Chapter 3

Traumatic Brain Injury in Very Early Childhood

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Much research has been published on the cognitive and behavioural outcomes of traumatic brain injury (TBI) sustained by school age children [1–3]. In comparison, limited research has focused on the recovery of children injured at preschool age and younger. This chapter focuses on (TBI) in infants and young children. In this chapter we focus on TBI occurring from infancy up to five years of age, referring to this age group as 'young children'. The chapter highlights the differences in epidemiology and physiology in this age group from older children and goes on to discuss the associated cognitive and behavioural outcomes within this age group.

Epidemiology of TBI in Young Children

TBI occurs at high rates in young children and is a major cause of death and disability [4]. For example, work by Bayreuther et al. [5] found that infants had almost double the incidence of injuries to the head than older children. Although there is a focus on abusive head trauma (AHT) in the younger age group, the majority of TBI are the result of accidents [6]. Major causes of TBI in children under 3 years of age are falls of short distances, often from furniture such as beds, couches, and change tables [7, 8]. In children aged 3–6 years, TBIs also occur from falls from playground equipment, bicycles and scooters [9, 10]. Motor vehicle accidents and AHT are the main cause of severe TBIs in young children [6]. However, riding a bicycle without a helmet is associated with significant TBI [9].

The Influence of Head and Neck Physiology of Young Children's Vulnerability to TBI

The physiology of the young child's skull and neck makes them particularly vulnerable to increased damage to the brain from a TBI. In infants, the skull is thin and pliable allowing the head to move through the birth canal. Whilst a

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necessary feature for a natural birth in the event of a TBI, the softness of the skull offers the brain little defence from external trauma [11]. From birth to two years of age, the skull becomes thicker and the sutures fuse. Prior to closing, the sutures are described as 'fibrous connections' offering minimal protection of the brain [11, 12]. In younger children the head, relative to body size, is disproportionately larger, and weighs between 10 and 15% of the total body weight compared to adults whose head contributes only 2–3% of total body weight [11]. The muscles of the neck are weak and therefore the head is not well supported [11, 12]. The combination of a large head and weak neck muscles makes the young child more susceptible to rotational and shearing forces [13]. The limited ability of the young child's skull to absorb biomechanical force means the brain is susceptible to significant injury [14] with an increase in diffuse as opposed to focal injury [12–15]. Significant brain damage including large lesions, subdural haematomas, and lesions in the subcortical white matter and frontal lobes are all found more frequently in young children compared to older children after a TBI [13]. TBI in a young child is more likely to produce shearing injury to the brain over contusions [16]. These differences are due to the young child's brain being a softer consistency, with myelination and development of glial cells ongoing combined with higher water content and smaller axons [11]. The subarachnoid space of the child up to 2 years of age is also thinner, providing less of a buffer if the head is subjected to trauma [11].

Neurological Outcomes of Accidental TBI in Young Children

As we have mentioned, the majority of TBI in young children are from accidental causes. The immaturity of the young brain results in intracranial trauma that differs to older children and adolescents. For example, coup and contrecoup contusions are commonly associated with falls in children and adolescents, however, these injuries occur rarely for younger children under 4 years of age [11]. Coup and contrecoup contusions result from acceleration–deceleration forces, however, young children are typically close to the ground and the acceleration produced is not sufficient to cause the contusions [11]. Falls from a low height, such as falling off a bed or couch, are generally not sufficient to cause a significant head trauma, unless the head strikes a hard surface (e.g. solid stone, concrete) at a particular angle [17]. Falls over 1 m are likely to result in TBI and falls greater than 1.5 m are associated with both skull fractures and intracranial trauma [17]. Skull fractures are commonly seen in young children with TBI [17] and are often associated with epidural haemorrhage.

