical features distinguish CP from other motor disorders, so the most effective interventions can be commenced earlier. The American Academy of Pediatrics have developed a policy for the surveillance and screening of developmental disorders (Council on Children with disabilities et al., 2006), however this paper focusses specifically on CP. The objectives of this review are fivefold:

1. Describe the nature of CP and what makes it different to other motor or learning disorders.
2. Outline the prevalence of CP.
3. Determine who is at high risk of CP, what are the predictors and early signs?
4. Identify tools that help clinicians to accurately predict CP.

5. Present an evidence based algorithmic approach to recognizing CP and developing intervention plans.

In the early months of life, global developmental delay and CP present similarly, if delayed, acquisition of developmental milestones is the only comparator. It is the movement disorders (e.g., spasticity and dystonia), the level of functional impairment, and the associated impairments that set CP apart from other milder motor disorders or learning disorders such as developmental coordination disorder (DCD). DCD is less severe and 25 times more common than CP affecting ~5–6% the population and current practice is not to diagnose before the age of 5. As a result, the diagnosis of CP is often delayed while the possibility of DCD is explored.

DCD is primarily a learning problem where children can achieve normal movement patterns and skills but have problems with learning and planning the movements. CP conversely is a physical disorder, where children are not able to achieve the normal movement patterns and the primary problem is motoric not learning, although deficits in learning may compound the motor problem.

DCD is used to refer to children who fulfill a certain criteria; poor motor performance which significantly interferes with activities of daily living which are not explained by any medical, neurological, or psychosocial condition. Thus a child with CP whose motor disability is neurological cannot have a diagnosis of DCD [Blank et al., 2011]. The physical disability of CP is life-long whilst DCD is more apparent in the window where the child is learning key motor skills for example, catching a ball, dressing independently, and handwriting.

WHAT IS CEREBRAL PALSY?

CP is an umbrella term which "describes a group of disorders of the development of movement and posture, causing activity limitations, which are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain.

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Table 1. Classification by Motor Type

		ACPR ^a + Reid, 2011a
Spasticity:	Overactive muscles that display a velocity-dependent resistance to stretch. Spasticity can cause secondary impairments such as loss of muscle length, joint dislocation and pain.	85 – 91%
Dyskinesia:	Dyskinesia is either athetosis or dystonia. Athetoid CP is hypotonic with hyperkinesia characterized by involuntary writhing-stormy movement and can co-occur with chorea. In contrast, dystonic CP is hypokinetic, involving involuntary, abnormal twisting postures or repetitive movements with hypertonia. Tone is typically fluctuating.	4 – 7%
Ataxia:	Ataxia results in tremors with a shaky quality. Ataxic CP involves a loss of muscular coordination where movements have abnormal force, rhythm, and accuracy.	4 – 6%
Hypotonia:	Pure, generalized hypotonia (decreased muscle tone) is the least common CP motor-type. Some argue that pure hypotonia should not even be considered a cerebral palsy sub-type.	2%

^aAustralian Cerebral Palsy Register.

The motor disorders of CP are often accompanied by disturbances of sensation, cognition, communication, perception, and/or behavior, and/or by a seizure disorder” [Bax et al., 2005]. This most recent definition acknowledges the complexity of the condition and the impact of the associated impairments.

What are the Fundamental Facts We Know About Cerebral Palsy?

Classification of cerebral palsy guides intervention decision making

CP is a heterogeneous condition, and to elucidate prognosis and guide selection of the most appropriate interventions (e.g., constraint induced movement therapy for hemiplegia and selective dorsal rhizotomy for diplegia) three major classifications are applied; motor-type, topography, and function. Clinicians often remark that a child may have two or three different descriptions of their CP within one medical file, evidencing the poor reliability of these traditional classification systems. Tables 1 and 2 outline the traditional motor types and topographies of CP and the proportions of a CP population with each type. In this paper, we refer to the Australian Cerebral Palsy Register (ACPR) when reporting rates and for international comparisons the Swedish Register and a study by Reid et al. [2011a] where registers throughout the world are compared.

To solve the problem of low inter-rater (and sometimes intra-rater) reliability

when identifying topographical subtype, the Surveillance of Cerebral Palsy Europe [SCPE, 2000] has recommended that traditional topographies be combined into two easily definable topographies: Unilateral (one side of the body), Bilateral (both sides of the body). The ACPR instead applies a limb by limb coding using the Australian Spasticity Assessment Scale (ASAS) [Love, 2007]. The ASAS scores the muscles’ response to rapid passive movement without the subjectivity and wording ambiguities of the modified Tardieu and Ashworth scales [Mutlu et al., 2008]. Nonspastic motor types are also coded, resulting in a “stick figure diagram” of motor impairment, which provides an objective picture of the CP. Figure 1 presents the CP description form. The descriptive form is also clinically useful for treatment decision-making, such as pharmacological options

and contracture management. The ASAS is currently undergoing further reliability studies, but it is freely available for use along with the description of CP form: http://www.kemh.health.wa.gov.au/services/register_developmental_anomalies/documents/CP%20Description%20Form%20-%20WARD%20website.pdf.

The gold standard tool for reliably describing motor function in CP is the gross motor function classification system (GMFCS) [Palisano et al., 1997]. GMFCS provides a common language that conjures up a “picture” of a child with CP. GMFCS is a five level classification system of gross motor function in people with CP. The classification is based on the person’s ability to self initiate movement with a focus on sitting, transferring, and mobilizing [Palisano et al., 1997]. Different classification descriptions exist at different age groups. Table 3 summarizes the system for 2–4-year olds, to coincide with the most common time of recognition and the proportion in a CP population with each level of GMFCS.

It should be noted that whilst the GMFCS classification can be applied to infants, about 40% change classification levels by age 2. After 2 years, the classification system is stable and thus GMFCS reassessment is recommended after age 2 [Gorter et al., 2008]. This is clinically and diagnostically very important, because parents are anxious to learn early about the severity of their child’s condition for future planning but in reality the most accurate description of function and severity can only be given at 2 years.

The presence of associated impairments and functional limitations affects the child’s outcome

For many children with CP, it is not just a physical disability. When seeking to prognosticate the severity of

Table 2. Classification by Topography

		ACPR ^a
Hemiplegia:	Hemiplegia/monoplegia is the involvement of one side of the body. The upper limb is usually more affected than the lower limb. Strong early hand preference or hand disregard is sometimes the first sign of a problem.	38%
Diplegia:	Diplegia is where both the legs are affected and are more affected than the upper limbs.	36%
Quadriplegia (Tetraplegia)	Quadriplegia refers to the presence of spasticity in all four limbs; where the affect on the arms is equal or more than the legs. Trunk and oro-facial involvement is also to be expected. In rare cases, one limb is spared and this is referred to as triplegia.	26%

^aAustralian Cerebral Palsy Register.

Table 3. Classification by Gross Motor Function at 2–4 Years

		ACPR ^a
Level I:	Floor sits independently, hands-free. Walks without assistive devices.	32%
Level II:	Floor sits independently, hands-free with balance affected. Walks using an assistive mobility device.	27%
Level III:	Floor sits using w-sitting. Walks short distances indoors using a hand-held mobility device with assistance.	12%
Level IV:	Floor sits when placed, uses hands for balance. Rolls, creeps or crawls for short distances.	14%
Level V:	Unable to sit independently. No form of independent mobility.	15%

^aProportion in Australia with each level of GMFCS.

CP and determine intervention plans, assessment of associated impairments must also occur. The likelihood and severity of associated impairments increase with the severity of motor impairment [Himmelman et al., 2006; Odding et al., 2006]. Some have reported that associated impairments impact more on function and quality of life than the motor impairment [Himmelman and Uvebrant, 2011]. A meta-analysis of CP registers calculated the overall rates of associated impairments and functional limitations in the CP population to be: three in four are in pain; one in two have an intellectual disability; one in three cannot walk; one in three have a hip displacement; one in four cannot talk; one in four have epilepsy; one in four have a behavior disorder; one in four have bladder control problems; one in five have a sleep disorder; one in five dribble; 1 in 10 are blind; 1 in 15 are tube fed; and 1 in 25 are deaf [Novak et al., in press]. Many will have a number of these impairments, and the presence of these impairments complicates therapy, decreases health status and quality of life for the individual and their family, and increases costs for the family and to society. The associated impairments of CP will now be discussed briefly.

