CEREBRAL PALSY

Introduction

Cerebral Palsy (CP) is the name given to a group of disorders characterised by impairments in the development of movement and posture due to non-progressive damage or malformation of the fetal or infant brain. Although the brain damage is non-progressive, its manifestations may change over time as the brain develops. Cerebral palsy is the most common physical disability in children, affecting around two in every 1,000. In addition to motor disorders, children with CP often have other comorbidities including intellectual disability (30–65% of cases), epilepsy (30–50%), speech and language deficits (40%), visual impairments (40%), hearing problems (5–15%), psychosocial and behavioural problems (20%) and autism spectrum disorder (9%). Medical complications of CP can involve multiple bodily systems including the genitourinary (incontinence, urinary infections, and voiding dysfunction), gastrointestinal (dysphagia, gastroesophageal reflux disease, constipation), respiratory (recurrent pneumonia, atelectasis, bronchiectasis, restrictive lung disease), and endocrine (reduced growth and osteopenia).

In clinical practice, diagnosis of CP is based on observation and parental reports of delayed attainment of motor milestones (such as achieving head control, sitting and standing), and evaluation of posture, deep tendon reflexes, and muscle tone. There is wide variation in the degree of impairment associated with CP. The least severely affected children have only minor limitations in speed, balance and coordination, while the most severely affected are unable to maintain anti-gravity head and trunk postures, or control leg and arm movements, and need wheelchairs and assistance with all aspects of daily life.

Data sources and methods

<table>
<thead>
<tr>
<th>Indicator</th>
</tr>
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<tbody>
<tr>
<td>Rates of cerebral palsy among 0–24 year olds</td>
</tr>
</tbody>
</table>

**Definition**

Hospitalisations of 0–24 year olds with cerebral palsy per 100,000 population

**Data sources**

**Numerator:** National Minimum Dataset

**Denominator:** Statistics New Zealand Estimated Resident Population (with linear extrapolation being used to calculate denominators between Census years)

**Additional information**

This section presents analyses where the condition was the primary diagnosis or was documented within any of the first 15 diagnoses (all cases). The rationale for presenting all cases is to highlight the full spectrum of health issues experienced by those with this condition, and their consequent requirement for acute health services. Analyses are per hospital discharge event, therefore events are only included if the condition is documented within either the primary diagnosis or within any of the first 15 diagnoses.

Codes used for identifying cases are documented in Error! Reference source not found..

National trends and distribution

There was a total of 58 deaths of 0–24 year olds with cerebral palsy as the underlying cause of death in New Zealand during 2009 to 2013, as documented within the National Mortality Collection.

The number of 0–24 year olds hospitalised between 2011 and 2015 with any diagnosis of cerebral palsy is presented in Table 1, together with the total number of hospitalisations with cerebral palsy as a primary or any diagnosis.

Since 2000 hospitalisation rates for cerebral palsy as a primary diagnosis have risen steadily for 0–14 year olds, peaking in 2012 for 0–4 year olds and 2014 for 5–14 year olds. Hospitalisation rates were lowest for 15–24 year olds (Figure 1). Overall there is a steady increase in hospitalisations of children hospitalised with cerebral palsy as the primary diagnosis (Figure 1). Similar patterns over time were seen in all ethnic groups.
The majority of hospitalisations of 0–24 year olds with a primary diagnosis of cerebral palsy had spastic cerebral palsy as a primary diagnosis. Other associated primary diagnoses included respiratory, digestive or musculoskeletal conditions (Table 2).

Table 1. Individuals hospitalised with cerebral palsy, 0–24 year olds, New Zealand 2011–2015

<table>
<thead>
<tr>
<th>Age group</th>
<th>Unique individuals (n)</th>
<th>Hospitalisations (n)</th>
<th>Ratio All:Primary</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cerebral palsy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hospitalisation</td>
<td></td>
</tr>
<tr>
<td>0–24 years</td>
<td>1,678</td>
<td>2,458</td>
<td>5,852</td>
</tr>
<tr>
<td>0–14 years</td>
<td>1,198</td>
<td>2,221</td>
<td>4,469</td>
</tr>
<tr>
<td>15–24 years</td>
<td>581</td>
<td>237</td>
<td>1,383</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset. ‘Primary’ corresponds to hospitalisations where cerebral palsy was primary diagnosis; ‘All cases’ = inclusion in any of the first 15 diagnoses. The sum of the age groups may total to more than the 0–24 year old total.

