Cognitive Recovery and Development after Traumatic Brain Injury in Childhood: A Person-Oriented, Longitudinal Study

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Abstract

Influence of childhood traumatic brain injury (TBI) on cognitive recovery and subsequent development is poorly understood. In this longitudinal study we used cluster analysis to explore acute stage individual profiles of injury age and cognition in 118 children with traumatic brain injury. Repeated measures of cognitive function were conducted at 30 months, indicating recovery, and 10 years post-injury, indicating development. Nine clusters were identified. Recovery was evident in three clusters, two of them with low functioning profiles. Developmental gains occurred for three clusters and an acute profile of higher freedom from distractibility (FFD) and lower processing speed (PS) was related to positive differences. One cluster, average low functioning and especially low verbal comprehension, demonstrated a slower development than peers. This suggests that developmental change after TBI in childhood takes place on a continuum, with both chance of long-term catching up, and risk of poor development. An acute profile of higher FFD and lower PS seemed to reflect injury consequences and were followed by developmental gains. These results challenge previous findings, and warrant further investigation.

Key words: childhood; cognition; cluster analysis; follow-up studies; TBI

Introduction

NORMAL POST-NATAL BRAIN DEVELOPMENT is characterized by a roughly linear cortical white matter volume increase, starting in the first year of life in the primary sensory-motor areas, and proceeding to early adulthood in the pre-frontal and temporal regions, most likely reflecting ongoing myelination of axons.^{1,2} In contrast, gray matter thinning, thought to reflect elimination of connections of neurons not part of neural networks, and a strengthening of remaining pathways, has been shown to occur in a stepwise manner.¹ Increasing cognitive capacity is thought to coincide with the refining of remaining synapses, seen, for example, in adolescence, when thinning of gray matter still occurs in temporal and frontal areas,¹ and coincides with ongoing development of executive functions,³ memory strategies,^{4,5} and a gradual increase in attentional capacity and processing speed.⁶ The connection between executive function and a specific cognitive function is exemplified in adolescent memory development, in which increased capacity rests on a smoother execution of cognitive operations,⁵ and a more efficient use of elaborative memory strategies.⁴

How is cognitive development affected by a traumatic brain injury (TBI)? We know that children with TBI often differ in their cognitive functions when compared with healthy controls, but the subsequent interaction between cognitive development and recovery processes after different injury ages remains poorly understood.

After a TBI in childhood, plasticity, in the form of recovery, is most pronounced in the first 1–2 years after the injury.^{7–9} Emerging skills seem to be more vulnerable to injury than established skills, possibly because of a disruption of the organization of networks mediating cognitive skills.¹⁰ For example, Lehnung et al.¹¹ reported that earlier developed spatial orienting strategies were less affected by TBI than later established ones.

After the first 2 years post-injury, changes in function are thought to be associated primarily with developmental processing rather than recovery. After significant injury, where deficits in attention, processing speed, and executive function are common, the process of cognitive development may be disrupted, with the acquisition of new knowledge being more challenging for children with TBI than for peers. In accordance, later maturing skills are

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found to be more affected in long-term follow-ups after TBI.^{12–14} Over time, the effect may be cumulative, suggesting that time since injury is influential on outcome. This has been documented for verbal IQ and verbal delayed memory¹⁵ and verbal learning.¹⁶ In contrast, demonstrating the inconsistency of results on outcome after pediatric TBI, Catroppa and colleagues¹⁷ found that early established skills such as attention were more compromised than later established skills when compared with controls. Longitudinal studies show that by 10 years post-injury, significant differences in cognitive outcome are present between children with TBI and healthy controls.^{14,17–19}

In the pediatric TBI literature, the most commonly used variable-oriented statistical methods do not investigate how different cognitive functions interact in individual functioning and development. In the context of variable-oriented methods, the significance of a variable is derived from its position in relation to the position of the same variable in other persons or groups.²⁰ In the complementary person-oriented methods, a more holistic approach is emphasized.^{20,21} Information about the individual is regarded as far as possible as an indivisible unit, as the single variable only receives its full meaning in the pattern of it co-working with other relevant variables.²² Bergman et al.²² further argue that individual development is best understood from the perspective of processes and from the use of holistic, interactionistic, integrated models, as psychological development often has a non-linear and dynamic character. Variable-oriented methods and person-oriented methods are complementary methods, providing answers to different questions. Cluster analysis is one of many person-oriented methods, linking individual profiles of chosen variables into clusters, maximizing similarities within clusters, and minimizing similarity between clusters.²⁰ Cluster analysis has been used in cross-sectional studies of cognitive function after TBI in childhood.^{23–26}