Neurological Outcomes of Abusive Head Trauma in Young Children

TBI in children from AHT are generally only seen up to 3 years of age and most occur within the first year of life [18]. Autopsy studies report that subdural haemorrhage is commonly seen with AHT as opposed to epidural hematoma [18]. Skull fracture is also common [19]. Gedde's landmark papers in the area refuted earlier assertions that AHT was commonly associated with traumatic diffuse axonal injury [20]. Early research suggested that shaking a baby caused rotational forces on the brain and resulted in traumatic insult, however, there is debate about whether the shaking is the main cause of significant intracranial trauma, with the damage generally prescribed to occur when the infant strikes a solid surface with force or speed (i.e. thrown against wall, etc.). Duhaime et al. [21] concluded from dummy model studies that shaking alone did not reach the thresholds for concussion, subdural haemorrhage or diffuse axonal injury [21]. Difficulties with understanding the differences in AHT to accidental TBI are further hampered by the reluctance of caregivers responsible for the injury to give accurate details on mechanism as well as timing.

History of Traumatic Brain Injury Research and Evolution of Theory in Very Young Children

The recovery of young children from TBI has been an area of intense debate. Margaret Kennard, a neurologist, principally studied the effects of neurological damage on primates. Her work led to the creation of the Kennard Principle, which posited a negative linear relationship between age of the organism at the time of a brain lesion, and the outcome expectancy. She concluded that the earlier in life a brain lesion occurs, the more likely it is for a compensation mechanism minimise the consequences [22]. In line with the Kennard Principle, authors have argued in prior research that young children recover better from TBI than adults, citing protective physiological factors including the relative flexibility of the child's skull, the lower frequency of intracranial haematomas, and the plasticity of the developing brain [22].

The opposing position is that the immaturity of the central nervous system young children present with poorer cognitive outcomes. Between one and two years of age, the neuronal proliferation and synaptogenesis in the frontal cortex reaches a peak. However, white matter development continues to age three or four years, and the frontal lobes and their functions as well as myelination continue developing until early adulthood [23]. As a consequence, early TBI may cause deficits in already acquired skills and hamper those functions yet to emerge [24], which leads to a reduced predictability of outcomes [25]. A critical review of the literature by Spencer-Smith and Anderson [26] concluded that neither early plasticity nor early vulnerability theories adequately and reliably explain the range of outcomes following injury to the brain at a young age and that such theories are likely an oversimplification.

In line with this, critical periods theories have been proposed and are the focus of more modern research. Critical periods in brain development consist of peaks and plateaus, characterized by the refinement and consolidation of neural networks [24, 27]. Recent theory postulates that a TBI during very early childhood disrupts the development of skills that are emerging and impede the acquisition of new skills, where skills that were acquired before the injury onset, are temporarily diminished and may return to pre-injury level [28–31]. As a consequence, younger age at injury onset has been associated with more severe damage of neural networks and skill development [29, 31]. However, the relationship between age and outcome depends on the neurological and cognitive maturational stage at the time of injury onset, which is not linear and varies among cognitive skills [26, 28, 31, 32].

Cognitive skills with a short window of development are less vulnerable to injury compared to those with an extended developmental trajectory [29]. High order cognitive skills require more time to develop therefore there is an increased vulnerability and reduced capacity for recovery [29]. For instance, focal and selective attention are relatively better preserved, and if affected commonly recover after TBI, but complex types of attention, such as shifting or encoding, are particularly vulnerable to early brain insult [28, 29, 33]. The extended developmental trajectory of the prefrontal cortex is the reason why executive function impairments are commonly seen after early TBI [26, 31]. During the first years of life brain networks are not yet refined and more brain regions necessarily participate in specific functions [26, 31]. As a consequence, early TBI often leads to generalized impairment [26, 31].

Neurocognitive Systems That Are Affected by Early TBI

A TBI sustained during early childhood has been associated with social skills, executive function, and memory deficits combined with compromised behavioural and emotional difficulties [34–36]. The prefrontal cortex plays a major role in the development of these functions, which are commonly affected by early TBI [36, 37]. A TBI during early childhood can alter the development

between the prefrontal cortex regions and the thalamus, basal ganglia, limbic system and posterior cortical systems [37]. Eslinger et al. [37] consider that an early brain lesion within the frontal lobe has a localized effect, also resulting in a reverberating effect that causes a disruption in the interaction among brain regions which are undergoing maturation. Brain lesion volume seems to be a predictor of cognitive impairment [34, 38]. Overall, recent studies support that early brain injury leads to a disruption of child development which is unlikely to recover to normal levels without the implementation of an intervention program [34, 36–38]. In particular, attention, working memory and social skills are vulnerable to early TBI [29].