Figure 1. Hospitalisations for cerebral palsy in 0–24 year olds, by age group, New Zealand 2000–2015

Table 2. Hospitalisations involving cerebral palsy in 0–24 year olds, by primary diagnosis, New Zealand 2011–2015

<table>
<thead>
<tr>
<th>Primary diagnosis</th>
<th>2011–2015 (n)</th>
<th>Annual average</th>
<th>Rate per 100,000 0–24 year olds</th>
<th>95% CI</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral palsy* in 0–24 year olds</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spastic cerebral palsy</td>
<td>1,958</td>
<td>392</td>
<td>25.49</td>
<td>24.39–26.65</td>
<td>33.5</td>
</tr>
<tr>
<td>Dyskinetic cerebral palsy</td>
<td>85</td>
<td>17</td>
<td>1.11</td>
<td>0.90–1.37</td>
<td>1.5</td>
</tr>
<tr>
<td>Ataxic cerebral palsy</td>
<td>5</td>
<td>1</td>
<td>0.07</td>
<td>0.03–0.15</td>
<td>0.1</td>
</tr>
<tr>
<td>Cerebral palsy, other or unspecified</td>
<td>410</td>
<td>82</td>
<td>5.34</td>
<td>4.85–5.88</td>
<td>7.0</td>
</tr>
<tr>
<td>Cerebral palsy total</td>
<td>2,458</td>
<td>492</td>
<td>32.00</td>
<td>30.76–33.29</td>
<td>42.0</td>
</tr>
<tr>
<td>Other diseases of the nervous system</td>
<td>375</td>
<td>75</td>
<td>4.88</td>
<td>4.41–5.40</td>
<td>6.4</td>
</tr>
<tr>
<td>Diseases of the respiratory system</td>
<td>662</td>
<td>132</td>
<td>8.62</td>
<td>7.99–9.30</td>
<td>11.3</td>
</tr>
<tr>
<td>Diseases of the digestive system</td>
<td>547</td>
<td>109</td>
<td>7.12</td>
<td>6.55–7.74</td>
<td>9.3</td>
</tr>
<tr>
<td>Diseases of the musculoskeletal system and connective tissue</td>
<td>482</td>
<td>96</td>
<td>6.28</td>
<td>5.74–6.86</td>
<td>8.2</td>
</tr>
<tr>
<td>Symptoms and/or abnormal clinical findings NEC</td>
<td>347</td>
<td>69</td>
<td>4.52</td>
<td>4.07–5.02</td>
<td>5.9</td>
</tr>
<tr>
<td>Other diagnoses</td>
<td>981</td>
<td>196</td>
<td>12.77</td>
<td>12.00–13.60</td>
<td>16.8</td>
</tr>
<tr>
<td>Total</td>
<td>5,852</td>
<td>1,170</td>
<td>76.19</td>
<td>74.26–78.17</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. *Cerebral palsy in any of the first 15 diagnoses; NEC = not elsewhere classified

**Demographic distribution**

Table 3 presents the demographic distribution of individuals with cerebral palsy in New Zealand between 2011 and 2015. Cerebral palsy rates were significantly higher in higher deprivation areas (NZDep2013 deciles 7–10) compared to lower deprivation areas (NZDep2013 deciles 1–2), and significantly higher among 0–4 year olds and 5–14 year olds compared to 15–24 year olds. Hospitalisation rates were significantly lower for Asian/Indian 0–24 year olds than other ethnic groups. The majority of 0–24 year olds with cerebral palsy were of European/Other ethnicity.
Table 3. 0–24 year olds hospitalised with cerebral palsy, by demographic factor, New Zealand 2011–2015