From the abovementioned literature it is clear that a person-oriented method of data analysis has the potential to expand our understanding of outcomes after TBI in childhood. In this study of cognitive development and recovery after childhood TBI, we used cluster analysis to investigate acute stage profiles of injury age and four indicators of cognitive function, measured by Wechsler Index scores. We then investigated cognitive outcome in each cluster at 30 months and 10 years after injury. Because of the exploratory nature of the study, we did not posit specific hypotheses. The aim was to investigate how acute profiles of age-bound cognitive profiles were related to cognitive recovery and development after childhood TBI. Based on earlier findings, recovery was expected to be complete by 30 months,^{7–9} whereas possible developmental consequences were expected to be seen at the 10 year follow-up. Cognitive change was evaluated by repeated measures of Wechsler scaled score differences.

Methods

Participants

Between June 1993 and June 1997, 192 children were seen at the Royal Children's Hospital in Melbourne, Australia, with a diagnosis of TBI. Inclusion criteria for participation in a longitudinal follow-up project, of which this study is a continuation, were injury age between 3.0 and 12.11 years, documented accidental TBI including altered consciousness, medical records sufficiently detailed to determine severity of injury, and, finally, ability to be assessed. Exclusion criteria were penetrating brain injury; non-accidental injury; previous TBI; or pre-existing physical, neurological, psychiatric, or developmental disorder. Eleven children were ineligible for the study because of pre-existing psychiatric or neurological problems, three children had previous brain injuries, and one child

had a non-accidental injury. The inclusion criteria were met by 177 children, and their parents were invited to participate. Of these, 27 declined to participate because of inconvenience, distance, or lack of interest. The invitation was accepted by the parents of 150 children. Cause of injury and severity are described elsewhere.²⁷ A further 27 children injured before the age of 3, otherwise meeting the acute inclusion criteria, were included in the follow-up project, described in detail in the study by Anderson et al.²⁸ After excluding children who were not able to participate because of disability, the total sample comprised 172 children.

Children were divided into injury severity groups as follows: mild injury, Glasgow Coma Scale (GCS)²⁹ on admission 13–15, no evidence of any lesion on CT/MRI, no neurological deficits; moderate injury, GCS 9–12, and/or mass lesion or other evidence on CT/MRI and/or neurological signs; severe injury, GCS 3–8, mass lesion or other evidence on CT/MRI and/or neurological signs. GCS scores were recorded in the hospital file by admitting medical officers. Neurosurgical observations were first made every half hour in the neurosurgical ward and were then reduced to observations every fourth hour until the child had regained consciousness. Pediatric neuroradiologists and neurosurgeons reported CT/MRI scans. A validity check of the severity groups at 10 years follow-up confirmed significant differences with regards to GCS, duration of coma, CT/MRI, presence of neurological signs, and need for surgical interventions.³⁰

This sample was invited to participate in several follow-up assessments, within the longitudinal project. In the current study we investigated the results of individuals from the acute stage, 30 months, and 10-year follow-up time-points. Data from most participants were available at these time points, which was advantageous for the statistical analysis.

Measures

Profiles in the cluster analysis were based on age of injury and levels of cognitive functioning. Data on cognitive function were taken from four indices, derived from age appropriate Wechsler Scales; Wechsler Preschool and Primary Intelligence Scale-Revised, (WPPSI-R)³¹ or the Wechsler Intelligence Scale for Children, (WISC-III),³² and, finally, Wechsler Adult Intelligence Scale-III, (WAIS-III).³³ The cognitive indices derived from the Wechsler scales were Verbal Comprehension (VC), Perceptual Organization, (PO), Freedom from Distractibility (FFD), and Processing Speed (PS) (mean = 10, SD = 3). For the WPPSI-R, indices were constructed with subtest scaled scores summed and averaged within each index. We used similar subtests as in the WISC-III indices: VC, information, similarities, vocabulary, comprehension; PO, picture completion, block design, object assembly; FFD, sentences, arithmetic; and PS, animal pegs.