Epilepsy. Epilepsy can potentially severely limit the quality of life for the person with CP and their family, and adults with CP and epilepsy are less likely to find employment [Michelsen et al., 2005]. Epilepsy occurs in 30% of individuals with CP [Arnaud et al., 2008; ACPR Group, 2009]. In 2% of individuals with CP, their epilepsy will be resolved by the time they turn 5 years of age [ACPR Group, 2009]. For those whose seizures are not resolved, epilepsy is a lifelong condition. Rates of epilepsy are higher in those with: spasticity born

at term (48%) compared with preterm (28%); bilateral CP (34–87%) compared with unilateral (23%); and those with intellectual impairment (61%) compared with no intellectual impairment (19%) [Carlsson et al., 2003; Wichers et al., 2005; Himmelman et al., 2006].

Intellectual impairment. Intellectual impairment can be defined by low general intellectual functioning as measured by IQ scores, in combination with difficulties with adaptive behavior, all manifesting before the age of 18. Practically, this means that people with an intellectual impairment have memory deficits, difficulty reasoning, learning new skills, attending and organizing information. 50% of individuals with CP have an intellectual impairment and between 20 and 30% [Jarvis et al., 2005; McManus et al., 2006] have a severe intellectual impairment. Formal assessment of intellect is essential (but at times difficult) for an individual with CP.

Communication. Communication disability can have a major impact on the individual with CP and their family. Impairment in this domain can impact on both understanding of language and expression. For individuals who have severe communication impairment, social isolation and poor self-esteem can result. Between 20 and 30% of people with CP are nonverbal which means that systems to support other forms of communication are required [Arnaud et al., 2008; ACPR Group, 2009; Andersen et al., 2010; Parkes et al., 2010]. They are more likely to be nonverbal if they are non-ambulatory (GMFCS IV–V, 57%) compared to those who are able to walk (GMFCS I–III, 4%) [Shevell et al., 2009]. Augmentative and alternative communication (AAC) systems, which can range from low/light technology systems such as signing or use of alphabet charts to high

technology systems such as speech generating devices, may be used to communicate. It is a fundamental human right to have the opportunity to communicate; however, high technology AAC systems are expensive, requiring wait listing and for some individuals will mean that they are unable to access systems that would support them to communicate.

Vision. Vision impairments can range from mild requiring glasses, to functionally blind. About 5–12% of individuals with CP have a severe impairment, or are functionally blind [McManus et al., 2006; ACPR Group, 2009]. Another 30% will have a mild to moderate vision impairment.

Hearing. Hearing impairments can also range from a mild impairment to bilateral deafness. Bilateral deafness occurs in 2% of people with CP while other hearing impairments occur in a further 10% [Surman et al., 2006; ACPR Group, 2009]. Assessment of vision and hearing in children with CP should be thorough and done early, as it can impact greatly on their ability to learn and achieve milestones.

Other. Other impairments strongly associated with CP are hip dislocation (8%), displacement (27–35%) [Hagglund et al., 2005; Soo et al., 2006] and spine deformities, sleep disorders (23%) [Newman et al., 2006], pain (70%) [Jahnsen et al., 2004; Arnaud et al., 2008], eating (8% tube fed) [Shevell et al., 2009; Sigurdardottir and Vik, 2011], excessive drooling (22%) [Parkes et al., 2010], bladder and bowel control complaints (24%) [Roijen et al., 2001], and behavior difficulties (26%) [Parkes et al., 2008]. These less well-understood impairments are more likely to occur with bilateral CP and intellectual impairment.

CP is the most common physical disability in childhood with prevalence unchanged for 60 years

The overall prevalence of CP is ~0.2% of the population (i.e., 1 in 500) in developed countries. As can be seen by a projected age distribution of one state in Australia (Fig. 2), even though the injury responsible for CP occurs in the developing brain, it is a lifelong condition, with most patients having a normal life expectancy. In reality, CP is not just a condition of childhood.

The true incidence of CP cannot be estimated as there are a proportion of infants who die in the intrapartum, neonatal and infant period, who had brain lesions that may or may not have met

DESCRIPTION OF CEREBRAL PALSY Part I: MOTOR IMPAIRMENTS

Child's name: _____ DOB: _____ Examining clinician: _____ Date: _____

1. Is there spastic tone in one or more limbs?

Yes ☐ No ☐

2. Is muscle tone varying?

Yes ☐ No ☐

3. Are there signs of ataxia?

Yes ☐ No ☐

Is there generalised hypotonia with increased reflexes?

Yes ☐ No ☐

Stick Figure 1

Instructions for completing Stick figure 1 above:

Axial tone: Hypotonic = ↓, Hypertonic = ↑, Fluctuating = ↕, Normal = N

Neck tone: (same as Axial tone)

Face tone: (same as Axial tone)

Limb tone: Highest Australian Spasticity Assessment score in that limb (see scoring criteria overleaf)

Stick Figure 2a

Stick Figure 2b

Instructions for completing Stick figures 2a and 2b above:

Please tick boxes and/or circles where signs are present

Please number tone/movement abnormalities present in this child in order of predominance (1 = most predominant or only abnormality)

☐ Spasticity

☐ Dystonia

☐ Athetosis

☐ Ataxia

☐ Generalised Hypotonia

Please describe CP type and severity in words as you would write in the medical record:

Form designed for the Australian Cerebral Palsy Register - 7 July 2008

Figure 1 Cerebral palsy description form. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

the criteria for CP. It has been suggested therefore that the closest rate to incidence (for CP) is prevalence of neonatal survivors (NNS). Western Australia (WA) is one register that reports in this manner, and is also one of the longest running CP Registers in the world. CP is mandatorily reported in WA, therefore it is assumed that this register has as close to a total population cohort as is possible. WA's CP rates reported in 2006 are 2.78/1,000 NNS increasing to 3.9/1,000 when post-neonatal CP is taken into account [Blair and Watson, 2006; Watson et al., 2006]. NNS are important when rates are reported by gestational age stratum. The lower the gestational age stratum, the more rates differ between NNS and live births. It is particularly important for those at the youngest gestational ages. When reporting rates in the birth years 2005 and 2006 for those born between 20 and 27 weeks in WA, the rate per 1,000 NNS was 72 (95% CI 32–110) compared to

live births 51 (95% CI 24–79) [Watson, 2012, personal communication]. If neonatal deaths are not taken into account, live births give a misleading lower rate. In term births (37+ weeks), where the rate of intrapartum/neonatal death is proportionally much less, the difference between NNS 1.7 (95% CI 1.4–2.1) and live births 1.7 (95% CI 1.4–2.0) becomes inconsequential. Despite this denominator being the most accurate, for comparison live births are the most widely used denominator.

Estimates of prevalence throughout the world vary depending on the methodology of "count," percentage ascertained and variations in selection criteria. CP Registers have identified rates ranging between 1.4 and 2.77/1,000 live births; surveillance programs range between 2.1 and 3.6/1,000 live births; and cross-sectional surveys range between 1.05 and 4.1/1,000 live births. The two largest data sets, the ACPR and the SCPE both have an overall

birth prevalence of 2/1,000 live births. In developing countries, it is thought that incidence is higher as the public health measures that help prevent some CP cases are not freely available in developing countries [Blair and Watson, 2006]. All data sets across the world agree there is a higher proportion of boys diagnosed with CP. Although CP is found across all socio-economic classes, there is a clear association between low birth weight and low socio-economic status, and in normal birth weight ranges, rates of CP are 2.42/1,000 live births for those in the lowest socio-economic groups, compared to 1.29/1,000 for the most affluent groups.

The overall rate of 2/1,000 has been fairly stable over the last 60 years in contrast to the dramatic falls in perinatal mortality rates. However, there have been some trends in gestational age stratum, shown in Figure 3. Rates in the extremely and very low

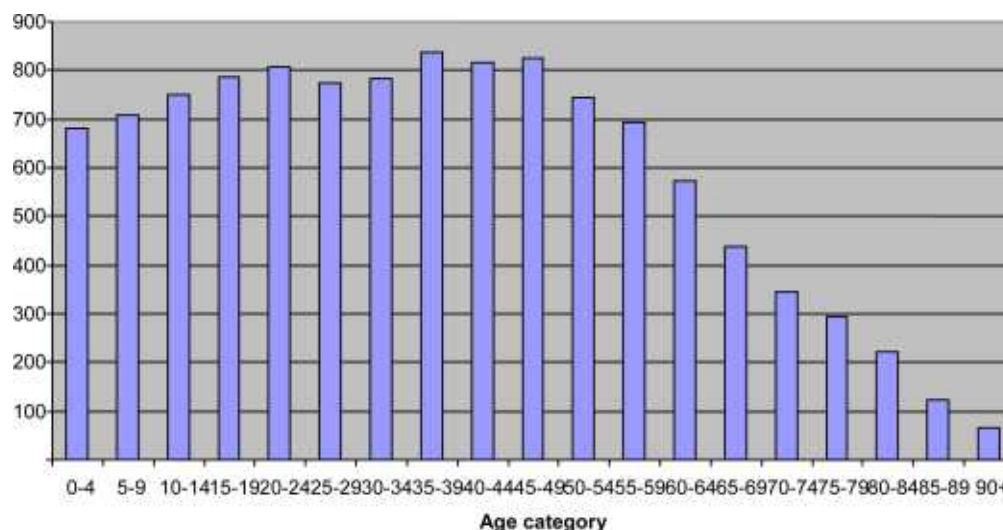


Figure 2 Estimated number of people living with CP in New South Wales, Australia. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

gestational groups rose during the 1980s, but are now trending down. Moderately premature infants' rates have decreased slightly, while in term infants the rates are unchanged [Blair et al., 2001; Watson et al., 2006]. Because the majority (>73%) of infants are born over 32 weeks gestational age, the increases and decreases in the extremely and very preterm groups have made little difference to the overall rate.