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unique individuals 2011–2015 (n)</th>
<th>Rate per 100,000 population</th>
<th>Rate ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral palsy* in 0–24 year olds</td>
<td>New Zealand</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ Deprivation Index quintile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deciles 1–2</td>
<td>305</td>
<td>21.49</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Deciles 3–4</td>
<td>328</td>
<td>24.53</td>
<td>1.14</td>
<td>0.98–1.33</td>
</tr>
<tr>
<td>Deciles 5–6</td>
<td>317</td>
<td>21.99</td>
<td>1.02</td>
<td>0.87–1.20</td>
</tr>
<tr>
<td>Deciles 7–8</td>
<td>458</td>
<td>28.19</td>
<td>1.31</td>
<td>1.13–1.52</td>
</tr>
<tr>
<td>Deciles 9–10</td>
<td>574</td>
<td>30.89</td>
<td>1.44</td>
<td>1.25–1.65</td>
</tr>
<tr>
<td>Prioritised ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>405</td>
<td>22.45</td>
<td>0.99</td>
<td>0.88–1.11</td>
</tr>
<tr>
<td>Pacific</td>
<td>191</td>
<td>26.95</td>
<td>1.18</td>
<td>1.01–1.38</td>
</tr>
<tr>
<td>Asian/Indian</td>
<td>126</td>
<td>13.14</td>
<td>0.58</td>
<td>0.48–0.69</td>
</tr>
<tr>
<td>MELAA</td>
<td>38</td>
<td>37.68</td>
<td>1.65</td>
<td>1.20–2.29</td>
</tr>
<tr>
<td>European/Other</td>
<td>936</td>
<td>22.78</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>723</td>
<td>19.26</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>955</td>
<td>24.32</td>
<td>1.26</td>
<td>1.15–1.39</td>
</tr>
<tr>
<td>Age group (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–4</td>
<td>439</td>
<td>28.15</td>
<td>1.52</td>
<td>1.34–1.72</td>
</tr>
<tr>
<td>5–14</td>
<td>943</td>
<td>31.60</td>
<td>1.71</td>
<td>1.54–1.89</td>
</tr>
<tr>
<td>15–24</td>
<td>581</td>
<td>18.52</td>
<td>1.00</td>
<td></td>
</tr>
</tbody>
</table>

Numerator: National Minimum Dataset, Denominator: Statistics NZ Estimated Resident Population. *Cerebral palsy in any of the first 15 diagnoses; Rate per 100,000 age-specific population; Rate ratios are unadjusted; Ethnicity is Level 1 prioritised; Decile is NZDep2013

Regional trends and distribution

Hospitalisation rates for cerebral palsy showed year-to-year variability in South Island DHBs between 2000 and 2015. The incidence of cerebral palsy being the primary cause of hospitalisation was much lower than all cases across the DHBs; note South Canterbury and West Coast are based on small numbers and suppressed for primary diagnosis (Figure 2). Numbers of unique individuals and hospitalisations between 2011 and 2015 are shown in Table 4.
Figure 2. Hospitalisations for cerebral palsy in 0–24 year olds, South Island DHBs vs New Zealand 2000–2015

Numerator: National Minimum Dataset and National Mortality Collection, Denominator: Statistics New Zealand Estimated Resident Population. "All cases" corresponds to hospitalisations with cerebral palsy listed in any of the first 15 diagnoses; Rates for South Canterbury and West Coast are based on small numbers and suppressed for primary diagnosis.

Table 4. Hospitalisations for cerebral palsy on 0–24 year olds, South Island DHBs vs New Zealand 2011–2015

<table>
<thead>
<tr>
<th>DHB</th>
<th>Unique individuals (n)</th>
<th>Hospitalisations (n)</th>
<th>Ratio All:Primary</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Principal diagnosis</td>
<td>All cases</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>51</td>
<td>44</td>
<td>162</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>11</td>
<td>10</td>
<td>29</td>
</tr>
<tr>
<td>Canterbury</td>
<td>214</td>
<td>253</td>
<td>705</td>
</tr>
<tr>
<td>West Coast</td>
<td>12</td>
<td>13</td>
<td>32</td>
</tr>
<tr>
<td>Southern</td>
<td>103</td>
<td>62</td>
<td>340</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1,678</td>
<td>2,458</td>
<td>5,852</td>
</tr>
</tbody>
</table>

Source: National Minimum Dataset. ‘All cases’ corresponds to hospitalisations with cerebral palsy listed in any of the first 15 diagnoses.
Evidence for good practice