Attention problems commonly hinder high order thinking functions, the child´s ability to acquire new knowledge, and later academic performance [29]. The attention models proposed by Posner and Rothbart [39] and by Mirsky et al. [40] had been well accepted in the field of neuropsychology. Posner and Rothbart [39] identified three networks: (1) alerting network, maintains and achieves sensitivity to incoming stimuli; (2) orienting network, selects relevant information from the incoming stimuli; (3) executive attention network, monitors and solve conflicts between thoughts, emotions and responses. The networks proposed in Posner´s model are drawn from neuroimaging studies that associated each network with different brain structures and chemical modulators [39]. Similarly, Mirsky et al. [40] consider attention a complex set of processes that can be subdivided into four distinct components: (1) focus attention, refers to the capacity to select specific information; (2) sustained attention, refers to the ability to maintain the focus and alertness during a period of time; (3) shift attention, refers to the capacity to change the focus of attention in a flexible and adaptive way; (4) encode attention, refers to the ability to register, recall and manipulate information. These components are underpinned by specialized brain regions that form part of an organized system [40] and can be impaired after the onset of TBI. For example, lesions in the orbitofrontal cortex had been associated with attention deficit/hyperactivity disorder (ADHD) after TBI onset [39].

Substantial studies described that children with early TBI are at risk of presenting new onset of ADHD [41–43]. ADHD is three times more common in children with a TBI [41]. However, these ADHD symptoms are less likely to be reported in young children with TBI, and become noticeable between middle to late childhood [43]. Pre-injury family psychosocial adversity and pre-injury child adaptive functions have been identified as predictors of ADHD secondary to TBI [41, 42]. Nonetheless, it is important to consider that children with ADHD have a higher tendency to experience a TBI [44], which may explain why pre-injury child adaptive function seems to be a predictor of secondary ADHD.

Working memory is a multicomponent system with limited capacity to store information temporarily during the performance of cognitively complex [45]. The components in this hierarchical model are: central executive, phonological loop, visuospatial sketchpad and episodic buffer [45]. The central executive controls attention, verbal and acoustic information is held by the phonological loop, visual information is held by the visuospatial sketchpad and the buffer episodic holds episodes through which information across space and time is integrated [45]. More severe injuries, earlier age at insult and attention span are predictors of impairments in working memory [46]. In addition, more time since injury is associated with a decline of verbal and visual-spatial working memory [47, 48]. The vulnerability towards impairments in working memory in younger children with TBI could be explained by the prolonged maturation process of the frontal cortex [47].

Impairments in social skills have a negative impact on children's quality of life [49]. The Social-Cognitive Integration of Abilities Model (SOCIAL) is a seminal model that defines essential aspects of social competency [49]. The first component of SOCIAL involves internal factors (temperament, personality, physical attributes) external factors (family environment, socio-economic status, culture) and brain development (neural base of social skills) as

mediators. The second component refers to emotional and cognitive elements (attention, socio-emotional and communication skills) required for the integrity of social skills [49]. These components interact at neural and behavioural levels resulting in social competence [49]. Theory of mind (ToM: ability to ascribe psychological states to others) and pragmatic language (ability to infer social meaning from complex language) are essential social skills that emerge during early childhood and are commonly impaired after TBI [50–53] Younger age at insult is a predictor of impairments in pragmatic communication [54]. However, these deficits may not be evident until later stages of life when social skills are expected to reach maturity [55]. Contrary to what is seen in young children, older children and adolescents are more likely to recover pragmatic communication and reach an adaptive level [54].