Identification of infants "at-risk of cerebral palsy" is possible; assessment and screening should follow

Since there are no identifiable biomarkers to accurately predict CP, and clinical risk factors only identify subpopulations of infants at risk [McAdams and Juul, 2011], understanding the term "causal pathways" is important. CP aetiologies are described in terms of causal pathways, as there is very rarely one specific cause of brain damage severe enough to cause CP. Much research has been published that attempts to discern the risk factors that lie on one or more causal pathways to CP. What researchers are beginning to realize is how little is known about how these risk factors interact on causal pathways. Risk factors can be described according to when they occur or when they are identified. The following examples have been identified for CP:

- Prior to conception: Previous gynecological history of stillbirths/multiple miscarriages/neonatal death/premature birth, family history of CP and other genetic predispositions, maternal diagnoses, for

example, intellectual impairment, epilepsy and low socioeconomic status.

- Early pregnancy: Infection, birth defects, multiple births, male gender, and other genetic predispositions.
- During pregnancy: Maternal disease, for example, thyroid disorders, pregnancy complications, for example, preeclampsia and bleeds in the second and third trimester, infection and inflammation, intrauterine growth restriction (IUGR), placental abnormalities and other precursors to premature birth.
- Around the time of birth and the neonatal period: An acute intrapartum hypoxic event, stroke, seizures, hypoglycemia, jaundice, and infection.
- Postnatal period: Infections, accidental and nonaccidental injuries, stroke both spontaneous and following surgery.

The rate of CP in neonatal survivors varies significantly with level of risk at birth. To describe the risk of developing CP, infants have been separated into three distinct groups shown in Figure 4: (1) premature infants (30–40% of all CP); (2) term born infants who shortly after birth have neonatal encephalopathy (NE), a clinically defined syndrome of disordered neonatal brain function (15–20% of all CP); and (3) term born "healthy" infants, who do not require special care in the neonatal period (40–50% of all CP) and do not appear to have identifiable risk factors at birth [Badawi et al., 2005; Wu et al., 2006; McIntyre et al., 2011]. *Premature infants.* When considering which babies are at risk of CP, preterm infants commonly come to mind. The risk of CP increases as gestational age

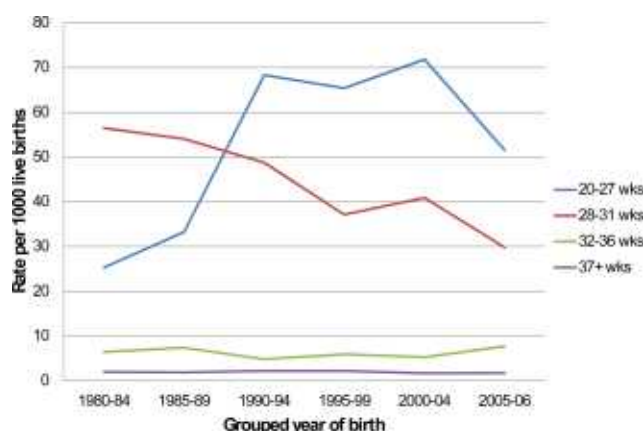


Figure 3 Gestational age specific rates/1,000 live births in WA, 1980–2006. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

decreases, therefore babies born at 36 weeks' gestation are at much lower risk than those born at 24 weeks. As a result, rates in premature infants range between 3 and 80/1,000 neonatal survivors, reflecting the wide variation in levels of risk across premature gestations. Premature infants constitute up to 40% of infants who develop CP [Kirby et al., 2011]. So why are premature infants at increased risk of CP, and which ones are at the highest risk?

The group of preterm infants can be separated according to gestational age, with the first subgroup being extreme prematurity, generally considered less than 28 weeks' gestation. There is much data in the literature which depicts the outcomes of extremely premature infants and much research has been conducted in this age group [Hoon and Faria, 2010; Reid et al., 2011b]. In the 1970s and 1980s, the frequency of CP in this gestational age group increased. This was attributed to the increasing survival of extremely preterm infants and their predilection to germinal matrix hemorrhage and periventricular leukomalacia (PVL) [Stanley and Watson, 1992; Hagberg et al., 1996]. Evidence from population-based samples in Europe, Australia and the United States, and analyses from CP Registers in Australia and Europe describing trends in prevalence, subtypes, and severity, suggest that this rise in frequency of CP in extremely preterm infants has reached its peak and is now decreasing [SCPE, 2000; Reid et al., 2011b; Watson, 2012, personal communication]. Up to 10% of extremely preterm infants (variations in reports exist from as low as 3–10%) and up to 5% of infants between 28 and 31 weeks gestation will be described as having CP [Himpens et al., 2008; Watson, 2012, personal communication].

Practice point. Mothers whose labor is imminent (and prior to 30 weeks gestation) should now be offered magnesium sulphate for neuroprotection of their child. Meta analyses have shown that CP can be reduced by 30% for infants under 30 weeks gestation [Crowther et al., 2002].

CP Registers in Europe report that this trend for decreasing rates continues into the group of late preterm infants (32–36 weeks' gestation or 1,500–2,499 g) [Andersen et al., 2011]. The overall prevalence of CP in these children had dropped from 12.2 per 1,000 live births in 1983 to 4.5 per 1,000 in 1997. There is conflicting evidence in Australia, with the rate being

maintained at between 5 and 7/1,000 live births since the early 1980s [Watson et al., 2006].

Cerebral lesions in particular PVL, intraventricular hemorrhage (IVH) and intracranial hemorrhage (ICH) grade III and IV, are the most important predictors of CP in very preterm infants [Tran et al., 2005; Beaino et al., 2010; Himpens et al., 2010]. In particular, PVL lesions in the corona radiata above the posterior limb of the internal capsule (PLIC) observed in coronal sections have been used to accurately predict motor prognosis [Nanba et al., 2007]. The presence of lesions in this region was highly predictive of CP (GMFCS 1 or higher) with sensitivity 100% and specificity 97%. A study by Himpens et al. [2010] that investigated the predictive value of ultrasound in brain injury found that deep grey matter lesions are a significant predictor for severe versus mild and moderate CP (OR = 6), and that cerebral infarction and hemorrhage grade IV are strong predictors of unilateral spastic CP versus bilateral spastic CP (OR = 49 and 24, respectively, $P < 0.001$).

Recently, there has been increasing interest in and evidence regarding the possible effects of intrauterine infection or inflammation early in the postnatal course, leading to CP. Carlo et al. [2011] recently argued that a late prenatal and/or early neonatal exposure to inflammation may predispose infants to neurodevelopmental impairment. Wu and Colford [2000] also found that clinical chorioamnionitis was associated with an increase in CP in preterm infants (OR = 1.9) and term infants (OR = 4.7).

Transient hypothyroxinaemia, bronchopulmonary dysplasia (BPD), and necrotizing enterocolitis have also been associated with premature birth and a later description of CP. A recent study of 1,047 preterm infants (<28 weeks) demonstrated that while all infants with BPD had a higher risk of CP those who were mechanically ventilated until 36 weeks PMA had at least a fourfold increased risk of CP [Van Marter et al., 2011]. In addition, preterm infants who have had surgery to repair a patent ductus arteriosus, or who required home oxygen have also been identified as at increased risk of CP [Tran et al., 2005].

Practice point. Infants born premature are at high risk of CP if they have abnormal cerebral imaging and a more complex course. These infants should

receive a general movements (GM) assessment before term equivalent age, and be referred to active surveillance and early intervention when they leave the hospital. (see Pathway A Figure 5, to be discussed in the following section).