In the acute phase post-injury parents were asked to complete the Vineland Adaptive Behavior Scale Interview edition (VABS)³⁴ based on their child's pre-injury functioning. The scale provides information on a child's level of adaptive function in the domains of communication, daily living skills, and socialization. A total adaptive behavior score was derived from these domains (mean = 100, SD = 15) and was used as a measure of pre-injury adaptive function.

Procedure

Data were collected by trained psychologists at the acute stage, 30 months, and 10 years after injury. From the acute stage, complete data from the Wechsler indices were available for 115 persons. Three persons had missing data in the FFD Index whereas one person was also missing PS data. For these three cases, data were imputed from persons with similar severity grade and injury age and similar scores in other indices, resulting in a group of 118 participants from the acute stage. Clusters were derived from the acute measures of cognitive indices of these 118 participants. At the

TABLE 1. DEMOGRAPHIC DATA OF THE CLUSTERS

Cluster	1	2	3	4	5	6	7	8	9
Participants, acute and 30 months, <i>n</i>	12	12	15	15	8	20	16	15	5
Participants, 10 years, <i>n</i>	3	7	7	8	5	10	9	8	2
Injury age, years	9.8	6.8	4.4	5.9	10.9	9.6	9.8	9.8	4.2
Mild injury, <i>n</i>	0	1	5	1	3	9	9	2	5
Moderate injury, <i>n</i>	2	6	10	10	4	8	6	9	0
Severe injury, <i>n</i>	10	5	0	4	1	3	1	4	0
Sex	M	M	M	F	M	M	M	M	F
Pre-injury adaptive function M (SD)	103 (13)	106 (13)	105 (20)	107 (20)	112 (13)	108 (13)	106 (17)	106 (15)	109 (29)

30 month follow-up, all 118 participants had complete data from the Wechsler indices and remained in the study, forming the base for the 30 months outcome data. At the 10 years follow-up, 59 of the 118 participants had complete data from the Wechsler indices, thus forming the base for the 10 year outcome measures.

Statistical analysis

Tests of group differences between the original group of 172 persons and the study groups at the three points of time were conducted, using multivariate analysis of variance (MANOVA), for continuous variables and Mann–Whitney U for categorical variables. A violation of test assumptions for equality of variances was found, and Pillai's trace was therefore used in the MANOVAs.

Cluster analysis was performed on *z*-standardized variables from the acute stage. Before analysis, data were checked for assumption violations, with no negative results. Hierarchical cluster analysis was performed in SPSS, version 19. As a measure of the characteristic of each individual profile we used squared Euclidean distances, as they take account of both the variation within and the level of each profile. The Ward method was then used to combine the individual profiles into clusters based on similarity. The determination of the optimal numbers of clusters was based on three factors: a striving to avoid a sudden drop of the explained variance, an explained variance of more than two thirds of the explained variance between the profiles, and finally, a striving for a solution with a theoretical meaningfulness.²⁰ To validate the solution, Complete linkage was used as an alternative method of detecting the number of clusters. The individual overlap in cluster membership between the two hierarchical clustering methods was calculated by taking the pair of profiles, identified as similar, and dividing the number of individuals in the complete linkage cluster from the number of individuals in the Ward cluster. A cluster in the complete linkage solution could have the same Ward cluster as its most similar profile. The percentages for the nine clusters were then averaged.

This is a test of whether the Ward result was caused by the method or the actual data. To obtain a measure of changed cognitive levels between the assessments, a mean difference was calculated based on a within-groups comparison: in every cluster, the acute outcome in each index was subtracted from the outcome of the same index at 30 months. Differences were summed and divided by four, thus providing a mean value for the within-group change between the acute stage and 30 months follow-up. Paired *t* tests were then conducted on the mean cognitive outcome of acute–30 months differences and 30 months–10 years differences. As a measure of effect size, eta squared (η^2) is presented, interpreted as suggested by Cohen³⁵ as 0.01 = small effects, 0.06 = moderate effects, and 0.14 = large effects. For a measure of change in the separate indices, acute outcomes in each of the acute clusters were subtracted from the same clusters' separate index outcome at 30 months follow-up. A mean change for the nine clusters was then calculated for each index. This was repeated for the period between 30 months and 10 years, and finally between the acute stage and the 10 years follow-up.