Radiological Predictors of Neuropsychological Outcomes—CT, PET, MRI

There is substantial research describing correlations between CT and MRI results with school problems, difficulties seen on a neuropsychological assessment, and overall recovery [56–58]. Beauchamp et al. [59] compared CT scans with susceptibility-weighted imaging MRI sequence (SWI/MRI) in children with TBI [59]. Their findings show that SWI/MRI technique can identify subtle neuroanatomic changes that CT scans overlook [59]. CT scans are effective in identifying injuries that require neurosurgical treatment [59]. However, SWI/MRI techniques can identify fine parenchymal lesions associated with cognitive and behavioural symptoms without exposing children to radiation, as opposed to CT scans [59]. Due to the diffuse nature of most TBI, cutting edge neuroimaging can link structural and microstructural findings with cognitive and behavioural outcomes [59]. There is an association between the number of lesions identified through SWI/MRI and intellectual functioning at 6-months post-injury [60]. Greater number of lesions have been found to lead to disruption of multiple neural networks and cognitive functions [60]. However, these studies were conducted in older children (5–16 years of age) and neuroimaging studies of social skills in early childhood are scarce.

More recently, using structural MRI, Ryan et al. [61] studied the association between ToM and neuroanatomical abnormalities in grey matter macrostructure at 24-months post-injury. They found that poor ToM was associated with neuroanatomical abnormalities in neural networks involved in social-affective processes.

To obtain more evidence of the neural regions implicated in the social brain network, Ryan et al. [62] investigated the neuroanatomic differences that children (aged 8–15 years) with TBI present in white matter microstructure with DTI. They found that at six months post-injury, poor ToM and pragmatic language was associated with abnormal diffusivity of the splenium of the corpus callosum, uncinate fasciculus, sagittal stratum, middle and superior cerebellar peduncles [62]. These are all structures that are comprised of white matter bundles with critical cortico-subcortical functional connectivity. While at 2 years post-injury, the same cognitive deficits were associated with abnormalities in the dorsal cingulum and middle cerebellar peduncle [62]. Their findings highlight the importance of studying changes in brain connectivity through the lifespan and indicate that using high-resolution imaging can allow early identification of children who are at risk of presenting with social cognitive dysfunction after TBI [61, 62].

Genc et al. [63] studied white matter microstructure with DTI during the subacute phase and its relation with injury severity and cognitive outcomes in children and adolescents (5–15 years) with TBI. Their results indicate that more severe injuries are associated with greater damage on white matter microstructure in the corpus callosum [63]. These microstructural disturbances were also associated with diminished information processing speed at 2 years

post-injury [63]. Injury severity and processing speed were key determinants of abnormalities in white matter development after paediatric TBI [63].

Using structural MRI, Yu et al. [64] investigated the long-term impact of childhood TBI on white matter, inhibition and cognitive flexibility at 16 years post-injury. They found that in healthy adults, inhibition and cognitive flexibility improved with increased cortical white matter [64]. In contrast, in survivors of childhood TBI, increase in white matter was associated with poorer inhibition and cognitive flexibility [64]. This study provides further evidence that TBI during childhood has a long-term impact on brain-behaviour connections that require further study [64].

High-resolution MRI techniques are specialized for intracranial arterial pathology and can provide more detail on the integrity of the developing brain after child TBI [65]. While the implementation of these techniques is limited due to clinical setting considerations, rapid advancements in the neuroimaging field may increase its accessibility [65].

Post-injury Management of Paediatric TBI

Phases of Post-injury Management

The recovery process can be divided into three phases [66]. However, these phases vary depending on the injury severity and case. The severity of the TBI is determined based on several parameters including level of consciousness, duration of altered consciousness and posttraumatic amnesia, evidence of skull fracture or cerebral pathology, and mental and neurologic condition. In a serious injury, the first phase in the recovery process is when the child is still in coma. During this phase the main goal is to maintain basic functions (feeding and physical strength) and monitored progress or deterioration [66].

The second phase starts when the child is medically stable and is able to receive an intensive rehabilitation. The goal of this second phase is to facilitate the child´s recovery and move towards discharge [66]. The nurse coordinates communication between medical, nursing and an allied health team. The allied health team may involve a Speech Therapist, to assess speech and language, an Occupational Therapist, to assess motor skills, a Neuropsychologist, to assess cognitive outcomes, a Social worker, to discuss family issues, a Clinical Psychologist, to assess adaptive behaviour issues, and Educational consultants, to communicate with the school staff [66]. The allied health team discusses rehabilitation priorities and works with the family to help them understand and enhance the recovery process. The time of discharged is decided based on the child's function, family's adjustment and the capacity of local services to provide ongoing therapy [66].