Term infants with and without neonatal encephalopathy

The overall rate of CP for term infants has been consistently 1.4–1.7/1,000 live births over the past 30 years [Watson et al., 2006; Himmelmann et al., 2010]. Multiple births born at term are at four times the risk of CP than singletons born at term. The risk rises again for surviving twins after the death of a cotwin [Pharoah, 2006]. Risk factors associated with the development of CP in the term population also include congenital malformations, maternal age over 35 years, chorioamnionitis, preeclampsia, placental abnormalities, meconium aspiration syndrome, IUGR, transient metabolic abnormalities, respiratory distress syndrome, neonatal infections and seizures. [Shankaran, 2008; McIntyre et al., 2012]. One of the most well known risk factors for term-born infants is NE.

The second piece of the pie (Fig. 4), with a well-recognized predilection to develop CP are term or near term infants with NE. For term born infants with NE, the rate of CP is between 100 and 125/1,000 neonatal survivors, and those born with severe NE are at the highest risk of CP of all infants. Infants with moderate to severe (Sarnat Stage 2 or 3) NE account for one in four cases of term CP [Badawi et al., 2005]. Kurinczuk et al. [2010] report an incidence of NE between 2.5 and 3.5 per 1,000 live births and that ~30% of cases in developed countries are associated with evidence of an acute intrapartum hypoxic event. These include sentinel birth events that are also rare but important risk factors for CP in term infants, such as placental abruption, cord prolapse, severe intrapartum hemorrhage, severe shoulder dystocia, and a tight nuchal cord. It is estimated that up to 8% of CP is attributable to an acute intrapartum event with moderate to severe NE [Blair and Stanley, 1997].

Practice point. Infants with moderate to severe NE following an acute intrapartum event benefit from hypothermia. This intervention prevents CP in one out of eight of those treated [Jacobs and Tarnow-Mordi, 2010]. A number of

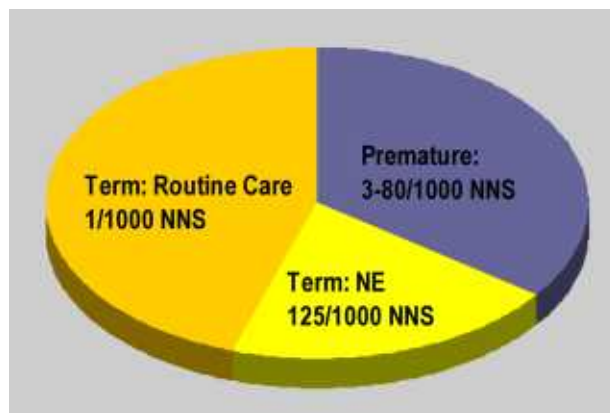


Figure 4 Rate of CP in NeoNatal Survivors. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

adjuvant therapies to help those that do not respond to cooling alone are currently in animal model and phase 1 neonatal studies, for example, erythropoietin, melatonin, xenon, and topiramate [Gonzalez and Ferriero, 2009].

In term infants with moderate to severe NE, imaging showing basal ganglia/thalamus injury has a positive predictive value for CP of 88% [de Vries et al., 2011]. In a study of 173 term infants with NE, the basal ganglia/thalamus pattern of injury was associated with the most severe motor and cognitive outcomes at 30 months [Miller et al., 2005].

Practice point. Term infants with moderate to severe NE and a basal ganglia/thalamus injury should be automatically described as “At high risk,” and go straight to Pathway B (Figure 5). They should receive a GMs Assessment, be referred to active surveillance and early intervention when they leave the hospital.

The remaining infants with NE that go on to be described as having CP have antenatal risks such as IUGR, intrauterine infection, metabolic abnormalities, syndromes, and birth defects [Badawi et al., 1998; Kurinczuk et al., 2010]. Perinatal arterial stroke occurs in ~1.7/100,000 live births. In the newborn period, it can also result in NE, but the majority of these infants present after the immediate neonatal period with seizures or hemiparesis. Mothers with preeclampsia and infants who have IUGR are at risk of perinatal arterial stroke [Shankaran, 2008]. Stroke with abnormalities involving the cerebral peduncle are also highly predictive of CP PPV 78% [de Vries et al., 2011].

Practice point. Infants with a cerebral birth defect, or stroke with involvement

of the cerebral peduncle should be identified as “at risk” of CP and should join Pathway B (Figure 5) at “assessment for CP.”

The risk of developing CP in term infants who have received routine care at birth, the third group of infants who go on to develop CP, is ~1/1,000 neonatal survivors and these infants are at the lowest risk. However, they represent 45% of all infants with CP and numerically comprise the largest group (Fig. 4). Why do these apparently “neurologically normal” children at birth develop CP, and can we identify them earlier so they can have access to active surveillance and early intervention?

From a total population case control study in Western Australia, McIntyre et al. [2011] compared the clinical descriptions of 295 term infants with CP with 442 term control infants none of which required special care. They identified six independent predictors of CP in the neonatal period: abnormal fontanelle OR 4.4 (95% CI 0.8–23); abnormal tone OR 7.3 (95% CI 2–26.8); birth defects identifiable in the newborn period OR 5.2 (95% CI 2.4–10); ventilatory assistance restricted to the labor room only OR 2.9 (95% CI 2.2–12); abnormal consciousness referred to irritability and lethargy, but none were comatose OR 3.7 (95% CI 2–7); and in the small group with abnormal temperature regulation temperature was down or fluctuating, not high OR 4.1 (95% CI 1.2–14). A number of these predictors are reminiscent of criteria for mild NE, and the presence of two or more of these factors yielded a high specificity (99%), but low sensitivity (14%) for CP. This is not surprising considering the unknown etiology of this group of infants. Of this low risk group who had CP, 58% did

not have any of these neonatal factors, yet 60% of these infants had moderate to severe CP.

This is not the first time a finding like this has been reported. The National Collaborative Perinatal Project reported that most children with CP did not derive from groups at high risk (low Apgar scores, or the presence of neonatal signs). About 43% were examined and classified as “neurologically normal” in the neonatal period and concluded that a large proportion of CP cases remain unexplained [Nelson and Ellenberg, 1986; Ellenberg and Nelson, 1988]. Earlier still, in 1970, Eva Alberman attempted to model what were at that time the three most important risks around birth: (1) parity >4; (2) abnormal method of delivery—breech, face or shoulder delivery, internal version, or delivery by an untrained person; and (3) neonatal illness in the 1st week of life—convulsions, cyanotic attacks, cerebral signs, hypothermia, jaundice, Rh incompatibility, or serious illness. Infants were at the highest risk of disability when all three of these risks were apparent. They were only a small group (0.1% of total births), but more importantly only 0.2% of those with a disability. When any combination of these three risks were used, 13.2% of all live births were classified as at risk, and this identified 26.3% of all those with a disability. A striking finding was that 74% of all those with CP, severe mental handicap, hearing, and sight impairments could not be identified using this model.

Very little has changed for those born at term without any noticeable signs during the neonatal period since the first studies of these cohorts in the 1950s. For these infants, failure to reach major motor milestones, such as rolling, sitting or standing, have often been the catalyst for the commencement of developmental assessments and interventions. Given that the window for milestone attainment is quite broad [WHO Multicenter Growth Reference Study Group, 2006], this usually leads to a “wait and see” approach where infants receive no intervention during their period of rapid neural development. In view of the fact that every second child with CP will be born at term and requires no special care in the neonatal period, it is imperative that frontline health professionals such as pediatricians, general practitioners and allied health practitioners have a best practice pathway to follow when a parent