Results

The comparison of injury and demographic factors revealed no significant differences for any of the group comparisons between the original group and the study group at the acute stage, 30 months, and 10 year follow-up for socioeconomic status, pre-injury adaptive function, age at injury, acute full scale IQ, injury severity, duration of coma, abnormal CT, fractures, surgical intervention, neurological signs, and sex.

In the cluster analysis, a solution with nine clusters was chosen, explaining 69% of variance, and resulting in differentiated profiles. Descriptive data for the clusters are presented in Table 1. Wechsler index scores at the acute stage and at 30 months and 10 years follow-up are presented in Table 2. The cognitive levels of the profiles are presented as Wechsler standard scores in Figure 1 a-i.

TABLE 2. WECHSLER INDEX SCORES OF THE CLUSTERS AT ACUTE STAGE, AND THE FOLLOWING OUTCOME AT 30 MONTHS AND 10 YEARS FOLLOW-UP

Cluster	Acute stage				30 months follow-up				10 years follow up			
	VC: M(SD)	PO: M(SD)	FFD: M(SD)	PS: M(SD)	VC: M(SD)	PO: M(SD)	FFD: M(SD)	PS: M (SD)	VC: M (SD)	PO M (SD)	FFD M (SD)	PS M (SD)
1	5.2 (1.9)	3.9 (1.8)	7.5 (2.4)	3.5 (2.0)	5.7 (2.4)	6.4 (2.4)	8.8 (3.0)	5.3 (3.0)	6.5 (2.1)	8.1 (3.7)	7.7 (4.9)	7.5 (4.7)
2	8.1 (0.8)	9.3 (1.4)	9.2 (1.6)	5.9 (1.4)	7.6 (1.8)	10.1 (2.5)	9.0 (1.7)	7.7 (1.6)	9.4 (2.2)	10.3 (3.3)	10.7 (1.9)	10.2 (3.2)
3	8.6 (1.2)	9.7 (2.6)	8.0 (1.5)	10.9 (2.0)	9.3 (1.8)	9.7 (2.6)	9.3 (2.7)	9.9 (3.8)	9.6 (2.3)	9.7 (4.2)	7.3 (2.7)	10.4 (4.7)
4	11.1 (1.2)	10.4 (1.6)	11.5 (1.2)	9.1 (1.4)	9.7 (1.5)	10.8 (2.1)	11.4 (1.9)	9.4 (2.1)	11.2 (3.2)	11.2 (3.1)	11.1 (3.2)	11.4 (1.8)
5	11.2 (1.5)	10.5 (2.7)	14.6 (1.2)	10.4 (1.3)	10.8 (1.3)	12.3 (2.7)	12.8 (2.0)	10.8 (2.6)	13.6 (1.9)	13.5 (2.7)	12.6 (2.5)	11.7 (2.3)
6	9.6 (1.3)	10.6 (1.2)	8.8 (1.2)	9.0 (1.5)	8.9 (2.0)	12.1 (2.2)	9.1 (1.8)	9.7 (1.5)	9.9 (2.0)	11.9 (3.4)	8.9 (1.7)	9.4 (2.1)
7	9.8 (1.2)	12.3 (2.0)	10.4 (1.7)	12.3 (1.6)	9.8 (1.7)	12.8 (1.9)	11.0 (2.5)	12.4 (2.3)	10.2 (2.8)	12.8 (3.3)	10.0 (1.6)	10.8 (2.6)
8	5.2 (1.6)	8.8 (2.0)	6.5 (1.7)	8.3 (1.8)	5.6 (1.2)	9.2 (2.2)	7.2 (1.4)	10.1 (2.1)	5.1 (1.6)	7.1 (2.4)	6.1 (1.9)	7.9 (2.2)
9	11.2 (0.7)	14.4 (1.8)	13.3 (1.2)	14.0 (0.7)	10.3 (2.0)	13.6 (1.2)	11.6 (1.6)	11.7 (3.6)	11.8 (2.0)	15.4 (1.1)	11.3 (6.4)	12.1 (1.8)

VC, Verbal Comprehension; PO, Perceptual Organization; FFD, Freedom from Distractibility; PS, Processing Speed.