The final phase of recovery process follows hospital discharge; in this phase children are treated as outpatients and school teleconferences with teachers and school visits may be required. The main intention is to encourage independence in day-to-day life and ease the child´s return to school and reintegration into the community [66]. During this phase physical (adaptive equipment, such as wheelchairs), environmental (extra time for tasks, well-structured classroom environment) and instructional (educational programs, individual tuition, retraining of social skills) adaptations need to be considered [67]. In periods of transition, children with serious injuries tend to require more medical input and rehabilitation [68]. The allied health team share responsibility and work together with the child and family participate in the identification of goals and decision-making process. This collaborative approach seems to improve family´s feeling of competency, engagement with goals and outcomes [69, 70].

Neuropsychological Assessment in Paediatric TBI

Obtaining extensive information of pre- and post-injury function from the family provides qualitative data unlikely to be obtained elsewhere. This history informs selection of assessment measures, and can highlight areas that might be challenging for the family and child. Routinely, following childhood TBI, assessment does not occur in the acute stages post-injury. Rather, comprehensive assessment is conducted prior to school reintegration, in order to best inform educational management. Even at that point, hallmark impairments in attention, speed of processing and fatigue need to be considered when testing and interpreting findings. Standardized assessment methods are not always helpful for children with severe injuries. Severe cases required the use of other techniques, including contextual observation (at clinic, home or school), and parent and teacher's ratings. Parents and teachers may complete questionnaires to provide information about the child's functioning. In addition, follow-up assessments one year after injury onset and reviews during transitional stages should be implemented.

Neuropsychological assessments in children with TBI aim to (1) provide information about the integrity of brain functions; (2) detect and diagnose symptoms or disorders; (3) identify child´s strengths and weaknesses; (4) guide rehabilitation; and (5) monitor cognitive and behavioural changes over time, including those caused by treatments or interventions [65]. The location of the injury may guide hypothesis testing, but due to the diffuse nature of most lesions is important to assess all cognitive domains [65]. Once the pre- and post-injury history was obtained, the neuropsychological assessment begins by assessing intellectual function using standardized test batteries. Bayley Scales of Infant and Toddler Development is used to assess global functioning in children from 1 to 42 months of age [71]. Intellectual functioning is usually assessed with the Wechsler Preschool and Primary Scale of Intelligence in children from 2.6 to 7.7 years of age [72]. However, it is important to consider that global intellectual functioning can be insensitive to the cognitive consequences of TBI [73, 74]. A neuropsychological battery typically involves assessment of motor skills, sensory skills, attention, working memory, problem solving, social perception, long-term learning and memory, language, visuospatial perceptual skills, and behavioural and adaptive function [65]. NEPSY-II can be used in children from 3 to 16 years of age for most of those cognitive domains, including social perception [75]. The Behavior Rating Inventory of Executive Function (EF)—Preschool Version is a questionnaire regarding behaviours thought to be associated with EFs in daily activities, based on the family's and teachers' reports, for children between 2 and 5.11 years of age [76]. Other EFs can be evaluated with tests for school age children: the Test of Everyday Attention for Children assesses attention through tasks designed for children between 6 and 15 years of age [77], and the Delis–Kaplan Executive Function System involves verbal and spatial tasks for individuals of 8–89 years of age [78].

Behaviour and adaptive function are usually measured using parents' and teachers' questionnaires. The most common are the Child Behavior Checklist [79], Behavior Assessment System for Children [80], Eyberg Child Behaviour Checklist [81], and the Strengths and Difficulties Questionnaire which is available online in various languages [82]. Some of these questionnaires provide a score of social function, which does not reflect the child´s social cognition [83]. Therefore, to date, social cognition is assessed with experimental and a few clinically standardized tasks. For example, The Jack and Jill task is used to measure false belief understanding, Theory of Mind (NEPSY-II) tests understanding of the thought process of others, Affect Recognition (NEPSY-II) and Emotional and Emotive Faces Task assess a child's ability to discriminate among affective expression and emotive communication, and The Ironic Criticism and Empathic Praise task to measure understanding of how indirect speech acts are

used to impact the mental or emotional state of the listener [84].