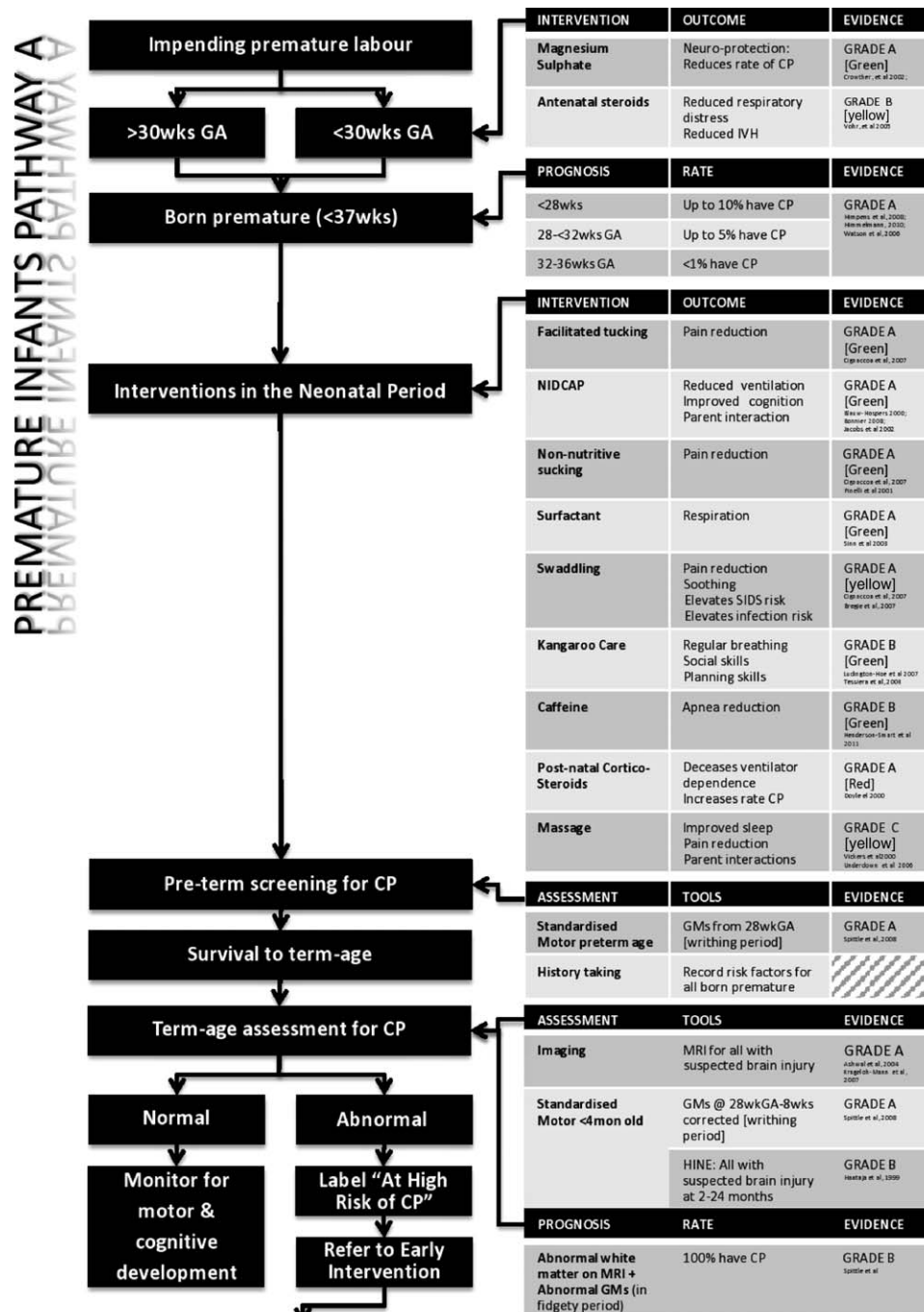


Figure 5 Recommended assessment for identification of infants at risk of CP.

presents with a child who falls into this category.

Practice point. When parents bring their term born child (3 months to 3 years of age) that did not require special care when born to a health professional with concerns regarding motor development or abnormal posturing they should go straight to Pathway B at “screen for CP.” We propose that a tiered approach as developed by Rosebaum et al. [2009] should be adopted. They recommend using the ages and stages questionnaire + three extra ques-

tions for parents. Consideration should also be given to risk factors during pregnancy and signs of mild NE in the neonatal period. When an abnormal result is derived, Pathway B (Figure 5) should be followed to “assessment for CP” through standardized motor assessments.

The description of cerebral palsy is traditionally given late but can be given earlier

This review is timely as “it is now universally accepted that the earliest possible diagnosis and treatment (of

CP) are essential to prevent, or at least minimize, the handicapping effects of a disability and to make the most of the assets a child possesses” [Alberman and Goldstein, 1970]. Yet, paradoxically, 40 years later families are not automatically receiving early intervention while they “wait and see” whether their child will “catch up” from simply a slower motor developmental trajectory or if their child actually has CP or DCD or an intellectual impairment with associated motor difficulties.

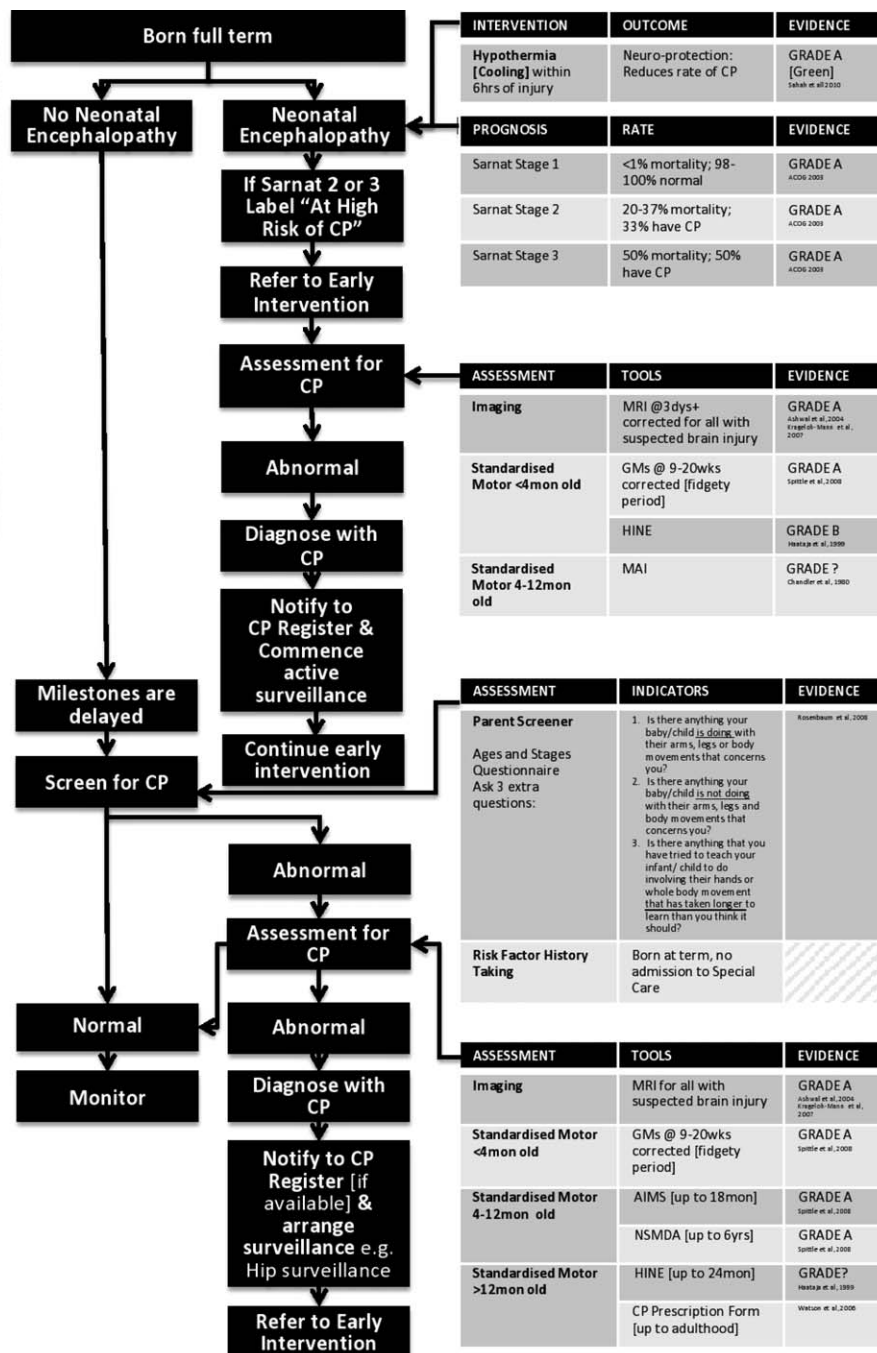


Figure 5 (Continued)

CP registers indicate the average age for a description of CP to be given is 19 months, but the range is wide. For those with severe motor impairment the description of CP can be given as early as 1 week but may take up to 3 years, and less surprisingly for those with mild or moderate motor impairment the description of CP is given anywhere between 1 week and 5 years of age [Watson et al., 2006]. The burgeoning body of recent neuroplasticity literature suggests that intensive, repetitive, task-specific intervention for CP ought to commence very early

while the brain is most plastic (i.e., in the first 2 years of life), which is almost never the case when the family is taking part in “wait and see” monitoring prior to description.