In the validity check, a nine-cluster solution provided almost identical clusters, but with a much higher degree of explained variance, 80%, and an individual overlap of cluster membership of 87%, thus supporting that the chosen solution in this study reflects the features of the data, and not the particular method employed.

Differences between acute and 30 months follow-up

After calculating the difference in mean cognitive function for each cluster between the acute stage and 30 months follow-up, those differences were ranked and are presented in Table 3. A

FIG. 1 (A) Index levels in Cluster 1. (B) Index levels in Cluster 2. (C) Index levels in Cluster 3. (D) Index levels in Cluster 4. (E) Index levels in Cluster 5. (F) Index levels in Cluster 6. (G) Index levels in Cluster 7. (H) Index levels in Cluster 8. (I) Index levels in Cluster 9.

FIG. 1 (Continued)

positive value indicates increased function. Three clusters, among them the two low-functioning clusters had significant improvements, indicating recovery. Conversely the high-functioning Cluster 9, with an injury age of 4.2 years, had a decrease in function that was non-significant but supported by a strong effect size. Five

clusters showed small, non-significant changes in cognitive functions between the acute stage and 30 months follow-up.

Differences between 30 months and 10 years follow-up

To measure developmental change after the primary stage of recovery, the same calculations were made for each cluster in the period between 30 months and 10 years follow-up. The ranked differences are presented in Table 4. Significant change, supported by strong effect sizes, indicates developmental gains for three clusters. An acute profile of higher FFD and lower PS in profiles is related to positive differences. The low-functioning Cluster 8 demonstrates a significant decline in function, supported by a strong effect size.

Discussion

Recovery and development

Overall significant cognitive recovery took place in three of the nine clusters, representing 40% of the total sample. Of these, two clusters showed initially low cognitive ability, moderate or severe injury, and average pre-injury adaptive functions, which suggests

TABLE 3. RANK OF DIFFERENCES IN COGNITIVE OUTCOME BETWEEN ACUTE STAGE AND 30 MONTHS

Cluster	Description of the clusters	Severity, md	Cognitive diff, SS	Paired t -tests			
				T	df	p	η^2
1	Age 9.8, low, low PO and PS	sev	1.5	-3.217	11	.01	.48
8	Age 9.8, average- low, low VC	mod	0.8	-2.628	14	.02	.33
6	Age 9.6, average	mild/mod	0.5	-2.409	19	.03	.23
2	Age 6.8, average-low, lower PS	mod/sev	0.5	1.002	11	.34	.08
7	Age 9.8, average, stronger PO and PS	mild	0.3	-1.090	15	.29	.07
3	Age 4.4, average, stronger PS	mod	0.3	-.806	14	.43	.04
4	Age 5.9, average	mod	0.1	.843	14	.41	.05
5	Age 10.9, average- high, very strong FFD	mod/mild	0.0	.098	7	.92	.00
9	Age 4.2, high functioning	mild	-1.4	2.363	4	.08	.58

Low = $\leq$ index score 7. Average = 8 – 9.9 index scores. High = $\geq$ 10 index score.

VC, Verbal Comprehension; PO, Perceptual Organization; FFD, Freedom from Distractibility; md, mode; PS, Processing Speed; SS, standard score.

TABLE 4. RANK OF DIFFERENCES IN COGNITIVE OUTCOME BETWEEN 30 MONTHS AND 10 YEARS

Cluster	Description of cluster	Severity, md	Cognitive diff, SS	Paired t tests			
				T	df	p	η^2
2	Age 6.8, average-low, lower PS	mod/sev	1.7	−2.515	6	.05	.56
5	Age 10.9, average- high, very strong FFD	mod/mild	1.3	−3.276	4	.03	.73
1	Age 9.8, low, low PO and PS	sev	0.9	−5.651	2	.03	.94
9	Age 4.2, high functioning	mild	0.9	−1.667	1	.34	.73
4	Age 5.9, average	mod	0.5	−1.070	7	.32	.14
6	Age 9.6, average	mild/mod	0.1	.448	9	.66	.02
3	Age 4.4, average, higher PS	mod	−0.4	−.181	6	.86	.01
7	Age 9.8, average, stronger PO and PS	mild	−0.6	1.441	8	.19	.20
8	Age 9.8, average-low, low VC	mod	−1.4	3.271	7	.01	.60

Low = $\leq$ index score 7. Average = 8 – 9.9 index scores. High = $\geq$ 10 index score.