Possibilities for prevention

The causes of the brain damage that produces cerebral palsy are not well understood therefore the possibilities for prevention are limited.\(^9,10\) While it was formerly believed that lack of oxygen during birth was the cause of CP, it is now thought that, in the majority of cases (at least 80%), the brain injury or malformation occurs before birth and that no more than 10% of cases are the result of perinatal factors.\(^4\) A small proportion of cases (5–10%) are due to perinatal brain injury in the early months and years of life, for example from cerebrovascular accident (stroke); head trauma due to motor vehicle crashes, falls or child abuse; near drowning and other forms of asphyxia; and infection such as meningitis.\(^4,15\)

Few studies investigate more than one risk factor yet it seems probable that, in many cases, CP results from the interaction of multiple risk factors.\(^12\)

Prenatal

There is a genetic component to cerebral palsy risk as the recurrence rate in families of people with CP is greater the closer the degree of genetic relationship.\(^13\) Nevertheless, the absolute risks for family members are low because CP is not a very common condition. Current guidelines do not recommend routine genetic testing.\(^14\)

In developed countries, increasing maternal age and the use of assisted reproduction therapies have both increased the rate of multiple births, which have a higher risk of CP than singleton births.\(^15\)

Among the few preventable prenatal causes of CP are severe maternal iodine deficiency,\(^16\) rhesus isoimmunisation,\(^17\) consanguineous marriages,\(^9\) and maternal methyl mercury poisoning.\(^18\) These factors cause almost no cases of CP in developed countries.\(^19\)

Babies who are born small for their gestational age or well above the normal weight for their gestational age are at increased risk of CP but it is uncertain whether the abnormal growth patterns are a cause or a consequence of CP.\(^20\)

Perinatal

High quality maternity care can prevent or mitigate complications of labour and delivery that increase the risks of adverse health outcomes for babies, including brain damage leading to CP.\(^21\)

Preterm birth is an important risk factor for CP and the risk increases markedly with decreasing gestational age, nevertheless more than half of all children with CP were born at term.\(^4\) Prevention of preterm birth is challenging as its causes are multifactorial and poorly understood.\(^22\)

In women threatening to or likely to give birth preterm, there is evidence that antenatal magnesium sulphate therapy substantially reduces the risk of their child having CP,\(^23\) although mothers can experience unpleasant side effects such as tachycardia, flushing and nausea/vomiting.\(^24\)

A recent review of risk factors for CP in children born at term in developed countries identified 10 statistically significant risk factors, three of which were considered possibly preventable.\(^25\) These three were: birth asphyxia, low birthweight and meconium aspiration.\(^25\)

In infants born at 35+ weeks gestation with evidence of peripartum asphyxia (such as needing mechanical ventilation or resuscitation at 10 minutes after birth) and encephalopathy (such as seizures), induced hypothermia (cooling) reduces mortality and neurodevelopmental disability in survivors.\(^26\)

Fetal heart rate monitoring using continuous cardiotocography (CTG) has not been shown in RCTs to reduce cerebral palsy rates but it does increase rates of caesarean sections and instrumental vaginal births.\(^27\)

The passage of fetal bowel movement (meconium) in the amniotic fluid is common, and it can (rarely) result in meconium aspiration syndrome if the baby inhales meconium during the birth process.\(^28\) According to a 2012 Cochrane review, curtailment of post-term pregnancies by induction of labour reduces the occurrence of meconium stained amniotic fluid and meconium aspiration syndrome.\(^29\) There is little research evidence regarding the benefits or otherwise of obstetric interventions such as expedited delivery when there is meconium-stained amniotic fluid without other evidence of fetal distress.\(^28\)

Postnatal

Because infants born preterm have a greatly increased risk of developing CP, early intervention programmes have been used in the hope of preventing cerebral palsy and promoting normal brain development in preterm
infants. The evidence indicates that general development programmes for preterm infants have no effect on CP rates in survivors of preterm birth.\textsuperscript{30} There is some evidence that such programmes improve cognitive outcomes in the preschool years although not at school age. There is little evidence for positive effects on motor outcomes beyond infancy.\textsuperscript{30,31} It has been argued that early intervention research to date does not provide sufficient evidence to exclude the possibility that early intervention could have long lasting benefits for infants at high risk of developing CP or with early signs of CP.\textsuperscript{32}

Interventions to prevent head injuries in infants and toddlers, such as promoting car seat use and preventing shaken baby syndrome,\textsuperscript{33,34} may help prevent post-natally acquired CP.