Good evidence shows that earlier detection of CP is both possible and accurate and, more importantly, diagnostic-specific early intervention is therefore possible. Rather than waiting for a formal description of CP to be given, infants should be identified as “at high risk of CP” when they are high risk, and therefore commence diagnostic-specific early intervention straight

away. For those who are not at high risk but have early signs, they should be regularly comprehensively assessed to ensure access to the most appropriate early intervention.

Why is Cerebral Palsy Missed and Why is the Description so Difficult for Doctors to Make?

Health professionals hesitate to use the terminology CP early for a number of reasons, but importantly the condition is not a diagnosis; it is a “clinical description.” There are no biological markers or definitive tests for CP. The

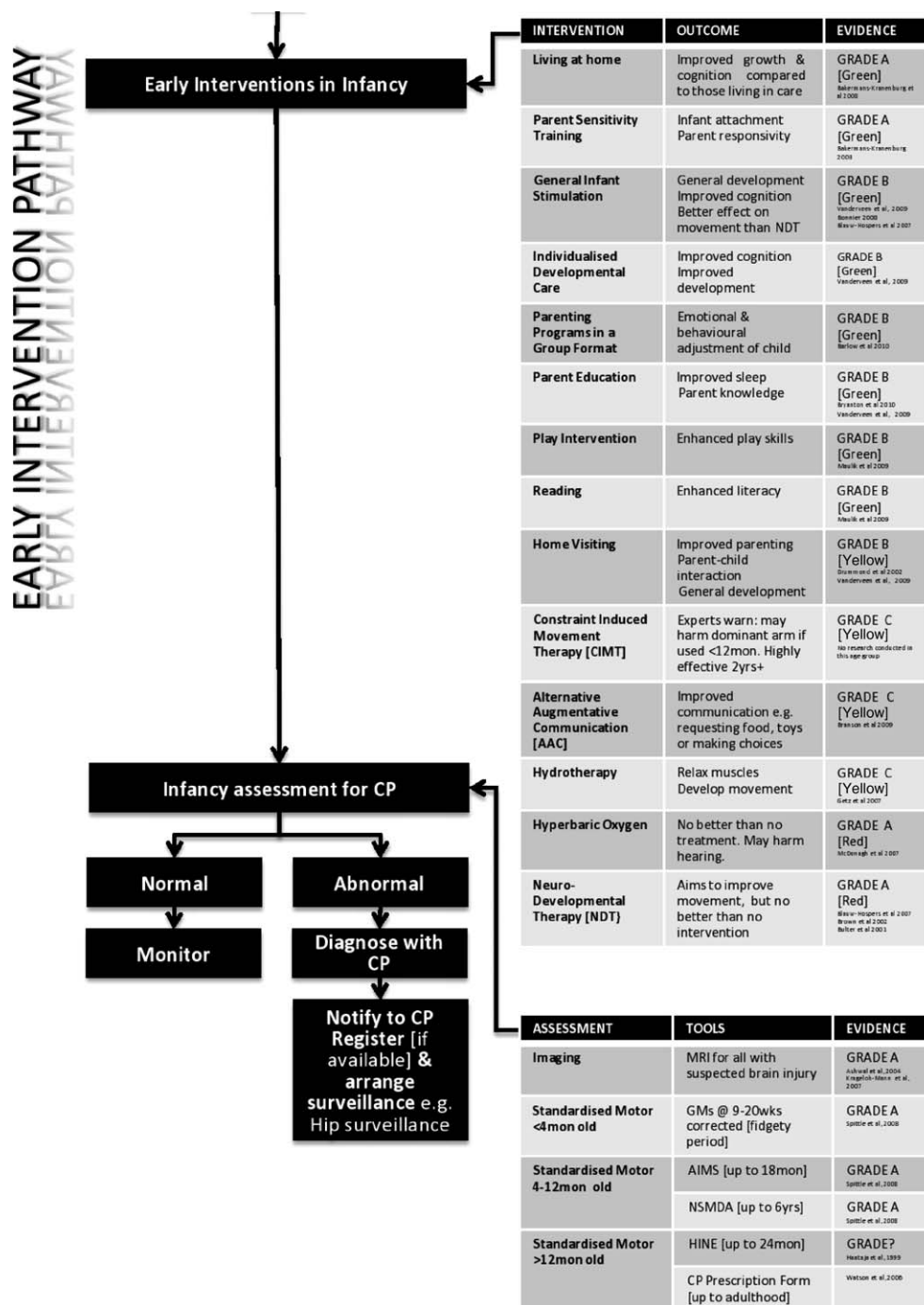


Figure 5 (Continued)

term does not infer etiology, and it has no prognostic value as severity and associated impairments are incredibly variable. However, 86% of parents know something is wrong with their child before a description of CP is given [Baird et al., 2000]. Leading up to this point in time, most parents experience being told by their medical team that the plan is to “wait and see.” When health professionals use the term “wait and see,” the intention is to use this time to rule out other diagnoses, delay the delivery of

bad news or provide time for the child to grow out of it.

Rule out other diagnoses

Doctors first rule out other diagnoses that may explain the symptoms. This is an important step as there are other conditions that mimic the early signs of CP which can have important treatment implications, such as: neuro-degenerative conditions (e.g., Ataxia Telangiectasia); metabolic syndromes (e.g., Glutaric acidemia); and genetic

conditions (e.g., Trisomy 18, Angelman Syndrome, Cornelia de Lange syndrome) [Badawi et al., 1998].

Delay the delivery of bad news

Doctors sometimes delay the delivery of bad news while exploring the possibility of a less severe, more common disorder such as DCD. Differential diagnosis is critical as it informs the selection of intervention strategies suited to the specific condition. For example, effective intervention for

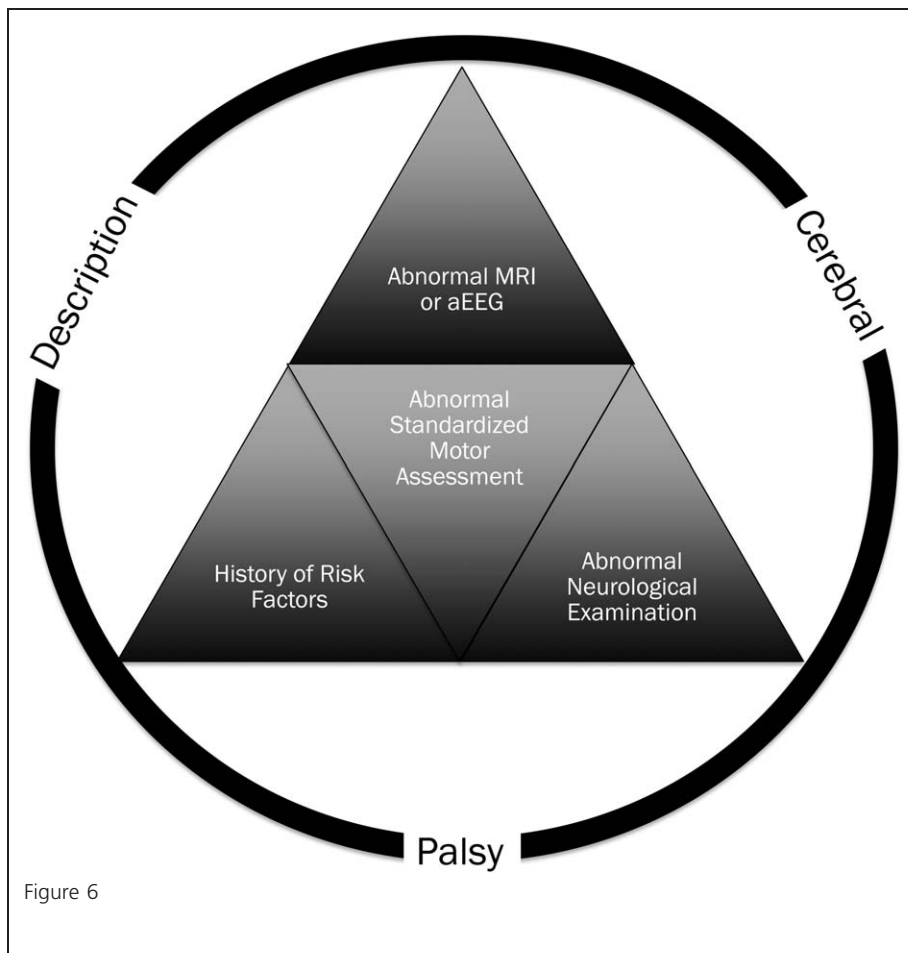


Figure 6

DCD involves cognitive approaches best suited to school-aged children, whereas CP intervention uses a variety of pharmacological, motor, social and cognitive intervention approaches that can commence early in life. It is therefore important that children with CP are differentiated earlier in order to get the right interventions early.