VC, Verbal Comprehension; PO, Perceptual Organization; FFD, Freedom from Distractibility; md, mode; PS, Processing Speed; SS, standard score.

that low acute profile reflects brain injury related deficits. Developmental gains between 30 months and 10 years post-injury were seen for three clusters with an acute profile of higher FFD and lower PS, representing 25% of the sample. The documented developmental gains challenge earlier reports that recovery is complete by 2–4 years post-injury.^{7–9}

No recovery or developmental gains

No overall cognitive recovery was evident in six of the nine clusters. Of those, five clusters demonstrated minimal or no developmental gains, whereas the average-low functioning Cluster 8, with low VC, had a significant decreased function, supported by high effect sizes. A decreased function is in concordance with earlier studies,^{15,16} providing support for cognitive functions being at risk of slower long-term developmental gains than peers after TBI in childhood. On the other hand, the result of no recovery for several subgroups might have been a consequence of the use of the measure of *mean* cognitive function. When looking at the mean changes of the group in the period of recovery, separate indices showed both minimal change (FFD: 0.1, and VC: −0.3) and a larger change (PO: 0.7). A recovery of PO may, therefore, be

hidden in the measure of the *mean* cognitive change. A check of the change in PO between the acute stage and 30 months follow-up reveals that Cluster 5 ($n=8$) had no significant change with regard to mean cognitive level, whereas the improvements in PO reached a level of 1.2 index scores. However, no other cluster had a significant change indicating recovery (Clusters 1, $n=12$, and 6, $n=20$) accompanied by PO improvement over 1.0 index score. Taken together, the two larger of the three clusters with no significant change in the period of recovery did not have a pronounced improved function in PO hidden in the measure of overall cognitive function.

Plasticity and developmental gains

The enhanced plasticity in the developing brain rests on an enhanced excitability across synapses, which increases the plasticity of the developing brain, but it also makes it more vulnerable to damage from drugs, seizures, sensory deprivation, and abuse.³⁶ Recovery after early brain insult is described as taking place on a continuum. Plasticity of the young brain, underpinned by a range of complex neural processes, is at one end of the continuum connected to good recovery, whereas the vulnerability at the other extreme is

associated with elevated risk of poor recovery of the developing brain.³⁷ The developmental cognitive gain and lack of appropriate developmental gains found in this study may indicate this continuum being apparent at 10 years after TBI in childhood.

Limitations and suggestions for further studies

Although the sample in this longitudinal study is quite large, attrition from the cohort at the 10 years follow-up is a limitation, but remains comparable to attrition rates reported in previous pediatric longitudinal studies.^{38,39} Because of careful documentation at the time of injury, we were able to establish that there were no differences in injury or demographic variables when comparing with the original group. This study is, to our knowledge, the first with repeated measures on small subgroups based on cognitive profiles at the acute stage after pediatric TBI. The relevance of our results has yet to be evaluated by other studies with similar designs. Gains in future research could be made by using more homogeneous samples, with regard to injury severity. With a more homogeneous sample, more inclusive profiles could then investigate the importance of different variables in greater detail, for example the impact of age on recovery and development. Larger samples could enable cluster analysis of cognitive profiles also at long-term follow-ups, making it possible to study how cognitive profiles change during recovery and development.

Conclusion

This study found that, contrary to previous findings, recovery or accelerated development is possible up to 10 years after pediatric TBI. Decrease of function was also evident, suggesting that long-term developmental change take place on a continuum, where plasticity of the young brain at one end of the continuum is connected to good recovery after TBI, whereas the vulnerability at the other extreme is associated with elevated risk of poor recovery. An acute profile of higher FFD and lower PS in profiles was related to developmental gains. Low PS within cognitive profiles and low function among moderately and severely injured individuals with average pre-injury adaptive function at the acute stage should be followed by rehabilitation services aimed to support both recovery and long-term developmental gains.

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