**Evidence-based health care for children and young people with cerebral palsy**

Children with CP often have multiple medical issues and they typically need services from multiple healthcare professionals including paediatricians, orthopaedic surgeons, neurologists, ophthalmologists, optometrists, audiologists, physiotherapists, dieticians, occupational therapists, speech language therapists and dentists.\textsuperscript{35} They also may need special education services, in-home care and respite care.\textsuperscript{35}

The evidence base for CP therapies is limited largely to systematic reviews, meta-analyses, and large multicentre prospective cohort studies because of the lack of well conducted prospective randomised controlled trials on this topic.\textsuperscript{11,36} The interventions that are best supported by evidence are: anticonvulsants, bimanual training, botulinum toxin, bisphosphonates to prevent osteoporosis, casting, constraint-induced movement therapy, context-focused therapy, diazepam, fitness training, goal-directed training, hip surveillance, home programmes, occupational therapy after botulinum toxin, pressure care, and selective dorsal rhizotomy.\textsuperscript{36}

**Coordination of care**

In English-speaking developed countries, and increasingly in other developed countries, family-centered service provision for children with special needs is considered to be best practice.\textsuperscript{37,38} This model of service provision recognises that each family is unique, that the family is the constant in the child’s life, and that they are the experts on the child’s abilities and needs. It involves the family working together with service providers to make informed decisions about the services and supports the child will receive and it considers the strengths and needs of all family members.\textsuperscript{39,40}

There has been very little research on family-centered care specifically for families affected by CP but a review of 24 studies (including seven RCTs) of family-centered care for US children with conditions associated with having special health care needs found positive associations between family-centered care and improvements in efficient use of services, health status, satisfaction, access to care, communication, systems of care, family functioning, and family financial impact and cost.\textsuperscript{41} It did not identify any negative outcomes.

While multidisciplinary teams are an integral part of child health services, they are often lacking in adult healthcare services and young adults with CP (or their families) can be left to coordinate their own healthcare at a time when they are dealing with many transitions in other areas of life including education and employment, finances and benefits, housing, transportation, leisure activities and relationships.\textsuperscript{42-44}

Caring for a child with cerebral palsy is often challenging for families and primary caregivers of children with CP have been found to have lower incomes that other parents despite similar levels of education, to be less likely to be working for pay and to have a greater likelihood of physical and psychological health problems.\textsuperscript{45,46} In contrast to parents of normal children, parents of children with CP can find that the demands of caregiving become greater as their child ages.\textsuperscript{47} Research has not consistently found that the severity of a child’s motor impairment determines the impact of CP on families’ wellbeing but it has consistently found that CP that is accompanied by intellectual disability and behaviour problems is associated with a more severe impact.\textsuperscript{48}

There is research indicating that respite care is an appropriate and effective intervention for decreasing parental stress and giving parents the chance to spend time with other family members.\textsuperscript{49} Respite care can also be considered a child abuse prevention intervention, particularly for children with challenging behaviours.\textsuperscript{49} There seems to have been little research on effective models for respite care for children with developmental disability and severe behaviour problems.\textsuperscript{49}

**Physiotherapy**

Physiotherapy is an established component of the management of CP. There is an increasing evidence base to support a number of physiotherapy interventions including constraint-induced movement therapy (where a child’s less-affected hand is restrained in a mitt or cast and the more-affected hand is given intensive training)\textsuperscript{50,51}, strengthening interventions for individual muscle groups\textsuperscript{52} and functional training (training
focussed on motor activities similar to those involved in daily living such as stair climbing, walking and moving from sitting to standing) but research studies have generally been small (< 30 participants).52,54

Exercise interventions may improve postural control in children with CP.55 There is a moderate level of evidence to support gross motor task training, therapeutic horseback riding, treadmill training with no body weight support, trunk-targeted training, and reactive balance training.55

Orthoses, especially ankle-foot orthoses which aim to facilitate standing upright and walking, are commonly prescribed for children with CP although the evidence for their efficacy is limited to low level evidence that they improve gait in the short term.56

To achieve their functional potential children with CP need to be motivated to persist with their rehabilitation therapy. There has been very little research on the effects of motivational interventions for children with CP.57

**Treatments for spasticity**

Spasticity (tight muscles as a result of damage to the parts of the brain that control movement) can be a significant source of functional disability in many children with CP. Spasticity inhibits movement and causes pain both directly through producing cramp and indirectly through producing extreme joint positions. Interventions to manage spasticity include physiotherapy, casting and splinting, orthopaedic and neurosurgery, botulinum toxin injections and oral medications.