Additionally, the clinician may decide to measure proximal environmental factors (parenting practices, parental stress, family functioning and parent mental health) that influence children´s development after TBI [65, 85, 86]. These factors can be assessed with the parent stress index [87], parenting scale [88], family burden injury interview [89] and depression and anxiety stress scale [90].

Approaches in Rehabilitation of Behaviour, Anxiety and Cognition

There is poor evidence from research focused on the rehabilitation of children after very early TBI. For this reason, in this section the rehabilitation approach implemented in the general paediatric population is discussed. An essential step in child rehabilitation is to provide caregivers and teachers information about the possible behavioural, emotional, social and cognitive short-term and long-term consequences of TBI [91, 92].

Woods et al. [93] developed the booklet 'Dealing with a Head Injury in the Family' (ABI booklet) and its accompanied facilitator manual [94], to provide parents of children with TBI information about the consequences of TBI and how these may limit child´s ability to cope with daily activities. One of the most challenging behavioural consequences of early TBI is difficult behaviour [95] and parents do not always understand that this is associated with the TBI or know how to respond to help the child redevelop adaptive capacity. The methods evaluated by a number of studies reduced behaviour problems in children with TBI by including parents in the intervention. Woods et al. [94] studied the efficacy of 'Signposts for Building Better Behavior' (Signposts) combined with the ABI booklet [93] in reducing challenging behaviour in children with acquired brain injury (ABI) and improve family-parental well-being and functioning. Signposts teach parents strategies to help them manage their child behaviour, parents set their own goals and put into practice strategies according to their child needs [96]. Signposts in combination with the ABI booklet demonstrated efficacy in preventing and reducing challenging behaviour in children with TBI and improving parental well-being within an Australian and Mexican population, and it is currently being studied in preschool children [97–99].

Similarly, Brown and colleagues [100] found that a parenting program in combination with Acceptance Commitment Therapy (ACT) were effective in decreasing a child´s behaviour and emotional symptoms, and reducing dysfunctional parenting practices. The ACT is part of a larger family of behavioural and cognitive therapies [101] that emphasizes acceptance rather than behaviour change only [102]. Take a Breath (TAB: 103) is an intervention programme that adapted ACT and problem-solving skills strategies for parents of children with life-threatening illness, including TBI [104]. TAB showed promising results in reducing parental stress and posttraumatic stress symptoms while improving parental psychological flexibility and mindfulness in parents [104]. This novel intervention is delivered via video conference to facilitate parent participation [105].

Children with TBI are at risk of presenting anxiety symptoms [106]. Some methods treat dysregulation symptoms, including depressive and anxiety symptoms [106]. Interventions aiming to improve dysregulation symptoms in children commonly use a cognitive behaviour therapy (CBT) approach [106]. A CBT program for managing anxiety [106] is being studied in children with TBI. The adapted program and the corresponding facilitator manual is now complete [107].

Attention and memory deficits are a common consequence of early TBI onset [108]. The Amsterdam Memory Attention training (AMAT-C) aims to improve children´s attention and memory after TBI [109–112]. This program was developed based on a model described by

Sohlberg and Mateer [113] in which cognitive domains are targeted based on its difficulty, from basic to complex. In AMAT-C daily tasks are done by the child under supervision of a coach (parent or teacher), in combination with weekly face-to-face sessions with the therapist [109–112]. Currently, our laboratory is studying whether replacing the face-to-face sessions with weekly online sessions increases participation [114].

There is substantial evidence describing social skills impairments in children with TBI [71, 115]. Social skills deficits negatively impact child psychological well-being, by diminishing the child´s ability to participate within their environment and develop meaningful relationships [51, 52]. However, there are no ecologically sensitive measures to identify impairments in this domain [116]. Our laboratory developed the Paediatric Evaluation of Emotions, Relationships, and Socialisation (PEERS) that will be the first ecologically sensitive, well-validated measure to detect social skills impairment in children [117]. PEERS aims to identify social skills strengths and challenges in children with TBI and other clinical groups [117].