Provide opportunity to grow out of it

Doctors sometimes delay the delivery of bad news to provide enough time for the possibility that the child may “grow out of it.” However for those few whose motor signs resolve, commonly they transpire to have an intellectual impairment or behavioral problems [Nelson and Ellenberg, 1981].

The brain injury responsible for CP may be suspected or even confirmed in the neonatal period, but the diagnosis for many does not occur until the motor impairments and activity limitations inherent in the definition are observable. This lag time is not useful to families or to the child.

“... I am very worried about my son, he is 5 months old, and over the last month I have noticed he seems to go

into strange positions, I especially notice it each time I pick him up. I went to the GP, who agreed and thought I should see a pediatrician. I went to the pediatrician who agreed they were unusual and said let us see how he is when he is 10 months old. That is too long to wait! So I went to another pediatrician who agreed again, it was abnormal, so now I am booked to go to a physiotherapist for further tests, and after that they will decide what to do” but I do not know what to do now...” (Personal communication, February 4, 2012, parent discussion with first author over the phone).

System barriers to description are also potentially at work. For example, for any mother and her newborn, obstetricians hold vital information about maternal-fetal health. If the baby is premature or ill, care is immediately transferred to neonatal specialists, where the primary patient is now the infant, not the mother, and some of the relevant preconception and pregnancy history about risk factors for CP may not be passed on. When the infant is well and discharged from hospital, care is likely to be transferred to a community based general practitioner or

pediatrician who may lack access to the relevant maternal-fetal and/or neonatal medical history. The pediatrician may then be assessing a healthy baby that may just appear slightly “delayed,” and it is not until later in infancy that the gravity of the problem may be evident, precipitating a late diagnosis.

What are the Most Important Things that can be Done in Clinical Practice to Describe Cerebral Palsy Earlier?

We propose a new clinical pathway that is designed to circumvent the existing screening and diagnostic barriers by tying together the relevant evidence needed to make an earlier diagnosis and commence earlier intervention (see Pathways A and B). These pathways have been developed using GRADE level evidence [Guyatt et al., 2008] and “traffic lights” to signify the effectiveness of the interventions [Novak and McIntyre, 2010]. Green equals “go,” (high quality evidence to support the use of the intervention, therefore use this approach). Yellow equals “measure” (low quality or conflicting evidence supporting the effectiveness of the intervention). Red equals “stop” (high quality evidence indicating ineffective interventions) [Novak and McIntyre, 2010].

The serious nature of these standard care limitations has led us to conclude that “waiting and seeing” is potentially harmful to children with CP and their families. We therefore have identified solutions to three of the major problems relating to the late diagnosis of CP, which are timely and possible for the health system to redress:

New clinical diagnostic and intervention pathways

When the system fails to recognize a child with CP very early due to using the “wait and see” monitoring mode, this decision essentially ensures that infants receive limited or no diagnostic-specific intervention within the critical window of brain development. The window of brain development, where the brain is actively sprouting and pruning in response to activity, is often missed in children with CP. In Pathways A and B, we review the evidence for early intervention possibilities in CP. The evidence tells us quite clearly that general early intervention and parent interventions, designed to enhance in-home care characterized by positive interactions, categorically improve a child’s cognition with the

best effect seen in children of low socio-economic status. However, more recent neuroplasticity evidence suggests that a skill-based, high-intensity practice approach to early intervention is required to impact on motor outcomes, as is the case in most adult brain injuries. These newer types of motor learning approaches, which are effective in older children with CP, require urgent study within the CP infant population. It is therefore the responsibility of the health professional who observes major risk factors or a motor delay to investigate further, diagnose “at risk of CP” early, and refer to early intervention at a minimum to optimize their cognitive function. We outline a way to do this via systematic use of risk factor history taking, neurobehavioral predictive tools, in addition to MRI (Pathways A and B).

Promotion of a climate for new research that will improve outcomes

Late description of CP is creating a major problem for recruitment of infants to promising early rehabilitative and potentially curative studies. Lack of diagnosis is impeding the advancement of regenerative medicine, early intervention and other well-recognized treatments for CP yet to be tested in the earlier years, for example, medical interventions for tone management, reflux, and epilepsy. When a health professional identifies an infant at high risk for CP, coupled with referral to early intervention trials, it will help to accelerate future discoveries for these children and change the landscape of the diagnosis and prognosis.

Promotion of good family mental health and resilience for the long-term

If late description is not helping infants or research, are we helping parents by sheltering them from bad news? A population study conducted in Britain found that parental dissatisfaction with delayed diagnosis of CP is associated with higher rates of parental depression [Baird et al., 2000]. So it would appear that sparing parents from bad news is unhelpful. Therefore early recognition and provision of early preventative mental health support for families may help parents manage the inevitable stress, which could help improve family outcomes long-term.

The concept of “at risk” is not a new one. During the 1960s in the United Kingdom, there were “at risk” registers, with the usual accompanying debate over their value and cost effec-

tiveness. It was deemed not practicable to have universal screening of all children, but it was felt essential that all children at risk be monitored. In a letter to the Lancet in 1967 defending the concept, Dr Ronald Mac Keith and colleagues wrote, “by the criterion of identifying handicaps which are in some cases undoubtedly, and in other cases probably, benefited by having treatment started without delay, developmental and neurological assessment from the age of 5 months is neither difficult nor inefficient” [Mac Keith et al., 1967]. The concept itself was deemed by most to be a sound one. The problem at this time was the “at risk” criteria used was identifying up to 60% of all live births in an area. The goal of these programs was to screen 10–20% of all births to identify the majority of the invisible handicaps that is, those that would otherwise not be identified until the 4th and 5th years of life. We recommend that the “wait and see” period is reframed to the “wait and be” period, where children are diagnosed “at risk of CP” early and are immediately referred to diagnostic-specific early intervention.

What Tools can be Used to Accurately Predict and Identify Early Signs of Cerebral Palsy?

Imaging

Practice point. All children with a presumed or suspected brain injury should have magnetic resonance imaging (MRI).

Neuroimaging is used as an integral part of the diagnostic process [Krageloh-Mann and Horber, 2007]. MRI is the gold-standard neuroimaging technique for elucidating the pathogenesis of CP: white matter damage of immaturity (WMDI) including PVL, lesions of the deep grey matter, malformations, focal infarcts, and cortical and subcortical lesions [Bax et al., 2006]. Cranial ultrasound (CUS) is a safe and inexpensive alternative used in the neonatal intensive care unit (NICU) to detect structural changes in the newborn brain. However, MRI has higher sensitivity and specificity than CUS as a predictor of CP in very low birth weight (VLBW) infants [Mirmiran et al., 2004]. Despite strong correlations between clinical findings and MRI, 12–14% of children with CP will have normal MRIs [Bax, 2006; Krageloh-Mann and Horber, 2007] and therefore MRI should not be used in isolation for making the description of CP.

Newer techniques and technologies are being developed which are likely to advance the role of imaging in the diagnostic process and treatment selection process. Advanced neuroimaging techniques such as diffusion weighted imaging (DWI) and diffusion tensor imaging (DTI) have been utilized to more specifically identify diffuse or subtle white matter injuries [Hoon and Faria, 2010]. Magnetic resonance spectroscopy (MRS), provides measures of brain biochemistry and is proving an effective tool in understanding prognosis in NE and preterm infants [Ancora et al., 2010; Van Kooij et al., 2012]. Large deformation diffeomorphic metric mapping (LDDMM), where a 3D atlas of the brain is produced, shows great promise for illuminating the structural brain abnormalities that occur in CP with the potential for informing selection, design, and measurement of rehabilitation interventions [Faria et al., 2011].

General neuromotor and developmental assessments

Many neuromotor and developmental assessments with sound psychometric properties exist for infants and young children. For diagnostic purposes, tools with predictive properties are the most worthwhile. However, there has been a historical preference by pediatricians and neonatal follow-up teams to use discriminative tools that assess a combination of: abnormal muscle tone of the trunk and extremities; the presence of primitive reflexes; the quality and quantity of voluntary movement (e.g., milestone acquisition); and the presence of involuntary movement. The problem with this persistent practice is that these tools are only useful for discriminating between infants who are developing typically from those who are not. Determining who is typically developing and who is not is even more complicated in premature infants because they have their own developmental trajectory [Heineman and Hadders-Algra, 2008; Spittle et al., 2008a]. Routinely used neuro observations and standardized developmental tests were not designed to specifically detect the presence of CP and thus further compound the complexity of the CP diagnostic process. They may be helpful to some diagnosticians but will lack adequate specificity for most.