Oral medications to treat spasticity include benzodiazepines, dantrolen, baclofen and tizanidine. The evidence base for the use of oral medications for spasticity in CP is limited because most studies were conducted many years ago and, by modern standards, had methodological limitations.58 The choice of medication is therefore largely based on personal experience or trial and error.

There is a growing body of evidence for the effectiveness of botulinum toxin A (Botox) in reducing spasticity and improving motor function in children with CP when it is used in combination with other treatment measures.59-61

There is a small amount of evidence that intrathecal baclofen (baclofen infused by a pump connected to a catheter directly into the subarachnoid space around the spinal cord) is an effective treatment for treatment of spasticity in children with CP in the short term.62

Selective dorsal rhizotomy (SDR) is an irreversible neurosurgical procedure involving cutting selected sensory nerve roots in the lumbar spine under intraoperative neurophysiological guidance. Intensive post-operative physiotherapy is necessary. Selective dorsal rhizotomy is effective for reducing spasticity in certain carefully selected young patients with bilateral spastic CP and can reduce the need for further surgical interventions and improve quality of life and independence with activities of daily living.63 Further research is needed regarding the long term outcomes of SDR, especially with regard to functional activity and participation.36,64

**Orthopaedic surgery**

Musculo-skeletal pathology develops in the limbs of most children with CP and orthopaedic surgery procedures such as tendon lengthenings, tendon transfers, rotational osteotomies, and joint stabilization procedures have been developed to address the various components of this pathology.65

Children with severe CP commonly develop hip problems involving displacement of the femoral head resulting in pain, caregiving problems, seating problems, pressure sores, fractures and contractures. Regular hip surveillance programmes for children with CP allow earlier identification of subluxation and reduces the need for surgery on dislocated hips.56 There is on-going debate about the best surgical intervention and the best timing of surgical intervention to deal with this problem.67

Equinus foot deformity is the most common musculoskeletal deformity in CP. There is low quality evidence supporting surgical intervention to correct this problem but no evidence to indicate which particular surgical technique is best.68

Surgical correction of thumb-in-palm deformity in children with spastic CP seems to improve hand function, to facilitate hygiene, and to improve the appearance and quality of life but the evidence is limited to prospective studies comparing pre- and post-operative outcomes.69

To avoid having to subject a child with CP to multiple hospital stays and rehabilitation periods it has become common to do single-event multilevel surgery (SEMLS) in which multiple levels of musculoskeletal pathology in both lower limbs are addressed by multiple surgical teams during a single surgical operation.63 The evidence
base for this approach is limited due to a lack of RCTs. Most studies to date have been retrospective studies or prospective cohort studies and most have had fewer than 30 participants.

**Feeding interventions**

Children with severe CP often have problems with sucking, chewing and swallowing their food. This puts them at risk of under nutrition and of recurrent aspiration pneumonia (due to inhaling food into their lungs). Both behavioural and surgical techniques may be used to deal with feeding difficulties. The available research literature provides little evidence on the effectiveness of behavioural therapies (such as positioning, altering food consistency, or use of feeding devices) due to a lack of RCTs and generally small sample sizes. There is no RCT evidence regarding feeding tubes. Evidence from six case series indicates that surgically placed feeding tubes (gastrostomy or jejunostomy tubes) improve weight gain but there is insufficient evidence regarding their effects on respiratory outcomes, parent and child quality of life, or long term morbidity and mortality. There is some low quality evidence that they increase the potential for over feeding and gastro-oesophageal reflux.

**Evidence-based health care for children and young people with cerebral palsy**

These national and international guidelines, systematic reviews, other publications and websites relevant to the prevention and management of CP are suggestions for further reading.

**New Zealand guidelines**


**International guidelines**

Evidence-based medicine reviews

The following review provides an excellent overview of the evidence for interventions for children with cerebral palsy:


**Other relevant publications**


**Websites**


• Australasian Academy of Cerebral Palsy and Developmental Medicine. [https://ausacpdm.org.au/](https://ausacpdm.org.au/)


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