Environmental Factors

A child´s development depends on an intact central nervous system and is shaped by proximal (within the family) and distal (outside the family) environmental factors [15, 53]. In comparison to older children, young children have few acquired skills. Young children develop cognitive precursors that will lead to high order thinking skills [53]. For example, joint attention and imitation are precursors of social skills, young children also develop inhibition which is required to regulate behaviour [118–120]. TBI in early childhood can interfere with the refinement and consolidation of neural networks and skills, alter proximal environmental factors (parenting practices, family functioning, parent mental health), and therefore disrupt a child´s cognitive, behavioural, social and functional development [85, 121].

Proximal (family functioning, parenting practices, parent mental health) and distal (social risk) environmental factors have been associated with a child´s risk of sustaining a TBI and the child´s functioning post-injury. Family factors associated with higher risk of TBI include low income, reliance on welfare benefits, minority status, frequent moves and high levels of parental stress [122–125].

Several studies found an association between family burden with child´s post-injury outcomes [126–129]. Proximal factors play an important role in young children's recovery [129]. High level of cohesiveness, supportive family relationships and low level of control had been associated with better outcomes post-injury and children from dysfunctional families present a higher risk of developing psychopathology post-injury [129]. Studies suggest that family environment pre- and post-injury influence behavioural and cognitive recovery [129–131].

Another environmental factor is parenting practices, commonly classified as authoritarian, authoritative and permissive styles [132]. The authoritative parenting style has been associated with positive outcomes post-injury [86, 133]. It consists of providing children clear expectations and reasonable limits, and encouraging them to formulate their own perspective and goals [133, 134]. The authoritarian style is characterized by the use of power and punishment to restrict the child [132]. Parents with permissive style allow children to regulate their activities and avoid setting limits [132]. Authoritarian and permissive parenting practices exacerbate internalizing and externalizing behaviours after TBI [86]. In contrast, the authoritative style benefits the child´s behavioural recovery [86]. Parenting practices are influenced by parents' mental health. In particular, high levels of parental stress hinder parents' ability to engage in warmth interactions with the child, and lead to dysfunctional parenting practices that may intensify as they face challenges associated with the brain injury, such as the need for rehabilitation, advocacy and additional support [134–136].

Social risk has been associated with outcomes after TBI [83, 137]. Social risk factors associated

with the parents include a low level of education of the primary caregiver, an unskilled occupation of the primary income earner, maternal age younger than 21 years of age during the child´s birth, single parents and English as a second language [137, 138]. Socio-economic status, family function and access to resources and support influence children´s recovery after TBI [83, 137].

Summary

Due to the flexibility of the skull, weak muscles of the neck and elasticity of the blood vessels, young children are more vulnerable to increase brain damage after TBI than adults. Primary and secondary mechanisms of TBI cause damage to the brain and may predict cognitive, behavioural social and functional outcomes. TBI at a young age disrupts the refinement and consolidation of neural networks and skills, and alter environmental factors. Due to the nonlinear maturational process of the neural networks, outcomes of TBI are not linear and vary among cognitive skills. However, cognitive skills with an extended developmental trajectory are more vulnerable to TBI. To study cognitive, behavioural, and social brain functions, cognitive models have been developed (e.g. attention, working memory and social competency). These cognitive models serve as a basis for assessment and management of early TBI. Post-injury management requires the participation of an allied health team to monitor progress, facilitate child´s recovery and encourage child´s independence. Neuropsychological assessments are part of the post-injury management. There are several assessment tools. PEERS is a novel assessment tool recently developed at the Murdoch Children's Research Institute that elucidates social cognition. Current studies are investigating intervention programs to treat behaviour problems, dysregulation symptoms, working memory, parenting practices, and parent mental health. Finally, environmental factors have a strong influence of early TBI outcomes that are considered during the post-injury management.

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