Ideally the aim of monitoring ought to be to differentiate why some children are not developing normally, to enable diagnostic-appropriate best-

available evidence-based intervention to be provided. This paper will now focus on the evidence for the best available tools for predicting and recognizing CP, distinct from tools better suited to suspecting global developmental delay (GDD). Clinometric reviews indicate that different tools need to be used at different ages to describe and detect CP and that a combination of tools is best practice [Heineman and Hadders-Algra, 2008; Spittle et al., 2008a].

Practice point. A combination of risk factor history taking, neurological examination that includes assessment of quality of movement, volitional movement and neuroimaging are required. A health professional with clinical expertise and experience in motor development should interpret and evaluate the findings generated by these assessments (Figure 6).

Tools predictive of cerebral palsy

Qualitative assessment of general movements [Einspieler et al., 2004]. Of all the tools available to predict CP, GMs is consistently the most predictive, with specificity and sensitivity rates higher than MRI [Burger and Louw, 2009]. The GMs assessment measures the quality of spontaneous movements with the infant lying supine. Scoring is done by trained assessors via observation of video footage and can be used from the preterm period until 20 weeks post term age (PTA). Two distinct time periods for assessment exist; the writhing period (up to 9 weeks PTA) and the fidgety period (from 9 to 20 weeks PTA). In both periods, the infant is scored with “normal” or “abnormal” GMs. Abnormal GMs are then further classified. In the writhing period, abnormal GMs known as “cramped synchronized” have been shown to be highly predictive of CP (sensitivity = 100%; specificity = 40%; PPV = 9.4%; NPV = 100% [Spittle et al., 2009]. If the abnormal GM of “cramped synchronized” is followed by the abnormal GM “absent fidgety” (in the fidgety period) this has consistently shown the highest predictive value for CP [Darsaklis and Snider, 2011].

A recent systematic review of 17 studies demonstrated the accuracy of the GMs assessment in predicting neurodevelopmental outcomes in infants up to 2 years with a sensitivity $\geq 92\%$ and specificity $\geq 82\%$ [Burger, 2009]. The GMs assessment has been found to be superior to ultrasound findings in predicting CP [Einspieler et al., 2004]

When correlated with MRI findings, namely white matter injury, the GMs assessment (specifically “absent fidgety”) has been shown to accurately predict CP 100% of the time in very preterm infants [Spittle et al., 2008a]. Evidence of the predictive value of GMs in full term infants with hypoxic ischemic encephalopathy (HIE) has also been demonstrated [Prechtl et al., 1993]. Importantly, the GMs assessment has good clinical utility because it is quick, inexpensive, and noninvasive. Rater training is provided by the GMs trust.

Hammersmith infant neurological assessment [Haataja et al., 1999]. The Hammersmith assessment is based on the Dubowitz and Dubowitz [1981] assessment of the newborn and is a simple method of examining infants between 2 and 24 months of age. There are three parts to the examination: neurologic signs, developmental milestones, and behavior. In the first section, the neurologic exam, an optimality score is obtained from the assessment of cranial nerve function, posture, quality and quantity of movement, tone, and reflexes and reactions. The second and third sections do not form part of the overall score but give important additional information regarding developmental progress. Recent studies have demonstrated the predictive value of the Hammersmith infant neurological assessment (HINE) for CP. A large study [Pizzardi et al., 2008] of 658 infants who were either preterm or term with NE were prospectively studied from birth until 12 months corrected age. ROC curve analysis was used to test the predictive power of the HINE. Global HINE scores showed high prediction of CP at all ages (ROC curve areas above 0.9), but most importantly movement quality and quantity test items had even higher predictive power.

A retrospective study of 70 infants diagnosed at 2 years with CP observed a strong ($r = -82$) negative correlation between HINE scores at 3–6 months of age and levels of GMFCS [Romeo et al., 2008a]. Infants in GMFCS levels 3–5 scored below 40, whereas those in levels 1–2 scored between 40 and 60. Combined use of the HINE and GMs at 3 months PTA can be used to describe an infant as at “high risk” of CP [Romeo et al., 2008b].

Practice point. Routine follow-up for preterm and sick infants should be scheduled at three-months and six-months corrected, not the conventional

four-months, to enable medical teams to use the best predictive tools to help make the description of CP earlier.

Practice point. When examining infants, do not discount CP when spasticity or dyskinesia is not identified. A period of time lapses between the original damage to the developing brain, whether in utero or during early infancy/childhood, and the appearance of impairments. It is well known that the brain, which begins development in utero, continues to develop during childhood. Thus a child’s neural development is “age-specific,” so brain dysfunction will manifest according to the brain’s development at that age [Hadders-Algra, 2004]. Compared with a mature brain which responds to injury with specific and localized signs, a young infant may present with generalized and nonspecific signs (e.g., hypotonia) [Kuban and Leviton, 1994; Hadders-Algra, 2004]. It is proposed that further brain development in an infant, including myelination of axons and maturation of basal ganglia neurons, must occur before spasticity and dyskinesia can manifest [Kuban and Leviton, 1994]. The infant with hypotonia may thus “develop” spasticity and dyskinesia by the age of 1 or 2 years, as the complexity of neural functions increases [Kuban and Leviton, 1994; Hadders-Algra, 2004].

Movement assessment of infants [Chandler et al., 1980]. The movement assessment of infants (MAI) is a criterion-referenced scale that evaluates neuromotor dysfunction in high risk infants at 4, 6, 8, and 12 months of age. The assessment is carried out by a therapist and takes 30–60 min to complete, requiring a manual but no specialized equipment. The MAI assesses tone, primitive reflexes, equilibrium reactions, and volitional movement. The test has been shown to be twice as sensitive as the Bayley scales of infant development in detecting early signs of CP [Harris, 1987]. Studies of predictive values at 4 and 8 months of age report sensitivity rates ranging from 73.5 to 96.0 and specificity of 62.7–78.2 [Spittle et al., 2008b]. A recent investigation of the predictive validity of the MAI at 6 months of age demonstrated a significant correlation between MAI scores and Bayley scales of infant development at 12 months, although sensitivity and specificity for CP were not reported [Metgud et al., 2011].

Other useful assessments. Several other neuromotor assessments, such as the test

of infant motor performance (TIMP) [Campbell, 2005], The neuro-sensory motor development assessment (NSMDA) [Burns et al., 1989], and the Alberta infant motor scale (AIMS) [Piper and Darrah, 1994], are appropriately used to discriminate infants with abnormal motor function from those typically developing. All have sound psychometrics. Of these tools, the TIMP has been shown to be sensitive to change in response to intervention [Campbell et al., 1995].

Assessment summary.

- High risk infants should be routinely assessed using the GMs preferably three times; during early admission, around term corrected (if preterm) and at 9–14 weeks (corrected for gestational age).
- “High risk of CP” designation should be given to infants at 9–14 weeks (corrected) with a combination of absent fidgety GMs and white matter injury on MRI.
- After 20 weeks (corrected), use the HINE or MAI.
- MRI is the best imaging tool to elucidate the pathogenesis of CP and should be offered to all infants who have abnormal findings.
- Use the CP description form to describe motor type and severity to inform intervention planning.

CONCLUSION

Until recently, CP was considered unpreventable, incurable, and almost untreatable. However, preventive efforts including: rubella vaccination, iodine supplementation in areas of severe iron deficiency, anti-D vaccination, preventing methyl-mercury contamination, reducing the number of embryos transferred in invitro fertilization (IVF) (in Australia), and enforcing laws for seat belts and fencing around swimming pools have been successful prevention strategies. Recently, magnesium sulfate and hypothermic intervention have also started to prevent a small proportion of CP. Both of these interventions occur very early and require health professionals to be mindful of CP as a potential outcome that could be prevented or cured. With advances in medical, public health, and allied health research, the likelihood of further breakthroughs are probable.

Further research is required to determine why infants born at term, not at “high risk” of CP in the newborn period go on to develop CP. Health professionals need to be aware that 45%

of all CP falls into this category. Therefore we recommend prompt response to parental concerns with screening and assessments as outlined, followed by immediate referral for intervention for those infants then considered “at risk.”

Premature and term infants with brain injury identified on MRI are at high risk of CP. We have identified pathways which make recognizing “at high risk” of CP easier for health professionals. We propose a change in diagnostic practice, a shift away from referral for intervention following a formal (most often late) description to one of referral when an infant is “at high risk” of CP. This will provide the opportunity for targeted research in early intervention, thus providing optimal outcomes for children with CP.

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